GENERALIZED PAIN

CLINICAL SYMPTOMATOLOGY OF INTERNAL DISEASES

PART II

GENERALIZED PAIN

BY

PROF. DR. NORBERT ORTNER

VIENNA

Only Authorized Translation Into the English Language of the 2nd German Edition

BY

FRANCIS J. REBMAN

WITH AN INTRODUCTION BY THOMAS WEBSTER EDGAR, M.D., NEW YORK

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AUTHOR'S PREFACE

THE favorable reviews of my book on "Abdominal Pain" which have appeared in the medical journals have encouraged me to write this Second Part, dealing with "Generalized Pain." In these two volumes I have carefully and clearly discussed all the salient features and symptoms of painful internal diseases, and I sincerely trust that the reader will derive that benefit which it was my intention to convey.

NORBERT ORTNER.

Vienna.

INTRODUCTION

"Generalized Pain," Part II of Clinical Symptomatology of Internal Diseases, as translated from the original German, represents a complete symptomatology of all the painful sensations experienced by the human organism, with the exception of the abdominal region, which is discussed in a separate volume.

Prof. Dr. Ortner has made it possible by this book for the physician to appreciate the selectiveness of the symptom *pain,* and the text may be used as a guidance in the differential diagnosis of many pathological conditions that heretofore have been clouded by a multiplicity of vague uncharted painful sensations.

This book "analyzes pain," and may be used to great advantage by the physician when questioning his patient.

The more than kind reception accorded Part I, "Abdominal Pain," undoubtedly will serve as a fitting introduction to Part II.

Prof. Dr. Ortner has made the subject of generalized pain preeminently his own.

The translator has chosen clear and simple English, making the book easier to digest and

more readily assimilated. The long, interwoven sentences of the original German have been rendered short, presenting to the reader, however, the original text in all its exactness.

THOMAS W. EDGAR, M.D.

New York City.

TRANSLATOR'S PREFACE

Dr. Ortner divides his book on Clinical Symptomatology into two parts, viz.: "Abdominal Pain" and "Generalized Pain." The first part deals entirely with pain which manifests itself in the abdominal region of the human body and all the subdiaphragmatic organs contained therein. This volume was published in the early part of 1922 and has established itself firmly in the medical book market.

The second part is now ready for distribution. It deals with pain felt by the patient in those parts of the anatomy which are not included in the first volume, and the author very appropriately gives it the title of "Generalized Pain."

The language employed is simple and not too technical, a quality which will readily appeal to the profession. The forthstanding features of the book are the marvelous clinical experience of the author, which he gives unstintingly to the reader, and the clever way in which he weaves the localized manifestations of pain and their irradiations into adjacent and remote parts together into a solid unit. The two volumes combined form a perfectly balanced whole which

can be conscientiously recommended to those who seek reliable information on the subject.

I sincerely hope that the book will meet with the approval of the medical men of this country, and that my labors as translator will reap a well-earned appreciation by the thoughtful student of symptomotology. The book is devoid of padding and redundancies. Every sentence is carefully weighed and a necessity in its place, so that without it the totality of the subject would be disturbed, if not seriously injured. I think that both "Generalized Pain" as well as "Abdominal Pain" possess the inherent centrifugal power of success.

I also take this opportunity to thank *Dr. Thomas Webster Edgar* for his great kindness and exceeding patience in looking over the proof-sheets.

THE TRANSLATOR.

New York.

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GENERALIZED PAIN

Pain in the Heart and the Cardiac Region

Pains in the Thoracic Wall.—If a patient complains about pain in the cardiac region, we should first of all ascertain whether this pain is really located in the heart itself, or its muscular system, or is erroneously claimed by the patient to emanate from these organs.

In this respect the existence of a pain-provoking disease in the soft or bony coverings over the heart, or in the skin, the mamma, the muscles, the intercostal nerves, the ribs or even the sternum, may be excluded. A careful examination and a proper knowledge of the symptomatology in such cases will quickly solve the problem. The reader is here referred for particulars to the chapters on "Pain in the Chest" and "Pain in the Bones."

What I wish to emphasize at once is the fact that women who have abnormally large breasts, with the left mamma still more developed, commonly complain of pain in the cardiac region or in the heart itself which is of a pressing or

oppressive nature. These patients are, as a rule, rather stout in general, and suffer from difficulty in breathing, especially when making any kind of bodily effort, conditions which often lead to a mistaken diagnosis of an affection of the cardiac muscles, or an anatomical defect of the heart proper. I have come across such errors repeatedly.

The suspicion that the pain is caused by the over-heavy mammary glands may be based on the following facts: (1) an abnormal development of the mamma; (2) the patients often admit, without being asked, that the pain is relieved as soon as they raise the mamma with the hand; (3) when the patient is lying on her back the pain continues, but disappears when the mamma is raised with a suspensory.

Pain in the cardiac region may also be caused through mechanical friction of the dress on the nipple. As soon as the cause is removed, the pain vanishes.

Moderate Heart Pain.—If we are satisfied that the affection is not located in the thoracic frame, we have ground to believe that the seat of pain is to be found in the heart itself, or in its large blood vessels. But we should also think of a reflex action arising from a disease in some neighboring or even distant organ.

The pain varies in intensity. Sometimes it consists only of a slight sensation of incon-

venience, sometimes it is moderate (subacute), while at other times it is violent (acute).

Acute Pericarditis.—Most likely we are dealing with an acute involvement of the heart, generally of the pericardium, but it may also be myocardiac or endocardiac in its nature. In acute pericarditis the patient complains of stabbing pains, and pressure in the region of the heart. In some rare cases the pains assume a very intense and troublesome character. But more about this in another place. The pains just described may also be present in subacute and chronic pericarditis. In the majority of cases, however, they seem to be absent even where a chronic, chiefly tuberculous inflammation of the pericardium exists. But nearly every kind of tuberculous serositis (pleuritis, peritonitis) runs a painless course. A pneumopericardium or a hematopericardium, it is needless to say, will also produce pain in the region of the heart.

Acute Myocarditis.—Similar pains, generally accompanied by slight fever, may be observed in every case of myocarditis acuta, when an abscess formation in the myocardium is present, coupled with or following an ulcerous septic endocarditis.

Overexertion. Paroxysmal Tachycardia.—Slight pain in the cardiac region, generally described as stabbing, piercing, painful pressure

at times follows in the wake of an acute overexertion, i.e., an acute straining of the heart due to sport or gymnastic exercises, intense psychic excitement (increase in blood pressure), or after an attack of tachycardia. These troubles, especially oppression in the chest, manifest themselves naturally only when a constitutionally weak, untrained heart is suddenly subjected to unusual exertion: shortness of breath, inability to draw a long breath, dryness in the mouth, constant irritation to clear the throat with shreds of blood in the mucus, abnormally large content of urates in the urine.

Similar conditions prevail when the heart is afflicted with a pre-existing anatomical defect and is suddenly subjected to unusual, severe demands; but they may also be due to an acute loss of tonus in an otherwise normal heart. A thin, scarcely palpable pulse, cold sweats, pallor of the face, coldness of the point of the nose, cold and clammy hands and feet, short breath, angina, oppression in the region of the heart, vertigo, dizziness, eventually acute dilatation of the heart, systolic murmurs, high blood pressure, all these may be added to the aforesaid symptoms.

Acute Insufficiency of the Suprarenal Capsules.—I refer here to that fierce pain which is described by some authors as a sign of insuffi-

ciency of the suprarenal capsules in *Addison's* disease. Personally, I have never observed it.

But an abnormally irritable heart may, even without previous overexertion, give rise to analogous complaints of pain or palpitation, especially if hypoplasia be present. Such an overexcitable heart may easily be traced by a pronounced and spreading apex beat (in cardiac hypoplasia even by a normal or also abnormally weak apex beat), by an abnormally loud second aorta tonus, often by a temporary systolic sound over the apex or the pulmonary artery, by inclination to tachycardia, by a choppy, peripheral pulse, or by other vasomotoric symptoms.

A pain which is strictly confined to the apex and rendered more acute by external pressure may also be the symptom of a chronic affection of the cardiac muscle, in fact a definite sign of a cardiac aneurysm.

Of high diagnostic interest are those cases in which after a pronounced stenocardiac attack pressure, oppression and moderate pain in the cardiac region irregularly persist. In most cases a slight rise in the temperature will be observed, the objective examination shows pericarditic friction, eventually the formation of a fluid exudate of the pericardium, i.e., a clear case of *epistenocardiac pericarditis,* caused either by an embolism of the branches of the coronary artery due to a pericardial infarct, or by a partial

chronic aneurysm of the heart. In the absence of pericarditis a similar symptom complex, i.e., pressure, oppression, slight pain, may endure after an anginotic attack. The cause for this will be found in an *epistenocardiac myocarditis* (*myomalacia*) coupled with dilatation of the heart. Its presence is indicated by a slight rise in the temperature. In some rare cases these pains assume an intensely bothersome character.

I wish to emphasize a fact which seemingly has not as yet been properly appreciated, i.e., if a patient complains of a sudden, solitary or irregularly recurring pressure or stitch in the cardiac region or behind the sternum, the possibility of extrasystolic contractions must not be overlooked as the probable cause of such solitary sensations. According to my own experience, the so-called frustaneous contractions are the primary exciting factors in these sensations, in conjunction with all the other *extrasystolic conditions.* This seems to me an indirect proof that cardiac pain may be the result of an ischemia of the myocardium. The same may be said about the presence of cardiac pain in *hemorrhagic anemia.*

A stitch in the region of the heart may, however, be a purely nervous symptom, recognizable when it is located to the left above, but without, the absolute heart-dulness, or at the point of the apex, where it is joined by a localized sensible

pressure, i.e., an hyperesthesia of the membrane over the heart-dulness.

It is a well-known fact that pain in the cardiac region of a convulsive, oppressing character is often the sequel of some violent *psychic emotion.* But it is well to remember that such emotions may lead to real anatomical pain, e.g., angina pectoris.

These stabbing pains cannot, however, always and definitely be ascribed to nervous conditions; they may just as well be the accompanying symptom of a *chronic anatomical disease,* such as chronic myocarditis, myofibrosis, myodegeneration of the heart, arteriosclerosis, renal heart with increased blood pressure, affections of the mitral or aortic ostium (the latter, however, produce pain rather behind the sternum), a total pericardial concretion with the heart (only in exceptional cases), fatty heart (polysarcia cordis et myodegeneratio adiposa), the beery heart, cardiac syphilis, affection of the heart due to growing, dropping heart, also every relatively weak heart, in fact all morbid processes which are connected with the left ventricle. They all lead to pains in the region of the heart, and behind the sternum, to a retrosternal sensation of pain, eventually to a painful dyspnea.

Enteroptosis, above all sclerosis of the coronary artery, frequently produce similar sensations of pain. Sclerosis of the coronary arteries

causes pain in successive attacks, whilst the other affections mentioned above produce a more or less enduring pain of varying degree and recurrent after bodily motion or exertion. Many patients who complain about recurrent pains in a certain localized point of the cardiac region will be found to have suffered a short time ago from an attack of pericarditis. In such cases I have ventured upon a diagnosis of *localized pericarditic adhesion.* Whether I was right I will not presume to say. But I know for a certainty that in other cases of definite (proved by post mortem) such like adhesions no traces of pain could be established. It is also certain that contractures of the pericardium with the diaphragm may be responsible for palpitation, dyspnea, and sudden stabbing pains in the cardiac region frequently accentuated by increased diaphragmatic excursions (deep breathing, sneezing, coughing, when ascending stairs). The diagnosis must lean on the anamnesis (previous pleuritis, inflammatory pleural affection in the subphrenic cavity, especially on the left side), and eventually on the Roentgenogram. Analogous manifestations may be provoked by contractures of the diaphragm with parietal pleura (*pleuritis adhesiva*).

In enteroptosis, particularly after cardioptosis, we hear complaints about heart pain, palpitation, anguish, chiefly when in left situ owing

to increased displacement of the heart. Similar painful sensations are experienced in every other *abnormal lateral displacement of the heart,* in sclerosis of the aorta, cachexia, neurasthenia, but in the latter also without abnormal lateral displacement because neurasthenics ars as a rule supersensitive; likewise in many other diseases of the heart, even by persons in apparently good health, probably when extraordinary demands are made on the cardiac organs, when the heart is pushed against the thoracic wall, and when the *diaphragm is raised* above its normal level by any cause whatever in the healthy as well as in the afflicted. Palpitation, oppression, anguish are the usual accompanying symptoms.

Arteriosclerosis is associated with pain in the region of the heart because it increases cardiac labor and raises the blood pressure. Of greater moment still seems to me the fact that these self-same pains with palpitation, early fatigue, headache, and dizziness are often enough experienced in the *presclerotic stadium,* and that at a time when we find a more or less distinct accentuation of the second aorta tonus with a ringing sound and of a vacillating intensity, likewise a moderate rise in the blood pressure subject to constant changes, and eventually a slightly elevated apex beat. All these manifestations may completely, or at any rate partially, disappear after a few months when the

causating factors (bodily overexertion, pre-eminently mental excitement, also chronic nicotinism) have been eliminated.

Retrosternal pains, chiefly above or behind the sternum, deserve special mention. They manifest themselves in the sense of oppression, pressure, constant or convulsive sensations, at times coupled with irradiation towards both sides of the chest or of the shoulders, rarely in the left arm under bodily exertion or mental excitement, caused by sclerosis or lues of the aorta (*aortalgia*).

They approximate anginoid pains with this difference, that they are devoid of the feeling of anguish and the fear of a recurrence of the trouble, that they radiate chiefly into the arms, preferably the left one, and assume the form of spontaneous attacks. But they frequently lead to anginoid or anginose attacks, because their basic affection of the aortic wall easily leads to a contraction or distortion of the ostia of the coronary arteries, or similar diseases. A distinguishing feature of these "aortal" pains may be found in their rapid disappearance upon the removal of the causative element (exertion, excitement). What they have in common with angina pectoris is *Head's* cutaneous hyperesthesia of the chest and the left arm. As in genuine angina pectoris, so here also moderate exercise in the open air—

but not in a closed room—will banish the pain promptly.

Continuous pains in the cardiac region may be due to affections which superinduce *spatial contractions of the heart* from without. The patient complains of pressure, oppression and at times of moderately intensive pain in the region of the heart. There we are confronted by three possibilities: (1) the formation of solid masses in the anterior mediastinum (neoplasms, cysts, gummata or any kind of inflammatory process). The same may happen in the posterior sternum. (2) Mediastinal pleuritis (diagnosis in detail is given elsewhere). (3) An accumulation of gas in the cellular tissue of the posterior mediastinum, the sequel of an interstitial emphysema which is easily recognized by a tympanitic (abnormally sonorous and deep) percussion sound in compensation of heart-dulness, or in other words by a deficient apex beat, dulness of the heart sounds despite an otherwise regular pulse rhythm, even without taking into consideration the symptoms of an interstitial emphysema over the lungs, in the clavicle and the supraclavicular fossa. But then every subdiaphragmatic disease which produces an elevation of the diaphragm either on both sides or on the left side only will also cause a spatial contraction in the region of the heart. I remind the reader here of abnormal accumulations of gas in the stomach or in the

intestinal canal corresponding with a flexura coli lienalis, conditions which, indeed, often enough, give rise to the afore-mentioned subjective disturbances. Yet it strikes me that this diagnosis is often made rashly at the bedside only to make room soon afterwards for a recognition of a lethal sclerosis of the coronary arteries.

Hernia and diaphragmatic eventration may also show the same effects. (See chapter on "Pain in the Chest.")

Aneurysms of the aorta claim here especial attention even though arrosion of the anterior bony thoracic wall should not as yet be in evidence. Now to the left, or to the right, side of and also behind the sternum a dull, at times also violent, continuous "hammering" pain will become noticeable. It is more or less frequently interrupted by painful paroxysms of an intensive character making excursions into one (right) or both arms, or in the shoulders, or in the back, either of spontaneous origin or due (chiefly) to bodily exertion.

In aneurysms of the pectoral aorta the patient does not so much complain of pain, but rather of a bothersome sensation of coldness in the cardiac region.

Aneurysms of the *innominate artery* will cause the same kind of pains which radiate into the right arm or to the right shoulder.

The diagnosis should be guided in these cases by the fierce intensity of the irradiating pains, the abnormal smallness of the pulse in the arteries of the right arm and of the right carotid, eventually also by paresis resp, paralysis of the right recurrent nerve, acceleration of the pulse in the right carotid and the subclavia (and their branches) only, and by the Roentgenogram. Pulsation behind the right sternoclavicular articulation or immediately above or below it, a dulness above it or a pulsating protuberance or tumor may not yet be noticeable in the earlier stages.

Another source of pain may be found in the irritation (no matter what its origin may be, *neuralgia, neuritis*) of the left phrenic nerve, f.i., due to diaphragmatic pleuritis, invagination of the pleural membrane, compression of the mediastinal glands, etc. The typical pressure points, eventually singultus and a conspicuous bradypnea lead the way to a proper diagnosis.

Piercing or pulling pain in the region of the heart (often radiating into the back) is a regular companion of *tetanic convulsions,* at any rate when they are of a pronounced character. Tetanic contractions are here the probable underlying cause.

Persons suffering from poisoning with *perchloride of mercury* often complain of severe, continuous pains in the heart region. Stomatitis

and mercurial enteritis coupled with intense pains in the extremities give expression to this toxic neuromyositis. A patient of mine who was so afflicted died, and the post mortem revealed a high grade degeneration of the musculature of the heart.

A dull pain in the heart will often constitute the most prominent, if not the only, symptom of *chronic tobacco heart.* Anamnesis should here be decisive.

In the aforementioned ailments we can speak principally of continuous pains in the cardiac region, but in *sclerosis or syphilis of the coronary arteries* (aorta) they assume the character of *intermittent* attacks due either to bodily exercise or mental excitement, or also to spontaneous causes. In their milder form they are known as *anginoid* attacks. The diagnosis will rest on the sensation of oppression and anguish which accompanies the pain.

The kind of painful sensation experienced by the patient should strongly appeal to the meditation of the diagnostician. The patient says that he feels a tense pressure back of the sternum, or a pressing, burning pain, a feeling as if a specially hot or rather large morsel of food had slipped down the esophagus. If we find this in company with a well-pronounced sensation of anguish, we are pretty safe in deciding on an arteriosclerosis of the heart or of the

aorta or of the peripheral vessels. But if in the absence of anguish we are dealing with pains of a milder nature, the suggestion of neuritis or hysteria lies closer. Angina pectoris is not always associated with very intense pains. (See section dealing with "Angina Pectoris.")

Localization of the pains is here the important factor for the differential diagnosis. As a rule they point rather in the direction back of the sternum, not to the region of the heart, as is the case in typical angina pectoris. In other cases the dragging pain is not located in the median line behind the sternum, but on the outer edge of the superior sternum, mainly to the left, sometimes travelling to the shoulder and the arm.

The neurotic, hysterical, anginoid heart pain seeks with predilection the region of the apex, frequently somewhat without and below it, or the 2. or 4. left intercostal space. It flares up under pressure of the finger unless the focus of the pain is centered in the nipple (hyperesthetic point). It would be a mistake to reduce this kind of pain to a functional disorder of the heart, especially so if we take into consideration that nervous concomitant manifestations are not infrequently associated with anatomical impairment of the circulatory apparatus.

Sensations of anguish and oppression in the region of the heart occurring chiefly in the night time (over-early awakening), sometimes also in

the daytime, even continuous, point rather to an inferior form of angina pectoris (*anginoid conditions*). But they may also constitute (even if only in exceptional cases) symptoms of a *cerebral arteriosclerosis.* We must consider here the age of the patient (beyond the 40th year) and the other symptoms of cerebral arteriosclerosis, viz., headache, vertigo, combined with nausea or vomiting, impairment of memory, especially for events of a recent date, changes of character such as abnormal irritability, depressing moods, intolerance of alcohol, etc.

In the differential diagnosis we encounter here a difficult question. Do *anginoid* conditions in the sense of painful attacks in the cardiac region coupled with sensations of anguish point to an incipient arteriosclerosis, or are they a symptom of *climacteric neurosis?* We meet them in both instances. The answer will be found in the proper consideration of the other classical signs of the latter, viz., congestion, paresthesia, attacks of tachycardia, *Heberden's* nodules, accumulation of adipose tissue, absence of motoric pain, but improvement under bodily exercise, success or failure of therapeutic measures (nitroglycerin, erythrotetranitrate show no results).

Basedow's disease (*hyperthyreoidism*) is also accompanied by anginoid attacks. Diagnosis is assisted here by the findings of cardinal symp-

toms of this disease, struma, exophthalmos, tachycardia, tremor, abnormally increased metabolism, i.e., progressive emaciation in spite of abundant food consumption.

Paroxysmal tachycardia may also evince anginoid attacks coupled at times even with anginose pains, owing to ischemia of the cardiac muscles.

In some cases of *psychopathia* of a depressive character which has so many points in common with neurasthenia, e.g., melancholia, anginoid troubles may also appear, especially in the sense of precordial anguish. An analysis of the psychic, likewise of the somatic, condition of the patient sustains the diagnosis

In *epilepsy* these anginoid attacks bear a double significance. They serve as a sensible aura which is easily recognized because the subsequent epileptic attacks sharply clear up the situation. On the other hand, they represent an epileptic equivalent: pre- and post-existence of typical convulsions furnish the proof. In cases devoid of these attacks the diagnosis is rather difficult. As leading points may be mentioned: youthfulness of the patient, consciousness temporarily clouded during the attacks, unusual accompanying symptoms, complete physical ability of subject during the free intervals in contradistinction to psychical changes in the interparoxysmal stadium.

Anginoid conditions often serve as concomitant symptoms in the following diseases: the so-called *anatomical* or *functional* affections of the heart, acute peri- or myocarditis, acute overstraining of the heart, chronic syphilitic myocarditis, defects in cardiac stimulation, in general in every initial myocardial weakness, especially in the dropping heart, mitral heart, myocardial tuberculosis, or in chronic pulmonary affections, contraction of the heart with the pericardium, arteriosclerosis, syphilis and aneurysm of the aorta, and also in kyphoscoliosis. Irregularity, retardation or acceleration of the heart beats, dulness of sound, etc., will complete the diagnosis. Often when special demands are made on the heart's action, for instance, in exceptional cases of sudden increase in the blood pressure (vesicular crises) we may hear of complaints about pressure, pain and oppression in the region of the heart, extending to the nape of the neck, by choice in the left arm.

In insufficiency of the left ventricle, nephritis, arteriosclerosis and chronic myocarditis, these anginoids come to the foreground in the shape of sensations of anguish and dyspnea, culminating eventually in severe attacks of *cardiac asthma.* They rarely fail to make their appearance in *essential arterial hypertension.*

Sometimes they follow in the wake of pilocarpin injections, presumably by effects on the

vagus branches of the coronary artery. They are generally found also as partial symptoms in *vagotony.*

At times it will be necessary to decide whether we are dealing with a bronchial pain in an arteriosclerotic patient or an anginoid condition due to sclerosis of the aorta.

Anginoid pains, even anginose attacks, are often coupled with other cardiac affections, such as palpitation (cf. under that heading), skipping pulse, precordial anguish, oppression due to a postcecal pneumatosis of the stomach, or to swallowing air or to abnormal gas formation in the canal. These processes, no doubt, are generally formed in nervous people, whose heart action as a rule is quite faultless, yet they always give rise to doubts about arteriosclerosis or adiposity being present. I can never shake off these doubts until I find definite proof of an unmistakable elevation of the diaphragm, succeed in removing the pain by evacuation of the gas, and am convinced that motoric dyspnea is not present.

If the *elevation of the diaphragm* is due to a direct attack on the diaphragmatic muscles, or to a lesion of the phrenic nerve, or if a basal adhesive pleuritis has impaired the motility of the diaphragm, the diagnosis will be much easier. The clinical and radiologic examinations are the directing guides.

Anginoid conditions in the sense of pains in the region of the heart, sensations of anguish, palpitation, shortness of breath, may also point to a *dry pleuritis* in the precordial region. More about this anon.

A final diagnosis may meet with almost insuperable difficulties in the following conditions: fatty degeneration of the heart, in the beery heart, sclerosis of the coronary arteries. Do the anginoid conditions point to these affections or to primary myocardiac troubles? In how far and how often does nicotinism play here a rôle? Does the bedside examination reveal these conditions?

If we deal with a very sudden and highly intensive pain in the cardiac region we may be pretty sure that we are confronted by an anatomical disorder of the heart or its larger vessels or of other organs located close by, or also some distance removed from the heart itself. Functional disease is in such cases rather remote. Particulars about this will be found under "Angina Pectoris."

Leaving this affection for the present out of consideration, I wish to say that intensive pains in the region of the heart caused by acute disorders are proportionately of rare occurrence. They come under our notice perhaps more in the shape of an overstretching of the myocardium in cases of paroxysmal tachycardia.

Very sudden, fiercely stabbing pains point to acute pericarditis coupled with a very pronounced dyspnea. The pericarditis may be the result of articular rheumatism or some other acute infection (sepsis, pneumonia) or it may be the forerunner of a rheumatic fever (acute) or a partial manifestation of polyserositis. A diagnosis of pericarditis under these conditions is open to error. In my earlier years, when I was a young assistant, I fell into this error. It was really a case of pleuro-pericarditis, i.e., pericarditis externa. Most of these cases are due to some hemorrhagic infarct, but sometimes also to pneumonia in the upper lobe or to an independent pleuritis mediastinalis ant. sin. A careful study of the friction fremitus will be a decided adjuvant in the diagnosis.

These sudden pains do not often make their appearance in cases of internal pericarditis, and are therefore a fairly safe guide in diagnosing for external pericarditis.

In internal pericarditis I have observed a certain particular kind of pain: In a case of fibrinous pericarditis the patient complained of an intensive, piercing pain in the cardiac region which set in with every heart beat. It was so distinct that temporary irregularities of the heart action were marked by a simultaneous arhythmia of the pain. In other cases—of rare occurrence so far as my own experience is concerned—the

patient complains of a violent, continuous pain in the region of the heart, with excursions into the left shoulder, of painful, superficial breathing, often connected with singultus and vomiting. The cause for these phenomena may, perhaps, be found in a prominent mitral affection of the pericardial branch of the phrenic nerve or in the latter itself.

Acute affections of the aorta are apt to give rise to complaints of sudden, at times very intensive, pains in the region of the heart. I have seen only two cases of acute infectious (not syphilitic) aortitis so far in my practice (more cases are reported in French literature). In one case the patient complained of violent continuous pains behind the superior sternum, with increasing demarcation and a ringing quality of the second aorta tonus. It was a case of florid acute articular rheumatism. The second case was that of a convalesecnt from abdominal typhoid. The patient, otherwise in good health and able to do hard work, complained of pains behind the sternum. When walking they increased in violence and forced him to stand still for a while. He had no fever, but a protracted dicrotia, strongly accentuated, ringing quality of the second aorta tonus, especially in the first intercostal space to the right of the sternum.

Rare cases of *aortic aneurysm,* due to acute infections (syphilis, tuberculosis, acute articular

rheumatism), belong in this category. But it seems to me that they develop rather on the ground of mycotic embolism as spurious aneurysms in the wall of an aorta that has already deteriorated through the loss of the inner layer. As a rule they are recognized only when the aneurysm has run its full course.

Sudden very violent pain in the region of the heart accompanied by oppression, collapse and vomiting may be the concomitant symptom of a *rupture of the heart* with hemopericardium, or of the ventricular septum due to anemic necrosis, or of the rupture of a papillary muscle, or of the aorta or of the coronary artery, or of the rupture of a cardiac aneurysm, or of an aortic valve or of an aortic valvular aneurysm, or of the rupture of a pulmonary vein, the *bursting of an aortic aneurysm* into an adjacent organ (pleura, bronchus, trachea, pericardium, mediastinum, esophagus), or the *bursting of a bronchial gland* into the aorta and thence into the pericardium, or it may be the sudden formation of a dessicating aneurysm of the aorta.

The kind of pain of which the patient complains, viz., the sensation as if something had been torn in twain within the chest, and the anamnesis are strong hints of the aforementioned catastrophe.

The diagnosis of a rupture of the heart or

also of a cardiac aneurysm or of the aorta can only be confirmed by the finding of an acute hemopericardium. This would also establish the diagnosis of the bursting of an aorta aneurysm if such a diagnosis was already previously made. Rupture of the aorta leads to death much quicker than a rupture of the heart proper.

The diagnosis of a rupture of an aortic valve depends upon the proof of an acute, i.e., suddenly arising, mostly incomplete insufficiency of the valves themselves. In the spot where an aortic aneurysm bursts into the mediastinum, or into the upper vena cava or any other mediastinal organ, a painful sensation will be rarely found, and we must look for other accompanying symptoms for our diagnosis. But there are cases in which severe pains in the chest with vomiting are experienced. The pain is generally located to the right of the sternum, below the right clavicle.

In the rupture of a papillary muscle the diagnosis can never be definitely established intra vitam; for besides the sudden, overwhelming pains, we find the symptoms of an acute insufficiency of the heart which leads to death in a few hours (galoping rhythm, embryocardia, acute dilatation of the heart, absence of pulse beats, dizziness, nausea, oppression).

The diagnosis of a desiccating aneurysm of the aorta can only then be made with approach-

ing certainty when we find an aortic aneurysm in a patient who has suffered a trauma, but previous to that was hearty and showed no signs of an aortic aneurysm under medical examination, a person who never had syphilis, so that the aneurysm came into evidence, so to speak, as an acute process.

The stomach is frequently the source of sudden, vehement pains in the cardiac region. There may be a perforation of a *ventricular ulcer* into the peritoneal cavity, or an acute peritonitis emanating from the stomach, or a *volvulus* of the stomach or a *gastritis.* We may be quite sure that no subjective pains will arise from these conditions in the epigastrium or in any other place in the abdomen, except and exclusively in the region of the heart. A definite diagnosis may be made if we have previously already examined the patient, and if the anamnesis has informed us about the existence of a ventricular ulcer. Where this is not the case and where we are confronted by a suddenly arising volvulus of the stomach, only the thought of the possibility of any one of these affections of the stomach, together with a local examination, can lead to a resulting diagnosis, and even then the consideration of an angina pectoris may offer complicating difficulties. This latter point craves serious thought, because in

many affections of the stomach the existing pains are accompanied by sensations of anxiety.

Destructive affections of the *gastric parietes,* foremost a ventricular carcinoma or ulcer, may create sudden, most incisive pains in the region of the heart, especially when they burst into the pericardium. The acute formation of a hemo- or hemopneumo-pericardium, aided by the preceding anamnesis and clinical examination, will facilitate the diagnosis.

The sensation of anguish, principally the so-called precordial anguish, i.e., oppression and constriction in the cardiac region and behind the sternum, often coupled with a choking sensation in the throat culminating in real death agony, constitutes one of the characteristic signs of *angina pectoris* (*stenocardia*). There are cases in which the sensation of anguish and oppression in the chest is of such a high degree of intensity, yet without pain, that the patient collapses and death intervenes. But in the majority of cases we come across a whole list of valuable symptoms: pain behind the median sternum following the line of the heart, radiation into the left arm, especially on the side of the little finger, likewise in the left side of the nape of the neck itself, and also of the face, not always very intense in its nature but rather secondary to the sensation of anguish which becomes well nigh unbearable; besides these there is pallor, the

Hyppocratic facies, clammy cold sweat, absolute rigidity of the body (the patient stands stock-still, or if the attack comes in the nighttime he sits up rigidly and immovable on the edge of the bed), avoidance and fear of deep breathing (in some cases, on the contrary, deep respiration brings relief), accelerated, but sometimes retarded, heart beats, overmastering the attack by a special physical effort, sometimes by abdominal straining during defecation, passage of gas either through the mouth or per rectum. Who has not observed these signs in his bedside practice?

With the whole complex of these symptoms before us, the diagnosis cannot go amiss. But I wish to lay stress on the fact that in spite of these symptoms angina pectoris is at times by mistake diagnosed as an affection of the stomach with *gastralgia,* because the attack may yield either spontaneously or through the means of a small dose of bicarbonate of soda to a sudden act of belching or eructation.

If a stenocardia has been correctly diagnosed, then we must inquire into the last cause of the attack: an acute, or an acute infectious, pericarditis, or a chronic myocarditis may bring about anginose attacks, or a concretion between heart and pericardium, or a stenosis of the aortic or mitral ostium, an embolism or thrombosis of the coronary artery, or chronic nicotinism may

be at the bottom of these attacks. Tabetic heart crises or purely hysterical or vasomotoric conditions may evince them. But the most frequent cause will be found in a sclerosis of the ascending portion of the thoracic aorta where the coronary arteries branch off, or in a sclerosis of these arteries themselves, i.e., endaortitis or endarteritis of syphilitic origin.

Due consideration of age and anamnesis will in all probability decide between *syphilitic endarteritis,* resp. *endaortitis* and *arteriosclerosis* as underlying cause. Age below forty years points to syphilitic endarteritis. But it is not an infallible sign, for our present knowledge teaches us that arteriosclerosis may also occur before the fortieth year is reached; in fact, at almost any time of life. Neither is the anamnesis a definitely deciding factor, even in conjunction with other coexisting metaluetic diseases, chiefly of the central nervous system (tabes, taboparalysis, cerebrospinal syphilis), because we should bear in mind that arteriosclerosis has a basic predilection for syphilitic affections.

The *Wassermann* reaction, although of great significance, is not always an absolutely reliable indication. It is true that in most cases this disease points to syphilitic influences. But I am not fully convinced that it may not be connected also with a true arteriosclerosis of the aorta or of the coronary arteries, and that, after all, an-

gina pectoris may be the exciting factor of this disease. In such cases of doubt we can only find assistance in the resulting efficacy of anti-syphilitic treatment.

Whether *embolism* of a coronary artery is the cause of anginose affections can be easily decided when the existence of an endocarditis of the aortic valves has been definitely established and when the patient, mainly of youthful age, quickly succumbs under the anginose attack. On the other hand, when the embolism originates from an atheromatous ulcer at the root of the aorta, the etiological diagnosis of the lethal anginose attack will have no higher value than in cases where a *thrombosis* in one (chiefly the left) coronary artery was the basic cause. When the outcome is not fatal a differential diagnosis of an angina pectoris on the basis of an arteriosclerosis in one of the coronary arteries at the time of the attack seems to me impossible. The following conditions may point to thrombosis as the exciting cause: unusually prolonged pains, high degree of cardiac weakness, quick but thin pulse, embryocardia, emaciated appearance, impaired diuresis, faint heart beats, splitting of the first apex beat, acute dilatation of the heart and acute vascular congestion.

If later on a partial cardiac aneurysm is recognized, the diagnosis of an acute thrombotic or embolic occlusion of one of the branches of

the coronary artery may be regarded as certain. Furthermore, when a sudden anginose attack is immediately followed by a feverish, at times only fibrinose, but also exudative pericarditis—in some cases this manifests itself during the first two or four days as a continuous sensation of pain or oppression in the region of the heart—it may be an indicator of an acute occlusion of a coronary artery branch with a subsequent necrotic infarct and the final formation of a partial chronic cardiac aneurysm. Such cases are by no means rare. But the diagnosis is infallible. I remember just such a case. The patient succumbed rapidly. The post mortem did not show an "epistenocardiac pericarditis," but a very pronounced sclerosis of the ascending aorta and its arch, with a number of atheromatous ulcers, one of which had burst through the wall of the aorta. This produced a periaortitis at the root of the aorta, followed by acute pericarditis. The anginose attacks were due to well-marked changes in the switch of the coronary artery. No traces of a sclerosis or occlusion in the coronary artery or in its branches could be discovered. Myomalacia of the heart was absent.

Some authors claim that an *acute infectious aortitis* may give rise to typical attacks of angina pectoris, but I have never seen such a case.

In recurrent attacks of angina pectoris, espe-

cially when the anginose pains with irradiation continue for several days, i.e., when a status anginosus exists, it is well to think of an *acute pericarditis* as the hearth from which the trouble proceeds. It is claimed that even a *dry pleuritis* in the precordium will induce typical anginose pains, i.e., pains in the region of the heart with excursions into the left arm, sensation of fear, palpitation, excited heart action and dyspnea. I have never had the privilege of seeing such a case, although I have treated quite a number of cases of extrapericardiac pleuritis with pains in the region of the heart.

The problem offers greater difficulties when angina pectoris is based upon a *concretion between the heart and pericardium.* In this condition the coronary arteries are held in a vicelike embrace, the circulation is impeded and anginose pains are the result.

But such a diagnosis can only be established when absolutely unmistakable symptoms of a concretion are found.

In *stenosis* of the *aortic* or *mitral ostium* we may observe angina pectoris either in a well pronounced form, or in the shape of a mitigated anginoid condition, probably as a symptom of an insufficient blood supply in the myocardium by increased activity. The diagnosis must rest upon the finding of the exciting cause. But we must not overlook the fact that painful attacks

similar to a coronary angina in mitral stenosis may also be the sequel of a consecutive sclerosis of the pulmonary artery. I shall revert to this later on.

Anginose attacks will also crop up in chronic inflammatory or degenerating diseases of the *myocardium* without affections of the coronary artery. To distinguish these rare cases from true sclerosis of the coronary arteries even with approaching certainty intra vitam seems to me impossible.

Chronic nicotine poisoning should be suspected in all patients, especially in females, who present one or more anginose attacks. We should be guided here by the other coexistent symptoms: palpitation, tachycardia, arythmia, especially in the sense of extrasystolic conditions, cold sweats, tremor during the attack, retrobulbar neuritis, dyspeptic troubles, intermittent distension of the thyroid glands, nocturnal palpitation of the heart. And yet all these symptoms may not constitute a definite proof for the toxic genesis of the anginose attacks and for a good prognosis. Nicotinism is a predisposing factor in arteriosclerosis, particularly in the coronary arteries. A genuine angina coronaria may already exist and the first visible attack may be lethal although the patient has experienced no previous inconvenience.

On the other hand let us remember that

stenocardia in an inveterate smoker may also be due to sclerosis or syphilis of the coronary arteries not necessarily connected with anginose attacks, but that the vascular cramps may be superinduced by the immoderate use of tobacco. If abstinence from smoking restores the heart to normal activity we have a definite proof of the toxic origin of the anginose attacks.

In rare cases of excessive use of *coffee* and *tea* anginose attacks will appear. Palpitation of the heart, tremor, and vasomotoric symptoms differentiate them from genuine angina pectoris. Even if these attacks are accompanied by bradycardia, the diagnosis for the aforesaid etiology is contraindicated. A true angina pectoris exists combined, like all other toxic anginas, with tachycardia of a moderate degree, but in the severest cases with bradycardia.

Among the toxic anginas we must count the rare cases of anginose, or in most instances angi*noid manifestations arising from chronic morphinism* or caused by *inhalation of* CO_2, e.g., after carbonic acid baths or by *tuberculin injections.* The diagnosis is governed by the anamnesis.

Toxic forms of stenocardia are also found by some authors in *lead poisoning,* in *gout* and in *diabetes mellitus.* I will not deny that such toxic stenocardias exist in gout, for instance, as vicarious attacks of an articular nature. But so far as my own clinical experience goes, I

speak with the utmost reserve. I remember two cases of gout—one a man, thirty-six years of age—in which I observed typical anginose attacks. In the heart and vessels I could find nothing abnormal. These conditions misled me into a diagnosis of gouty, i.e., toxic angina pectoris. But both patients soon succumbed to a renewed attack of stenocardia. Clear cases of sclerosis of the coronary arteries. In a third case, I insisted on a diagnosis of angina coronaria. Three of my colleagues stuck to uratic angina pectoris. The patient died of a classical anginose attack in a tepid bath.

In *tabes dorsalis* syphilis of the aorta or of the coronary arteries may bring about anginose attacks. Insufficiency of the aortic valves goes together with tabes also. This last named combination alone is often an indication that the tabetic condition originates at the point of division between the coronary artery and the aorta which latter is either affected by syphilis or arteriosclerosis. But this is a mere opinion, because true angina pectoris is of rare occurrence despite the frequency of insufficiency of the aorta in tabes.

On the other hand there are cases of tabes dorsalis in which the anginose attacks assume the full meaning of tabetic crises. The existing causes, no doubt, will assist in distinguishing between these two subdivisions.

The true stenocardiac attack arising from an affection of the coronary arteries is primarily superinduced by some physical exertion (running for instance after a train or street car, mounting steps, or a brisk walk after a heavy meal) or by a sudden change in the temperature (going from a warm room into the cold open air), preceded by a faulty diet or a sumptuous dinner or some psychic excitement. But the tabetic crisis is independent of such causative factors and comes into existence with a leap, as it were, and without apparent reason.

Basedow's disease is likewise guilty of at times most severe retrosternal pains shooting off into both the upper extremities. These characteristic and obvious symptoms should remove all barriers for a proper diagnosis.

Nervous or *hysterical angina pectoris* is distinguished from a true coronary angina by the peculiar behavior of the patient. Both, however, share that important symptom of centrifugal progression into one or the other arm, likewise the sensation of oppression behind the sternum and in the neck. In coronary angina the patient remains perfectly still; in nervous angina he groans and cries out with pain, is extremely restless, and makes startling movements. Still these distinguishing manifestations do not always serve as deciding factors. I remember a case of lethal angina pectoris in which

the patient rolled about the floor in most terrible pains. Other patients, especially males, toss about in bed in the most restless fashion, constantly wailing and jabbering and gesticulating until death puts an end to the agony. *Rosenberg* relates cases of lethal coronary angina in which horrible contortions of the face and even *arc de cercle* were observed.

The hysterical patient often describes the pain as a sensation of stretching, a feeling of fullness in the throat emanating from behind the lower sternum. But in coronary angina the complaint is more of the sensation of compression as if a heavy weight were lying on the chest, as if the breast were held in a vice, also the feeling of intense burning especially in the region of the manubrium of the sternum, due, at least in part, to reflex contraction of the intercostal muscles.

Pain radiating into the region of the left ulnar nerve, even into the fingers, seems to point more to anatomical than to nervous angina pectoris.

The true anginose attack occurs, at any rate in the beginning of the disease, generally at intervals of weeks or months, preferably in the night time. The individual attacks are of short duration. In nervous pseudoangina we encounter again the noisy, theatrical element. The attacks are more frequent, 30 to 40 follow each other in rapid succession, mostly in the day-

time and wind up in tears and sighs of a convulsive character. The basic element will at times assist us in arriving at a satisfactory conclusion. In nervous angina pectoris the attack does not mature from a bodily movement, but it comes on top of a psychical emotion—in true angina pectoris this may be even the exclusively provoking element—which is principally conditioned by a special, definite external cause, e.g., when entering a public place of meeting, a church, a theatre, a hall filled with people or when attending a social function. The object of the patient is to have all eyes turned toward her or himself.

The blood pressure during and between the attacks, it seems to me, is a useful point of differentiation. In true angina pectoris there is generally a marked rise, but between the attacks a depression. In the nervous attacks the pressure often rises considerably during the attacks, but no marked hypotension is noticeable in the intervals.

The presence of bradycardia during the attack points strongly to a true angina pectoris. Abnormal difference between the rectal temperature and that in the armpits (0.6 to 1.0° and above in favor of the former) speaks for true and against neurotic angina pectoris.

Other conditions which are foreign to cases of coronary angina, such as tachypnea, true

hysterical or nervous stigmata, abnormal vita sexualis point to hysterical or nervous angina. To avoid dangerous errors it will be well to remember that the so-called hysterical globus is universally accepted as an important hysterical stigma. But a similar sensation may also be observed in true coronary angina. Some patients complain that in stenocardiac attacks they experience the feeling of constriction in the fauces resp. in the upper esophagus. This has been erroneously accepted by some as a globus hystericus, and in consequence a false diagnosis of angina pectoris was made.

A special subdivision of nervous angina is what might be called *anginophobia,* very prevalent among the members of the medical profession. There is pressure, pain, so to speak, of a bursting character in the region of the heart and behind the sternum, also irradiation, oppression, palpitation, also vasomotoric manifestations, psychic affections, especially when recalling the picture of certain morbid symptoms, but hardly ever after physical strenuous efforts.

In this negative organic state, in this frequent persistence of pressure sensation, in the recurrent hyperesthesia in the region of the apex, in the psychical provocation, in the fact that the patient admits to be in the ban of a morbid conception that forces him to anticipate a threatened attack

when starting some physical movement, but absent when his attention is averted elsewhere, in pronounced psychic depression, or hypochondriac moods, difficulty in breathing, also in certain minute manifestations which are in absolute contrast to those of a true angina pectoris, there is a wealth of determining factors for a correct diagnosis.

I would like to suggest here a classification of *dyspragia* as it appears in different parts of the body (extremities, heart, intestines, brain). In the first place I would put the purely functional form which is caused by absolute vasoconstriction in which the walls of the vessels remain perfectly intact (purely nervous or in nicotinism), and in the third place the arteriosclerotic form without vascular contractions. In the second place there would appear that form in which the convulsive attacks based on vasoconstriction are provoked by the arteriosclerosis itself; I mean to say, the existence and recurrence of which is conditioned solely in the anatomical defect of the vessels and the subsequent paroxysmal vasoconstriction. Then there is a fourth form also coupled with arteriosclerosis with reflex actions on the vascular nerves by way of abnormal stimulation, yet not to such an extent that the morbid manifestations of dyspragia are thereby produced. Only when an additional extraneous agent which increases the

stimulating action of the vasoconstrictors forms a union with the arteriosclerosis are the manifestations of dyspragia elicited and maintained until the aforesaid extraneous agency is removed. So far as prognosis is concerned such a classification should prove of importance. For diagnostic purposes I think it would be a profitable graduation. Of course, in some cases only the final outcome, i.e., the gradual disappearance of the manifestations would bring the solution, although the anatomical disease itself would continue to exist. But we should bear in mind that the retrogression of morbid symptoms may be effected through collateral channels, especially in those cases which originate exclusively from anatomical affections.

In the differential diagnosis between anginophobia and a true coronary angina we may also bring into service the observation that in the former the sensation of fear consists rather of the troublesome suspicion of a possible disease of the heart and more so than of a real feeling of oppression. This may also be the case in nervous hysterical angina. It is a golden rule to be exceedingly conservative when making a diagnosis of absolute nervous angina pectoris in patients who have gone beyond the age of forty. In most cases we are dealing with a combination of a nervous component with an anatomical

arteriosclerosis if not with an atypical angina vera.

Attacks of nervous angina pectoris based on sexual neurotic conditions and marked by oppression, pain in the cardiac region, anguish, and even final collapse, can very often be reduced to excessive masturbation. Other points that will assist in a correct judgment are palpitation of the heart—a symptom wholly foreign to genuine angina—also the localization of the pain not behind the sternum, but rather in the cardiac region proper where the skin especially around the apex often has an hyperesthetic appearance.

In *vasomotoric angina pectoris* it will be noticed that the patient is extremely restless and finds relief from the pain by constantly moving about, as is the case in hysterical angina. The painful attacks are frequently combined with palpitation of the heart and are preceded by the appearance of peripheral angiospasms, pallor, feeling of cold or acrocyanosis of the extremities and of the face, paresthesia in the former, giddiness, ague, syncope and collapse, generally relieved by cooling applications. Careful observation of the blood pressure is a useful adjunct in the differential diagnosis: in true sclerosis of the coronary arteries a marked rise in the blood pressure, rarely a depression; in the vaso-

motoric form the blood pressure is but slightly raised, if at all, but not over 20 *mm.* Hg.

Angina pectoris following in the wake of acute, infectious, and also at times chronic myocarditis without affections of the coronary arteries may easily be recognized by its anginose manifestations of a clinically inferior character. It very likely arises from an irritation of the cardiac nerves, by way of some myocardial disorder, in the same manner in which the same trouble springs in stenocardia from an acute pericarditis.

Mild attacks of angina pectoris in which the patient complains only of pressure in the cardiac region and behind the sternum when engaged in bodily movements or in mental action may be observed in myodegeneration of the heart without giving rise to a notable sclerosis of the aorta or the coronary arteries. At any rate we have no clinical proof of it. What arrests the attention is rather the obvious dullness of the heart sounds even when the patient is leaning over in the front, the smallness of the peripheral pulse and a splitting of the heart sounds. The prognosis is much more possible for a long life if good care is taken of the heart. In these cases the thought always lies near that we are rather dealing with an overstretching of the heart during some physical exertion.

In some cases of *paroxysmal tachycardia* the

patient ocmplains of severe pains behind the sternum with extensions into the arm, obviously anginose attacks reduceable to an ischemia of the myocardium. Although we find acceleration of the pulse as an accompanying symptom in painful attacks of true angina pectoris, this should not be a disturbing element in making a proper diagnosis of paroxysmal tachycardia in which the pulse rate is much higher (160 the minimum, a rate never obtained in coronary sclerosis).

It is worth the while to emphasize here the fact that attacks of coronary angina are sometimes accompanied by symptoms which are apt to lead to errors of a serious character for the patient as well as the attending physician.

There are cases of true angina pectoris of the severest type in which the sensation of anguish is totally wanting. But it would also be an error to infer from the presence of this sensation that the attack rests upon an anatomical basis.

It cannot be disputed that the true stenocardiac attack is elicited by some physical movement. On the other hand there are cases of true angina pectoris in which the attack does not follow a bodily motion, not even a physical overexertion, but in which, the same as in nervous or hysterical angina the spell comes only during the night abruptly shaking the patient out of his sleep. Other patients suffer from the

evil turn by day as well as in the night, the nocturnal attacks generally being of a longer duration. These latter cases bear unmistakable signs of coronary sclerosis.

Furthermore, there are cases in which no palpable reason for the occurrence of nocturnal anginose attacks can be discovered, e.g., a late, unusually heavy dinner. They come spontaneously without any recognizable provocation, and physical exercise seems to have a beneficent effect on the patient. And yet the disease is a true coronary sclerosis beyond a doubt. Two of my patients died during such a nocturnal attack. The same observation has been made by others (*Cushman, sen.*)

If the attacks are of a milder type it might be proper, from the therapeutic as well as from the diagnostic standpoint, to advise the patient to take some food during the night which might forestall a possible attack. If the result is favorable, as no doubt it will be, it would be a proof that the nocturnal attacks coming on during the resting period are due to ischemia of the heart derived from lessened irrigation, whilst bodily effort stimulates the circulation in the cardiac muscles and thus aborts the attacks.

The reverse is the case in *dyspeptogenous angina pectoris,* in which the intake of certain food stuffs, or of any kind of food, is the signal for the attack, and bodily exercise is neutral

in its reaction. In this peculiar etiologic factor the clinician may find a hint for the diagnosis; likewise, according to French authors, in the milder character of the attack, its longer duration and less dangerous moment; also in the undisputed dilation of the heart during the attack and in the accentuation of the second pulmonary sound. But I share with *Krehl* his doubts on the subject. What I have seen myself were true cases of arteriosclerosis in which acute dyspepsia was the provocative cause. *Krehl* attributes the attacks to nervous supersensitiveness of the heart. As a rule I scent especially in older patients a coronary sclerosis as the exciting focus.

The diagnosis of an hysterical angina pectoris due to psychical conditions is sometimes used as a factor in the differential diagnosis of a stenocardia arising from a sclerotic affection of the coronary arteries. But I hesitate to subscribe to such an opinion, because I have seen cases of true stenocardia which beyond a doubt originated from psychic emotions rather than from any bodily exertion.

Accompanying polyuria, urina spastica, is frequently considered an important symptom of nervous, hysterical angina pectoris. But this may also be misleading. I remember one of the gravest cases of coronary angina with polyuria immediately after the stenocardiac attack.

Polyuria parallel with salivation is not an infrequent obvious reflex action of a stenocardiac attack.

The influence of cold and palpable vasomotoric disturbances preceding the attack, paresthesia, shivering, pallor of the face and of the extremities may be looked upon as decisive symptoms in vasomotoric angina pectoris. But even here the utmost precaution must be exercised. Cold, to be sure, is one of the principal causes. A sudden change from the atmosphere of a well heated room into the cold temperature out-of-doors, or the cold clammy sheets of the bed may provoke an attack. But what seems to me of still greater import is the fact that vasomotoric phenomena may also come before an attack of true coronary angina. Not long ago I saw a patient with the whole characteristic symptom complex of stenocardia. The post mortem showed the presence of a grave mesaortitis and mesarteritis.

Vasomotoric symptoms may also be observed in both the upper extremities, or perhaps only on one side of the body, or in everyone of the four extremities; and again there may be a periodic constriction of a bronchial artery as against the contralateral, even a local asphyxia, eventually confined to one side only. And yet there is a true angina pectoris.

Vertigo or intermittent unilateral blindness

when associated with stenocardiac attacks, are accompanying vasomotoric symptoms.

It seems to follow that at the very best only the whole complex of vasoconstringent symptoms in its entirety, and then only with prudent reserve, may be considered as indicating vasomotoric angina, but when it is confined to the narrow limits of certain corporeal regions—mainly the left upper extremity—it cannot serve as a criterion against a serious coronary angina.

Certain morbid manifestations which suddenly make their appearance during violent weather disturbances, tornadoes, simooms, severe thunderstorms, are frequently looked upon as mere nervous or vasomotoric disorders. But my own experience has led me to the firm conviction that they are occasioned by these disturbances themselves.

There is a series of *painful attacks* which present themselves in a manner *similar* to those connected with angina pectoris. In the front rank are the diseases of the *circulatory apparatus* as well as of more distant organs. I refer here first to those diseases which have already been mentioned as the source of very intensive pains in the region of the heart. In a rupture of the organs referred to in that chapter the location of the pain is often falsely placed in the precordial region, sometimes in the epigastrium or the abdomen. In consequence it is errone-

ously taken for a lethal angina pectoris. Only the proof of an existing hemopericardium or an acute insufficiency of the aorta can protect us against such an error when the pain is properly localized as existing in the precordium and not behind the sternum.

Attacks similar to those in true angina pectoris—mostly of a recurrent nature—are also produced by other diseases, especially by *sclerosis* of the *pulmonary artery.* But then the patient complains of, besides anguish and oppression, pain in the cardiac region with a feeling as if this pain were penetrating away down into the chest, yet without irradiation into the periphery which is so charactertistic of coronary sclerosis. There is but little shortness of breath or none at all, but a high grade cyanosis, in contradistinction to pallor in angina, or to the slighter degree of cyanosis in stenocardia springing from mitral stenosis. The differential diagnosis must be determined by the remaining symptoms, viz: generally existing abnormally large dilatation of the right ventricle with simultaneous primary stenosis of the mitral ostium, the unusual resonnance of the second pulmonary sound, dilatation of the pulmonary artery, and eventually a relative insufficiency of the pulmonary valves.

The same conditions exist in those isolated cases of pseudoanginose attacks which arise from thrombosis of the pulmonary artery or of the

right ventricle. Notice the solitary deepseated pain associated with the feeling of anguish, cyanosis, rapid asphyxia, all of which affect the differentiation of coronary angina. But when the beginning and the progress of such a thrombosis is only gradual we may observe repetitions of the pseudoanginose attacks coupled with cyanosis and dyspnea and even with distinct dilatation of the right ventricle. The accompanying cyanosis and dyspnea, the deepseated pain devoid of peripheral radiation, and regular dilatation in the right region of the heart are, indeed, the guiding points in the differential diagnosis.

Embolism of the trunk of the aorta leads to pain in the cardiac sector. Accompanying dyspnea and cyanosis, deepseated pain, the missing participation of the affected side of the thorax in the breathing rhythm, bloody sputum, and above all the conspicuous frequency and smallness of the arterial pulse are signs full of meaning for the diagnosis.

Painful attacks have repeatedly been observed in innate *pulmonary stenosis.* The determining factors in the diagnosis of this disease are: systolic crepitus of a whirring character in the second intercostal space to the left of the sternum, in the left interscapular fossa and possibly in the left carotid, peripheral cyanosis, eventually hypertrophy of the left ventricle, tuberculous lesions in the lungs, and pre-senility age.

Tumors in the right ventricle or the *left auricle* deserve mention here. But I will say at once that the diagnosis in these cases will always be more or less guesswork. Perhaps, the most distinguishing feature in this connection is hemorrhagic pericarditis which so frequently and rapidly accumulates again after a paracentesis. In addition there are spasmodic attacks of suffocation when changing the posture of the body, metastasis of *Troissier's* ganglion, inexplicable embolism, pronounced cyanosis mostly without dyspnea when the tumor is in the right, but bloody sputum if in the left auricle.

Indurated mediastinitis may in my opinion occasion similar painful attacks. Pains behind the sternum may appear during a walk, only to disappear again when the patient sits down to rest, likewise painful dysphagia. The most prominent adjuvants in the diagnosis are a strongly marked *Oliver-Cardarelli* sign and the Roentgenogram.

Mediastinal neoplasms, e.g., a lymphocarcinoma may at times simulate an angina pectoris, for in this state the patient also complains of retrosternal pain experienced during walking or accentuated by more lively physical exercise. This pain is undoubtedly due to the dragging of the mediastinal tumor in the more vigorous respiratory movements of the chest.

Pseudoangina with cyanosis of the higher type

may also be found in *thrombosis* of the *superior vena cava.*

Perforation of an *adjacent organ* into the *pericardium* will induce sudden, very violent painful attacks. Sensation of anguish and collapse with lethal results often follow. If the perforated organ is cavernous (pulmonary cavity, neoplasm of the lung, carcinoma of the esophagus) we have before us a pneumopericardium, with classical symptoms, especially in auscultation; otherwise it is pericarditis which in connection with the initial pains should facilitate a definite diagnosis unless the basic disease was never fully recognized.

Among the diseases of distant organs I remind my readers here of the perforation of an *ulcus ventriculi,* or an acute *peritonitis* originating from the stomach, i.e., *gastric volvulus.* In these cases we may even come across a very intensive sensation of anguish coupled with acute overwhelming pains in the region of the heart resembling in every detail a typical attack of coronary angina. But when we consider that in angina pectoris the pain is localized rather behind the sternum and less in the cardiac region, and concentrate our attention on the pressure and hammering sensation, the tension in the epigastric abdominal wall, occult hemorrhages in the stools, etc., we shall be able with the aid of the anamnesis to arrive at a definite diagnosis.

If the attack is accompanied by vomiting, once only or repeatedly, I would look upon this symptom as an indication against rather than for angina pectoris, unless the patient has shown signs of cerebral or syncopic complications. In such cases vomiting is often enough a concomitant symptom in angina pectoris and of no further value so far as the differential diagnosis is concerned. Furthermore, if vomiting occurs in the intervals between the attacks as well as at the time of the attacks themselves, this would likewise render the symptom worthless for differential diagnostic purposes, for it would at the utmost only indicate a catarrhal gastric congestion arising from the steadily increasing insufficiency of the heart in stenocardia. Of course, in such a case there would be other admonitory symptoms of cardiac insufficiency present such as tachycardia, abnormal weakness of the heart beats and of the pulse, embryocardia, eventually also dilatation of the heart and additional manifestations of congestion. Blood pressure, if observed, will also be a guiding symptom. In angina pectoris it is generally higher, in peritonitis it shows a downward tendency.

If a patient comes to you complaining of a sudden, perhaps, recurrent pain of a spasm-like, crushing pain in the region of the heart and behind the sternum, sometimes even, it is said,

with a radiation in the left arm and in the left trigeminal region associated with feelings of anguish, then think of the possibility of an existing *cholelithiasis.* The pains sink deep into the epigastrium and into the right hypochondrium with excursions into the dorsal region. There is also distension of the liver, tenderness in the notch of the gallbladder, febrile attacks with periodical enlargement of the cystic bile ducts. When such conditions come under our observation it is wise to follow this procedure: if there is cardiac pain resembling stenocardia make a thorough examination of the liver, especially for engorgement under palpation and percussion, sensitiveness to pressure in the region of the notch at the acme of the inspiratory act and other signs of a gallbladder colic; do not overlook an approaching chill, a rise in the body temperature, irradiation into the dorsal region, hepatic zones of the skin, and urobilinuria. If you do, you will avoid missing the correct diagnosis of an angina pectoris situated ad portam hepatis, and in error bring in a verdict for anginose or pseudoanginose attacks when it is a case of hepatic colic.

It would likewise be an error to look upon a lesser and brief rise in the temperature obtaining during an attack as a contraindication in the diagnosis of an angina pectoris, for it is not an impossible factor in these affections.

Myocarditis and myomalacia are by no means exceptional offsprings of a diseased heart, not to speak of epistenocardiac pericarditis.

The diagnostic situation may become more complicated when we have to deal with a patient who complains of periodical very severe pains behind the sternum and who upon examination shows an insufficiency of the aorta. Here we are apt to find a slight dilatation of the left ventricle, a sudden nocturnal attack of very violent pains behind the right hypochondrium, thence ascending behind the sternum to the level of the right mamilla. The pains may settle in this region and persist with the utmost intensity; they may become intermittent or remain for hours in the retrosternal circumference. I have such a case in mind. One of the attending physicians diagnosed gastric colic; another, vascular pains; a third, achylia of the stomach; and a fourth, chronic nicotinism. But suddenly one of the attacks was followed by an attack of icterus with pronounced bilirubinuria which lasted four days. This led to a final diagnosis of liver colic. The patient had incomplete insufficiency of the aorta, a positive *Wassermann* —he once had a chancre—, the liver was disproportionately enlarged and the spleen was distended. I attributed the nightly attacks of pain to *syphilis of the liver.* The absence of motoric pain, the long intervals between the attacks for

months at a time, the statement made by the patient that flatus per anum always relieved the distress, the fact that the attacks often lasted for hours and were without the sensation of anguish, the engorgement of the liver and of the spleen and the almost exclusive arrival of the attacks in the night time, should have been sufficient indications for correcting the diagnosis of "liver colic."

There is another situation in which erroneously a false diagnosis of an impending abdominal angina pectoris may creep in. A patient, seventy years of age, came to me complaining of sudden sharp pains in the epigastrium diagonally across the abdomen, travelling quickly up behind the superior region of the sternum, followed by oppression and dyspnea accompanied by rasping sounds in the chest and expectoration of a watery, frothy sputum. My first thought was of angina pectoris with cardiac asthma, resp. pulmonary edema, especially so as the patient bore every evidence of a severe arteriosclerosis and very strong dilatation in the left ventricle, besides a muscular mitral insufficiency. But a more mature analysis of the symptoms brought me to the conclusion that the pains did not originate from an angina pectoris at all, but rather from a *peracute congestion of the liver,* for the liver appeared to be enlarged and the pain in it on pressure resembled in localization

and character in every detail that pain which always sets in with the attack. Of course, the diagnosis is much easier if one has the opportunity to watch the patient during the attacks as well as in the intervals and observe the rapid fluctuations in the size of the liver and all the other symptoms in this congested organ (also urobilinuria). I have seen three such cases in which there were, however, also pains in the left epigastrium and behind the sternum as symptoms of the peracute engorgement caused by preceding physical overexertion. I was enabled to make a correct diagnosis only by a close study of the patient during the whole time that the pain lasted. It is of interest to know that in all these cases—they were men over fifty years of age with moderate arteriosclerotic myocarditis—the pains never set in except immediately after brisk bodily movements. With rest in bed they disappeared again within three or four days, as did also the engorgement of the liver.

A similar localization of the "liver pain"—frequently it is rather of a mild and not colicky character—seems to occur in the morbid affection of the *left* lobe of the liver, be this condition of a concomitant or independent nature. It has been recently described in a case of acute engorgement due to an acute *spirillosis of the liver* (*Plaut-Vincent*), a concomitant symptom of *Plaut-Vincent's angina.*

But not only a hepatic colic but a painful attack provoked by an acute necrosis of the pancreas or else by an acute *pancreatitis* may be localized by the patient in the cardiac region and in consequence erroneously attributed by the attending physician to angina pectoris. But in such a case the diagnosis of an acute affection of the pancreas should be made without difficulty on the ground of rise in the temperature during the attack, pressure and throbbing sensation in the epigastrium, nausea, vomiting, bulging of the epigastrium and possibly in the region of the cecum and ascending colon while the rest of the abdomen shows no signs of distention but is rather sunk in, and finally and especially the acute collapse.

Among the chronic affections of the pancreas it is principally *carcinoma* which gives rise to violent crises of pain. These may very well wear the guise of an angina pectoris when reaching up into the cardiac territory, which, however, is a rather unusual coincidence. But the absence of irradiation in the arm, that fan-like extension of the pain over the entire abdomen and even into the sacrum, together with the other typical symptoms of a pancreatic disease should proffer the key to the solution.

We have already mentioned perforation of a tumor into an adjacent sphere and also volvulus ventriculi as causes of very intense pain.

It is meet to add here that other *diseases of the stomach* may elicit pain in the cardiac and in the retrosternal region, with irradiation into both arms, and that this pain may be easily mistaken for an anginose attack.

I have in mind here that form of angina pectoris which is looked upon by some authors as a reflex action of the stomach. I have never seen such a case myself. The cases of purely nervous dyspepsia with secondary nervous angina pectoris cannot belong here. Neither can those of purely secondary cardiac neurosis in which strong palpitation, arythmia, oppression and the feeling of anguish are the prominent symptoms, whilst pain is of subordinate import. Of course, there are other points to be taken into consideration, such as anginoid, similar to those in stenocardia, also with irradiation in the left arm, in company with the rest of the symptoms already enumerated. These manifestations may show up after a heavy meal and disappear again with a proper regulation of the digestive apparatus by the aid of an emetic or the stomach pump. Ravenous eating, insufficient mastication, overloading of the stomach seem to be the essential causative factors for these anginose attacks. But in my opinion they do not seem to bear the convincing stamp of an existing reflex angina. I am inclined to attribute them to a preexisting weakness of the heart or to an

affection of the aorta or of the coronary vessels, conditions which may easily give rise to painful manifestations when spurred by an acute disturbance in the digestive organs.

But I admit the possibility that in *ulcus ventriculi*—even without perforation—pain may rise from the epigastrium upwards behind the sternum with excursions into the left or into both shoulders or also into the left arm. The pain may be the product of bodily exertion, especially when an adhesive perigastritis is present, the same as happens in a true angina pectoris. Bicarbonate of soda and subsequent belching promptly relieve the situation. Our decision will be supported by the proper consideration of local muscular tension, sensitiveness to pressure in the epigastrium in ulcus ventriculi, local hyperesthesia of the skin, *Boas's* pressure point, vomiting — in stenocardia only under certain conditions—, examinations for occult hemorrhages and the Roentgenogram. The objective finding of an arteriosclerosis should be used with the utmost discretion, because this condition materially advances the formation of a round tumor in the stomach.

Similar conditions prevail in rare cases of *carcinoma* of the stomach. There also the patient complains of pains behind the upper sternum aggravated by walking or physical exertion almost exclusively. If however, an

arteriosclerosis, especially of the aorta is also present, the difficulties will be considerably increased and only the most careful examination of the gastrointestinal tract together with the anamnesis proffer the desired help.

Ulcus and carcinoma ventriculi lead to angina-like pains much sooner when they are associated with *pylorostenosis.* Pylorostenosis may at any time resemble stenocardia on account of its painful attacks. In these colicky affections the patient is apt to complain of pressure behind the sternum, of pains coming on in the night time coupled with feelings of anguish, oppression in the chest. Ructus brings relief. The latter after H_2S, the colicky character of the pains, copious vomiting, sarcinae in the vomitus and stools and the X-ray are the typical guides. A combination of angina pectoris and pylorostenosis is not an uncommon phenomenon.

Intestinal disturbances such as stubborn obstipation and gastric dyspepsia may also provoke troubles in the chest. They come in the form of anginose attacks, oppressive pains behind the sternum, sometimes with, at other times without irradiations just as in true angina pectoris. The differential diagnosis between a reflex or toxic angina pectoris and a true stenocardia can only be decided when we have definite proof that the anginose manifestations absolutely coincided with the gastric disturbances

and that the former disappeared with the cessation of the latter. It is well also to bear in mind that there is a strong connection between true anginose attacks and constipation and the accompanying straining efforts to expel the scybala. In some of these cases a possible weakness in the cardiac vessels, that is a slight inclination to vasoconstriction might attract our attention. I have in mind here male patients who have been treated—properly or insufficiently, it matters not—for syphilis and in whom there may be a suspicion of a syphilitic coronaritis. When the patient tells us that brisk walking causes a slight oppression, and we find a positive *Wassermann* reaction, especially in syphilitic suspects, we may gather valuable information about such anatomical lesions in the vessels of the heart.

The discharge of mucous masses from the canal in *colitis membranacea* is also reputed to bring about attacks of precordial pains of an anginose type through a spastic reflex action on the coronary arteries. I have never had the opportunity of seeing such a case.

The differential diagnosis of true angina pectoris and certain neuralgias craves special attention in this place. I have previously mentioned that angina pectoris very often sets in with vasomotoric manifestations in the left upper extremity. It may be added here that if it appears

on the ulnar side of this extremity it may be accepted as the first signal of an anginose attack. This pain starts from its accustomed irradiation field, ascends to the shoulder and then settles behind the sternum resp. in the cardiac region. In some cases it specializes only in the upper, in others only in the fore-arm. There is a certain resemblance to thoraco-brachial neuralgia. This may become so accentuated that, as it happens in true angina pectoris, paresthesias and hyperesthetic zones continue to prevail, especially in the ulnar region, for a considerable time after the attack. The affected (nearly always the left) arm becomes so sensitive that even the slightest touch evokes intensive pain. In these cases the evidence of typical pressure points, the sensation of weight and stiffness, the sensibility disturbances in the affected part, and the accentuation of pain when moving the upper extremity involved, are the deciding factors.

Of course, when the pain is entirely confined to the chest and when irradiation does not exist, the question will naturally arise: Is it angina pectoris or *intercostal neuralgia?* The evidence of typical pressure points will speak for the latter. Additional proof for the differential diagnosis will be found in the circumstance that intercostal neuralgia does not show that axiomatic dependence on bodily movement which is so conspicuous in most cases of angina pectoris.

But there is another condition which may intervene here as a disturbing element. There are cases of true coronary sclerosis without aneurysm of the aorta or even without a considerable distension of the aorta itself in which anginose pains are felt not only behind but simultaneously also to the right and left of the sternum, generally in the second or third intercostal space; likewise laterosternal pressure points on both sides as well as in the second and third intercostal space on the level of the mamillary line. The resemblance to intercostal neuralgia becomes thereby still more marked.

Although hyperalgesias of the skin over the left breast in a more or less extended circumference are often enough noticeable, it seems to be rather difficult to demonstrate in most cases the other lateral and retrosternal pressure points of intercostal neuralgias. This fact alone, independently of the other findings, the causative agents of the attack and the remaining accompanying symptoms, appears to be the most reliable guide to a correct differential diagnosis.

Sometimes an angina pectoris is erroneously diagnosed when it is in reality a *neuritis* of the *phrenic nerve*. Here, too, the patient complains of sudden severe pains in the chest with heavy oppression. But the differences are considerable. Regardless of the basic disease (pleuritis, pneumonia, pericarditis, subphrenic inflamma-

tion, polyneuritis) of a neuritis of the phrenic nerves and the incumbent rise in the temperature we find here generally very pronounced dyspnea in superficial breathing, while in angina pectoris the patient breathes without hindrance, or, perhaps with reserve, i.e., as little and as lightly as possible in order to forestall pain. Cardiac asthma, however, may coexist, not necessarily, but if it does it may be readily recognized from the sputum and other pulmonary conditions. Moreover, in neuritis of the phrenic nerve the typical pressure points, singultus are in evidence, whilst death-agony is absent, fear of suffocation prevails.

Vagus neurosis of a purely functional nature may also assume the appearance of angina pectoris. Severe oppression in the chest arising from the stomach, most intensive pains with death-agony, a sensation as if the heart came suddenly to a standstill, followed by an abnormally quickened cardiac activity are among the salient features. Negative organic conditions carry no importance, but strongly symptomatic are: high grade dyspnea, volumen pulmonum auctum, a host of vasomotoric manifestations such as chills, a general feeling of cold, pallor of hands and feet—signs, all of which fit in the frame of vasomotoric angina pectoris.

Anginose attacks come to the surface not only in functional disturbances of the *vagus*—

Gowers' so-called vasovasal affections—but also in anatomical lesions of the nerve itself. Physiologic observations show that the vagus nerves carry vasoconstricting fibers to the coronary vessels. It follows that an angina pectoris may be the possible outcome of an irritation of this nerve. *Variot* has observed a similar condition in children suffering from tuberculosis of the bronchial glands assumed to be due to vagus compression.

Fusiform dilatation of the *esophagus* needs to be mentioned here. Mechanically irritating particles of food may cause the rise of gases or the acid contents of the stomach, and the passage of a dilated piece of food may occasion a sudden spasm in the muscles of the esophagus and thus cause very intensive spastic pains behind the sternum with a feeling of a heavy weight on the chest and in the stomach, with anguish and dyspnea. The pain may even sneak along the ribs in the form of an intercostal neuralgia, and also radiate to the shoulders and arms.

The diagnosis should offer no difficulties if we keep our eye fixed on the typical manifestations such as congestion of food, regurgitation, fits of coughing and dysphagia, also a puriform dilatation of the stomach due to a cavity above the latter, stagnation of foodstuffs which may easily be separated by physical and chemical

examination from the stomach contents; and the X-ray. The proof of a bipolar occlusion of the cavity above the stomach can be established when the flow of the water through the stomach tube comes to a sudden stop.

Every form of *esophageal spasm,* no matter of what origin, may resemble angina pectoris. The main differential points are these: the victim of a stenocardial attack complains of a painful feeling in the chest as if he had swallowed too large or too hard a morsel, and of spontaneous or forced belching.

Moreover, the pains in spasms of the esophagus, generally located behind the lower sternum, are frequently coupled with dyspnea, feeling of anguish, palpitation of the heart, and fainting spells—not unknown in angina pectoris either. The resemblance between the two diseases is very strong. But the differentiation ought to be assisted by the thought that the esophageal attack is directly due to the act of deglutition (the intake of food), a causal moment which in the anginose attack is lacking. If in some cases spastic attacks in the esophagus cannot directly be attributed to the swallowing of food, yet the cause will ever lie in the act of deglutition, i.e., the swallowing of saliva—dry gulping. Still another cause may be found in a central or peripheral lesion of the vagus. The stomach pump, the X-ray and eventually esophagoscopy should

remove any remaining doubts. It is noteworthy also that at times solid food will pass, while liquids provoke spasms.

Yet, there are cases in which, quite independently of gulping, a feeling of spastic contraction rises from the region of the cardia to within about the lower third of the sternum. It is an undulating, cramp-like, ascending sensation which after a few moments—less than a minute—recedes, only to repeat again a second or a third time and finish with a belch. No difficulty in deglutition is experienced. In my opinion we are dealing here with a sensibility neurosis in the section of the cardia and the lower third of the esophagus, an *esophagalgia,* a true *cardialgia.* To accept the term "cardialgia" as identical with gastric spasm I consider misleading, *"Gastralgia"* is the proper name that should attach to the latter complaint. It is not unlikely that some forms of cardia—esophagalgia, inclining to enteralgia, is connected with a concurrent motility—neurosis of a lesser degree. I look upon it as the expression of an anatomical lesion of the esophagus, the accompanying manifestation of an esophageal diverticulum, a peptic ulcer of the esophagus. This would, no doubt, render the diagnosis much easier.

In some cases indubitably a true neurosis exists, rarely idiopathic in its nature, but rather a reflex-neurosis emanating from the gastro-

intestinal canal—an upshot of acute indigestion with diarrhea—a sign of habitual excess in smoking, especially of cigarettes; perhaps, a functional vascular pain in the affected region, an intermittent esophageal dyspragia due to nicotinism. When the originating cause is not quite so palpable, an error as to angina pectoris will be obviated when we observe that the patient seeks relief in walking about and shows no signs of oppressing anguish. In cases of doubt the following points will be of assistance: the pains extend from the region of the xiphoid process to about the lower third of the esophagus and not, as in angina pectoris, higher up behind the corpus sterni; the ascending, undulating movement of the cramps, and the fact that the pain is not due to physical exertion, but rather relieved by it. I have come across several cases of cardia-esophagalgia in sclerosis of the thoracic aorta. This might be a sympathetic or causative reflex action of the vagosympathieus. But why not the result of a minor constriction at the portal end of the esophageal artery or a sclerosis of the same? If that is so then these pains which are so similar to anginose attacks and frequently accompanied by difficulty in deglutition would be nothing else than a true *dyspragia intermittens angiosclerotica esophagi* based on anatomical conditions. This contention, however, is open to discussion and further

research. Should it prove correct, another difficulty—but not insuperable—would be added to the differential diagnosis.

For other affections of the esophagus the reader is referred to the chapter on "Pains in the Chest."

The fact that in angina pectoris the irradiation sphere is so prominently localized in the occipital region, or in the teeth or in the left lower maxilla is frequently the cause for an erroneous diagnosis of occipital or trigeminal neuralgia, and that angina pectoris may also give rise to pains in the epigastrium, in the porta hepatis, in the lumbar region, and may even reach out into the testicles, especially the left one, and also into the lower extremities, should not be left unnoticed. I refer the reader to my book on "Abdominal Pain," Rebman Company, New York.

I have repeatedly pointed out that *dypnea* is not to be considered as an important factor so far as angina pectoris is concerned, but that it should rather lead us in the direction of a vagus stenocardia no matter whether the affection of the vagus nerve is of an anatomical or functional nature. The probability is that, when stenocardia and dyspnea appear together, cardiac asthma has associated itself with the anginose attack. It is quite natural that in these cases we witness a mixed, principally an expira-

tory dyspnea, and that the stenocardiac pallor is due to hepatic conditions. But even under this assumption and also in the total absence of cardial asthma and a cardiac pulmonary edema arising therefrom, the diagnosis should offer no difficulty. The periodicity of the manifestations must here be carefully studied in order to understand the connection between the existing asthmatic and stenocardiac conditions, a combination which prevails particularly also in thrombosis or embolism of the coronary arteries.

In every case of angina pectoris a thorough scrutiny of the aortic and cardiac conditions becomes a necessity, especially for the presence of an aneurysm in the aorta. The latter as well as stenocardia equally arise from the same arterial affections, for which reason they are frequently companions. Some authors claim that in this connection stenocardia is due to neuritic affections of the aortic plexus, but, so far as I know, a definite proof for this assertion has not as yet been advanced. I have seen, however, cases of periaortitis and plexus neuritis, i.e., a lesion of sympathicus and vagus fibres arising from a primary sclerosis of the aortic intima. There were present arteriosclerosis of the ascending aorta, recurrent asthmatic attacks, moderate attacks of angina pectoris and persistent sympatheticus paresis of the eyes and of the left side

of the face. The post mortem only can establish an affection of the aorta.

A question. Is there such a thing as *febrile stenocardia* due to disease of the aorta or of the coronary arteries? I once saw a patient who complained about pains stretching diagonally across the breast, especially behind the sternum whether he was in motion or at rest. Subfebrile temperature for three weeks or more. The attending physicians diagnosed influenza. I found a slight aneurysmatic dilatation of the aorta and of the left ventricle and a positive *Wassermann* as the patient admitted that he had had syphilis. My diagnosis was angina pectoris due to syphilitic febrile aortitis. I saw this patient again later on in a very severe anginose state.

When the attacks of true stenocardia come in such rapid succession that there is scarcely an interval between them, we speak of a *status anginosus.* This does not, however, render the diagnosis more difficult, unless there is an abnormal situation in the pain itself. But if this is not present, a confusion between such a state and *epistenocardiac pericarditis* seems to me excluded. True, the patient complains of constant pain in the region of the heart; but this is not as intense as the anginose pain is, and less connected with violent sensations of anguish or not at all. There is rise of temperature, pericardiac friction and signs of fluid pericardiac

exudates. It is worthy of notice that after an epistenocardiac pericarditis the anginose attacks discontinue; also that an ordinary acute pericarditis may produce a modified status anginosus. The slight rise in the temperature should forestall any mistake so far as epistenocardiac myocarditis is concerned in this connection.

It is much easier to err in the direction of *crises gastriques* due to tabes, or other spinal affections or also to diabetes mellitus. Generally speaking, they dwell in the epigastrium or in the entire abdominal cavity, but may localize at times exclusively in the region of the heart. But the presence of vomiting, "dry vomiting," the fact that the pains are not so much localized behind the sternum than rather in the cardiac region, the other typical signs of tabes or other causative diseases, should secure the diagnosis. Difficulties might be encountered when tabes or diabetes mellitus are also associated with insufficiency of the aorta, in which case a diagnosis of angina pectoris might possibly result. But such an error can be easily avoided if due regard is given to the typical symptoms of these gastric crises, proper localization of the pains is made and the periodicity of the attacks is observed.

Pain in the Sacrum

In cases of pain in the sacral region let the patient first of all show the exact spot where the pain is felt. Pain in the back is such a general term among the people at large that no specific meaning attaches to it. With some it means the lumbar, with others the mesial or also the sacral region, for which reason it is quite proper here to give due consideration to all the morbid conditions that may occur in these different sections. For fuller particulars see my book on "Abdominal Pain." Upon closer examination it is generally found that the patient eventually locates the seat of the pain above the ilium below the 12th rib, in other words in the direction of the caudal section, that is, the region of the sacrum.

If the pain is lodged in the lower two-thirds of the lumbar region, it portends undoubtedly trouble in the muscular tissue. These pains in the small of the back are provoked by gymnastic exercises, digging in the garden, or chopping wood or any kind of physical over-exertion. But also in normal constitutions they may be due to want of rest and loss of sleep, and are found in myasthenia, in debility of the musc.

erect. trunci, in *Basedow's* disease and in tetany, likewise in all youthful individuals with a weak muscular system. The derivation of the pain and the tenderness to touch in the affected muscles are sure guides for the diagnosis. The same pains are experienced also after a long surgical major operation, likewise in the spring when heavy clothing is discarded for the lighter apparel of the season. Emaciated people have the same experience owing to an overburdening of the muscles in the loins. And who has not felt that pain after standing on one's feet for a long time or after stooping down repeatedly?

This same pain is the steady companion of certain infectious diseases. It springs from a myalgia obtaining, for instance, in the secondary stages of syphilis before and with the advent of exanthema and other muscular pains in the extremities. In this connection the pains are generally sharper in the night time. The lumbar pains which generally follow in the wake of various infectious diseases rest on the same basis, i.e., musculo-asthenic or else nervous. As a rule they travel in company with general fatigue, muscular aching in the legs and low vitality power; they disappear with progressing convalescence and the gaining of strength.

Lumbago is without doubt the most formidable muscular pain in the lumbar region. It may come on in the form of an acute attack,

or may exist as a chronic myalgia. In the former case it arrives suddenly with a sharp rapid pain; the latter is of a creeping, insinuating character. It would be an error to rest the diagnosis upon the assertion made by the patient that the pain is sharpened by bodily movement or when stooping down and trying to erect the body again. The same manifestation occurs in spinal diseases and in intraabdominal affections, for instance, of the kidneys, the stomach, the intestines, the abdominal aorta, etc. The distinguishing symptom in the diagnosis of lumbago is the exquisite tenderness to touch of the involved muscles, especially of the musculus longissimus dorsi, or sacro-lumbalis and musculus quadratus lumborum. In addition there are: hyperalgesia of the skin to the electric current, scoliosis in the lumbar segment (nearly always on the sound side), the missing transformation of the physiologic lumbar lordosis by erect posture into an arching kyphosis by a thoracic forward movement, the limitation of the lateral movement, the "ludicrous" sort of pain.

A traumatic affection of the lumbar muscular system, the *rupture* of a *muscular fasciculus* or fibre (traumatic lumbago) may easily be mistaken for the common form of lumbago. The differential diagnosis should not offer any difficulties in this connection.

If the pain has come on very suddenly after

or during an overexertion, e.g., lifting a very heavy burden, and if the patient has never been subject to rheumatouratic troubles and there is evidence of painful indurations, cords, callosities in the affected muscles, we must look for a muscular rupture. But do not forget that uratic arthralgia or arthritis, or uratic myalgia or myositis is often associated with a local trauma. Whenever a headache comes on very suddenly our attention should be directed to the possibility of some acute, painful muscular affection, especially in the lumbo-sacral sphere. Tetanus, for instance, sets in with pains in the sacrum.

When these pains in the sacrum are very persistent, especially in persons of unusual girth, our thoughts should be turned to those morbid processes in which an *extravagant lordosis* of the lumbar vertebrae exists with subsequent unusual weariness. This is frequently the case in the later stages of *pregnancy,* and is also observed in invalids who walk about with *large ascites* (due to cirrhosis of the liver) or with a big *tumor in the abdominal cavity* (ovarian cysts). The pains disappear with the removal of the cause.

Lumbar pains in people with a *pendulous abdomen* are often enough associated with other abdominal disorders, such as *enteroptosis.* We find here anomalies of the intraabdominal circulation, a dragging, pulling sensation in the

mesenterium, sometimes only as a manifestation in part of a generalized *habitus asthenicus* in which the patient is apt for muscular reasons —impaired condition of the locomotorium—to complain of lumbar pain. A quick recognition of the stigmata of the so-called constitutional anomalies such as a chicken-breast, or a flat, long thorax, a sharp epigastric angle and a 10th costa fluctuans, should soon clear up the situation.

Any kind of static overstraining of the spinal column due to anomalous conditions, e.g., *flat-foot,* may give rise to pains in the sacral region and in that way resemble lumbago. The same may be said when lameness or the abbreviation of one limb puts a special task upon the other lower extremity; also in lumbar lordosis due to the wearing of high heels on the shoes.

Pleuritis fibrosa after thoracotomy for empyema must be mentioned here, also *lumbo-abdominal neuritis,* especially when associated with lumbar herpes zoster, likewise *diaphragmatic pleuritis.*

Next in order are the diseases of the vertebral column. In acute attacks the first thought will be of *rheumatism in the vertebrae,* often enough the direct predecessor of *articular rheumatism.* It may also happen that an acute attack of gout primarily rests in the lumbo-sacral vertebrae and thus leads to an erroneous diagnosis of lumbago

or acute muscular rheumatism. A thorough test of the purin metabolism is here the most potent factor. In chronic uratic deathesis the presence of pain in the sacrum seems to me an expression of gouty affections in the lumbosacral vertebrae. The trouble disappears under antiuratic treatment.

Similar symptoms are observed in *acute spinal meningitis,* in fact in all infectious diseases that set in with headache—Acute rheumatism is frequently confounded with acute spinal meningitis of tuberculous origin—A proper differentiation—but not in cerebral rheumatism—can only result from a consideration of those accompanying symptoms which are foreign to acute articular rheumatism, but pertain to meningitis. Both have in common local pressure and throbbing sensations, immotility of the spinal column, difficulty in sitting up or turning around, and rise in temperature. But exclusively in meningitis we find: violent initial headaches, hammering sensation in the cranium, turbulent conditions in the sensorium, hyperesthesia of the skin or at the trunks or extremities of the muscles, tâches spinales, *Kernig's* symptom, indrawn abdomen, and stubborn obstipation. If only a few of these meningitic manifestations are present, the diagnosis for acute articular rheumatism falls. That for meningitis will be corroborated by lumbar puncture and the examination of the

fluid obtained, which would also definitely eliminate the question of cerebral rheumatism.

An epidemic cerebrospinal meningitis—not tuberculous—might obscure the diagnosis, but a careful scrutiny of the symptoms enumerated above should easily dispel all doubts. We must not lose sight, however, of a possible early appearance of very acute herpes in the face, or of an initial exanthema, but an examination of the spinal fluid, also of the naso-pharyngeal secretions, should bring the necessary light. I wish to point out also that in epidemic cerebrospinal meningitis articular pains with or without disfigurations in the articular outlines must claim our attention. This is by no means strange, because epidemic meningitis is a bacteriemic disease in its nature. The fact that these articular pains set in only in the maturer and not in the earlier stages of the disease should remove what barriers there may be to an adequate diagnosis.

During the incipient stages of acute poliomyelitis very intense lumbar and sacral pains beset the victim and continue to hold sway during a goodly portion of the course of the disease. Hyperesthesia of the skin, differentiation of the tendon reflexes (patellar reflexes are not present), proclivity to perspire and leucopenia are telling points in the diagnosis.

In all cases in which the patient complains of

sudden attacks of severe headache it is our duty to differentiate between the whole group of infectious diseases that are accompanied by headache throughout the whole course. *Tetanus* belongs here when the port of entry of the infection, i.e., the lesion lies in the sacral zone. *Variola, yellow fever, influenza, grippe, pseudo-influenza, influenza nostras* and *exanthematous fever* are further members of this group. Yellow fever is specially discussed in the chapter on "Muscular Pains." In exanthematous fever the pains in the sacrum are sometimes so aggravating that the patient can find no resting place in the sickbed. We can always get a good portrait of the disease from the contemplation of the following signs: chills, rise of temperature in the continued fever, early acute splenic tumor, the xanthic, dry coating of the fissured tongue, roseola spreading rapidly over the whole body, clouded sensorium, leucocytosis, *Weil-Felix* reaction, progressive changes of the roseola patches into petechiae, the decline of fever between the tenth and fourteenth day of the illness, and the quick convalescence of the patient.

In *gastro-intestinal malignant pustule* epigastric as well as lumbo-sacral pains are prevalent. The remaining symptom complex resembles that of a generalized septic disease with repeated vomiting, diarrhea with traces of blood, meteorism, early involvement of the circulatory appa-

ratus and of the brain, and alarming dyspnea. Bacteriologic examination of the stools, of the blood and of the lumbar puncture fluid and consideration of the occupational condition of the patient should warrant a correct diagnosis.

Variola (*smallpox*) is always ushered in by pains in the sacrum. An early recognition of the other symptoms will promptly establish the diagnosis. I enumerate here the following: initial vomiting, headache, epigastric pains, brisk rise in the temperature, symmetric initial exanthema marked with small hemorrhagic lines, somewhat resembling purpura, generally in the ilio-femoral triangle or in the back, in the armpit or on the palate. All these manifestations are of differential diagnostic import, also in *varioloid,* except that the incipient stages, the whole course of the disease, also the changes in the temperature are of a milder character, unless the typical exanthema has already set in. Severe headaches are of rare occurrence in *varicella* (*chickenpox*) a circumstance which distinguishes it sharply from variola and varioloid.

Severe pains in the sacrum are more frequently found in the *plague* than in *spotted typhus.*

The diagnosis of *influenza* is firmly based on the evidence of articular pains, catarrhal conditions of the mucous membrane of the respiratory tract, gastrointestinal symptoms, tenderness on

pressure in the nasal cavities resp. the processes of both the first trigeminus branches, also of the occipital nerves, the bacteriologic findings of the sputum and nasal secretions.

Of very pronounced intensity are the pains in the sacrum and along the two ischial nerves in *contagious* or *infectious erythema.* The spot in the face where the affection appears feels burningly hot to the touch.

An erroneous diagnosis of influenza may slip in in cases of *trichinosis.* The patient complains of lassitude, gastrointestinal disturbances and poignant pains in the sacrum. Eosinophilia of the blood, the evidence of trichinae in the muscles and in the blood, muscular pains and edema save the diagnosis.

In some infectious diseases pains in the sacrum do not assume such a prominent position. I include *abdominal typhoid* and the illnesses due to inoculation with *anti-typhoid serum* beginning with chills, giddiness, pains between the shoulder blades, behind the ribs, especially on the left side and also articular pains; *paratyphoid, septic* and *acute leucemia, malaria, recurrent fever, Weil's disease, Malta fever and dengue.* In the latter there is slight stiffness in the whole spinal column. This might possibly be due to impaired motility of the vertebrae. In Malta fever we might look for a sympathetic affection of the sacro-iliac vertebrae, and in *Weil's* disease

for muscular affection. The rest of the acute infections enumerated above are mostly based upon toxic infectious hyperemia of the meninges of the spinal cord.

Of different meaning are pains in the sacrum, with preference on the right side (rather in the lumbar region) in *acute cystopyelitis,* especially during pregnancy and the lying-in state, but also at other times in the female sex. An acute feverish illness coupled with sacral pains should always prompt us to look for a renal succussion. Bacteriologic, microscopic, cultural and cytologic examination of the urine, also when necessary a cytoscopic examination, are in demand, as otherwise the existence of an acute pyelitis might be overlooked. Of course, sacral pains may also arise from other affections of the kidneys, such as *renal tuberculosis.* For fuller particulars consult my book on "Abdominal Pain."

Gonorrhea likewise belongs in this category, but only insofar as this disease does establish itself at times in lumbo-sacral vertebrae. In any case of recent gonorrhea the diagnosis should offer no difficulties. But in cases of long standing where the affection of spinal vertebrae has only crept in by a slow process, the diagnosis may meet with obstacles. The fact is that such a gonorrhoic arthritis may within a given time, perhaps, during a period of several years, settle in the zone of the lumbo-sacral vertebrae, espe-

cially when the latter have been exposed to repeated traumatic influences. In the confusion a diagnosis of traumatic neurosis, vertebral tuberculosis or chronic syphilitic meningitis may erroneously result. Ankylosis of the affected vertebrae in youthful individuals should always be taken as a very suspicious symptom. Diffuse rigidity in the upper portion of the spinal column is another interesting signal.

Syphilis may also lead to an analogous vertebral rigidity. But in this case the entire spinal column is involved. The classic therapeutic measures will promptly correct the malady.

Rhizomyelic spondylosis or *rhizomyelia* or chronic *ankylosing spondylitis* and *arthritis deformans* are concatinated with this series of affections; arthritis deformans especially when coxitis coexists. In both diseases the patient complains sometimes that the pains appear after he has been standing upright or stooping down for a while. A change in the weather may bring them about. In others they appear in the morning, decline during the day time and during rest vanish altogether. There are manifestations of neuritis in the roots (irradiating pains, hyperaesthesia, hyperalgesia, muscular irritation and paralytic manifestations). In some cases rigidity in the spinal column, difficulty in walking or when resuming an erect position are the predominent complaints. The clinical picture does

not often aid us in differentiating between the two diseases, especially so in the earlier stages, although arthritis deformans occurs nearly always only in the later years of man's life. The radiologist can usually solve the question.

There is a form of rigidity of the column of myogenous origin in which there is no trace of changes in the vertebrae, but pregnant sensibility to pressure and very marked stiffness and atrophy of the muscular plexus. The two last named signs, however, may also be observed in arthrogenous columnar rigidity. I am inclined to believe that there is another form of rigidity besides those mentioned here, viz., the arthrogenous form in which no evidence of articular changes appears on the X-ray plate.

Pain in the sacrum is a guest also in diseased inflammatory conditions of the *sacroiliac articulation* (*synchondrosis*). The distinguishing marks are: the patient seeks relief by inclining the body to one side, there is pressure pain along the line of communication between the sacrum and ilium, the painfulness is sharpened under bilateral compression of the pelvis, or an edema in the sacro-iliac zone.

The pains arise from various causes. In pregnancy, in parturition they are due to serious infiltration of the joint ligaments or to merely mechanical influences. Even a quite normal menstruation, especially in mothers, may bring

about sacral pains during the period through an unusual relaxation of the sacro-iliac joints. Furthermore, there is a possibility of painful spells in this same joint in acute and chronic articular rheumatism, more rarely in gonococcal rheumatoids, but also in infectious septic rheumatoids. But all this happens chiefly when other joints have been previously or are simultaneously affected. Then and there it is the primary or else the solitary seat of the articular affection which may, however, also originate from osteomyelitis—not necessarily tuberculous. I firmly believe that gout itself may settle in the sacroiliac joints. In Malta fever the pain-stricken sacrum is the result of an accompanying affection of the sacroiliac joints. In various static conditions (flatfoot, pendulous abdomen) the sacroiliac joints are the mischievous element insofar as sacral pains are concerned.

Diseases of the *bony substance of the vertebrae* and of the *adjoining ribs* will provoke sacral pain. Besides fractures of the lumbosacral vertebrae (breaking of the 5th lumbar vertebra in lifting too heavy a burden, often the cause of pain for a period of years) proved by the X-ray, caries, syphilis, actinomycosis, infectious (paratyphoid) spondilitis and neoplasms belong in this place. Their diagnosis will receive proper attention in the chapter on "Backache."

One item of interest, however, I must mention

here, viz., that pressure and pulsation sensitiveness in the spinous processes of the sacral, or else of the lowest lumbar vertebrae with pains in the sacrum, also without any kind of disease in the vertebrae themselves, are warning signals of gastro-intestinal trouble (rectum, bladder, internal portion of the genital organs). And still another point may be added: pain in the sacrum is often the register of a carcinoma metastasis in the lumbo-sacral vertebrae. The primary neoplasm has not been manifest until the sacral pain gives the signal for a hunt of its favorite habitat either in the prostate, the testicles, the ovaries, the mamma, the thyroid, the adrenals, or in the penis or the gastro-intestinal canal.

Diffuse diseases of the skeletal frame frequently start with pains in the sacrum, especially *osteomalacia.* The fact that these pains arise in the early stage of the puerperium, if they have not already done so during the latter period of pregnancy and that they are aggravated by walking, points to the diagnosis of osteomalacia, especially when we find the additional symptoms of pressure sensitiveness in the sacrum or the os pubis or the ilium—the latter is easily evinced by a quick lateral compression—the rostrate extension of the symphysis, the cordiform pelvis, waddling gait, spastic adduction, osteoscopic pains (ribs, sternum), reduced stature.

The late war has given us copious proof of the existence of *starvation malacia of the spinal column.* There is weakness in the legs, tremor, incapacity for work coupled with pains, local painfulness, axillary compression, impaired motility and a characteristic X-ray picture.

In *congenital malformations* of the *pelvic bones* pains in the sacrum are of frequent occurrence. In such cases radiology is the imperative adjuvant of the diagnosis. It will definitely reveal any abnormal conditions that may exist in the bony lumbo-sacral column such as spina bifida occulta, hyperplastic changes in the transverse processes of the fifth lumbar vertebra with secondary bursitis and contraction of the last lumbar vertebra with the sacrum.

It goes without saying that *diseases* of the *sacrum* itself such as caries or osteomyelitis carry local pains with them, especially in the softer teguments, e.g., *bedsores* over the os sacrum.

The same is the case in affections of the *spinal cord* and its *membranes.* In *acute tuberculous* or *epidemic meningitis,* as has already been pointed out, pain in the sacrum is the foremost symptom.

In *acute poliomyelitis* there are not only pains in the sacrum but also along the whole of the spinal column. The differential diagnosis needs to fall back in such cases upon the other symptomatic manifestations of meningitis. Chronic

spinal lumbar meningitis must not be forgotten here. It is not unlikely that the lancinating pains in the sacrum which so frequently appear in tabes are really due to such a chronic meningeal affection. Internal spinal pachymeningitis whether it is hemorrhagic in its nature or not, and also the syphilitic form belong to this order. The exceptionally intensive and often long continued pains in the sacrum so characteristic of *membranous tumors* of the *spinal cord* should always remind us of a possible morbid growth or some other painful affection of the cauda equina specially marked by unilateral pains, paresthesias, pain in the region of the ischiadic nerve and absence of achilles tendon reflexes, while in tumors of the conus terminalis disturbances of sensibility in the ano-vesical and sexual zones are more prominent.

In tumors of the cord itself pains in general, particularly such in the sacrum are rare. They may be distinguished from extramedullary pains by the observation that in extramedullary tumors pain is the primary symptom and continues to hold a predominating position for a long time. The sacral pains in intramedullary tumors are due to secondary compression of the neighboring nerve roots. In true *myelitis* they are of a less vicious character, but if they increase in acuteness we are warned of a parallel meningeal affection, i.e., *meningomyelitis.*

Syringomyelia as well as multiple sclerosis, tabes and paralysis, the paraplegic (pseudo-myelitic) form of *hydrophobia* carry sacral pains as companions with them, although they do not appear so vividly in the clinical picture.

The sudden onset of sacro-lumbar pains after a trauma arouses at once the suspicion of an *injury* to the *spinal column,* not to speak of a muscular breach. *Meningeal apoplexy,* even without a preceding trauma, is not excluded. The patient usually cries out aloud with pain and collapses as both the lower extremities are paralyzed. Hemorrhages in the substance of the spinal cord, acute *hematomyelia* may likewise be the activating cause of local lumbar sacral pains, plainly due to compression of the adjoining nerve roots.

Functional nervous diseases are fertile ground for complaints of pains in the sacrum. *Spinal irritation, neurasthenic muscular pains in the back* arouse the fear of some kidney trouble or affection of the spinal cord and promptly lead the way to the consulting room. The patient will tell you that he feels as if the small of the back were broken in two, of physical and mental fatigue, any kind of excitement provokes or aggravates the sacral pains, while on the contrary—and this is an important symptom—moderate exercise alleviates or banishes the pain altogether. Add to this the proof that the pain

is located in the lower lumbar zone or to one side of it, that it radiates towards or along the vertebral column, the constant change in its intensity, the total absence of other usual objective symptoms—excepting the neurasthenic signs —and the diagnosis should easily crystallize.

The proper understanding of such a condition should largely influence our judgment that we are dealing not with an organic, but with a functional disorder, e.g., of the stomach or of the heart. Nevertheless caution is necessary. In sexual neurasthenia, for instance, the chief complaint is that of pains in the sacrum, especially after sexual exercise, or after masturbation or pollution. Other complaints are strangury, mostly in the day time, less so at night (in some cases, especially of long standing, there is also nocturnal pollakisuria), subjective hyperesthesia, paresthesia of the external genitals, genital hyperhydrosis, permanent feeling of cold feet due to spermatorrhea and nervous impotence (in persons of advanced age suspicion of incipient malignant neoplasms!), urethral or prostatic neuralgia, orchiodynia and similar manifestations. Inadequate sexual satisfaction, excess in venere, long continued coitus interruptus are also frequently the parents of pain in the sacrum.

In *hysteria* these pains are not so common. The diagnosis here must be based upon the usual pertinent stigmata, the presence of local

hyperesthesia of the skin, the fact that a deep pressure often causes less pain, than a soft, superficial touch, and the finding of a similar painful zone at a higher section of the vertebral column, commonly situated between the shoulder blades.

Some psychiatrists have reported cases—I have no experience in this matter—in which backaches and pains in the sacrum have been of so violent a character, that the afflicted persons were unable to perform their vocational duties, sometimes for a year or more during the period of adolescence. This affliction has been yclept "dementia precox."

Another source of hieralgia is every form of *retroperitoneal disease.* The various *renal* and *adrenal affections* including *bacteriuria* and *alcaptonuria* and all pancreatic diseases are comprised in this classification because all of them father pains in the sacrum and in the lumbar region. *Splenic tumors* likewise belong here. Not only the primary but also the secondary pains must be taken into consideration, for the latter, radiating even into the shoulders, are frequently symptoms of an ulcer in the posterior wall of the stomach or in the duodenum with proliferations into the pancreas, or of an ulcer in the pancreas associated with a gastric carcinoma, or perigastritis which may lead to an arrosion of the pancreas.

So far as kidney affections are concerned it may be said that pains in the sacrum appearing on one, chiefly the right side only, are often the sole complaint in acute or subacute *strangury* in pregnant or parturient women. Fever and renal succussion are not present because there is no infection of the urinary apparatus. Nevertheless it is a hint that these pains, although primarily due to the gravid state, may also arise from a concomitant hydroureter. The fact that in these cases the ureter is sensitive on pressure in the iliac region, should easily prevent the error of a mistaken appendicitis. This is also true in some cases of chronic strangury in the male, for instance in hypertrophy of the prostate.

In affections of the kidneys (also of the pancreas) the site of the pain is not always in the upper lumbar region—where it anatomically belongs—but apparently it is in the lower lumbosacral zone. In benign as well as in malignant renal tumors this happens frequently.

Backaches in diseases of the *gallbladder,* in *cholelithiasis, cholecystitis, pylorostenosis* and *duodenal ulcer* deserve mention here. When sacrolumbar pains are present in stenosis of the pylorus they are due to a distension of the stomach, unless they are caused by a pyloro— or gastrospasm. In this connection we shall always find a high grade tension or flatulent sen-

sation in the epigastrium which disappears with spontaneous or voluntary ructus or vomiting.

Duodenal ulcer, both benign and malignant, is not always of necessity associated with sacrolumbar pain. It passes away often enough without them with the manifestations of a chronic obstructive icterus insofar as a carcinoma of the diverticulum Vateri is concerned. But if in the course of the disease intensive pains in the sacrolumbar region suddenly set in the diagnosis of accessory retroperitoneal complications may not be amiss.

This thought is quite opportune when such pains turn up in infectious diseases of the small pelvis which do not by nature carry sacrolumbar pains with them, such as ascending phlegmons of the *retroperitoneal cellular tissues,* lymphangitis, retroperitoneal lymphangitis, arising from primary lesions of the internal female genitalia, or of the rectum (fissure), the internal male generative organs, the external genitals, the lower extremities, and also appendicitis, etc. When tenderness to deep upward pressure along the spinal column or arcual kyphosis of the lumbar vertebrae are also in evidence, the diagnosis is made easy.

Neoplasmatic affections arising from a new growth in the small pelvis, e.g., a uterine carcinoma may be the causating element of these pains. In this case, of course, not only the

lymphatics but also the lymph glands themselves are involved.

Every disease of the retroperitoneal lymph glands may provoke pains in the sacral and median region (see my book on "Abdominal Pain") for the obvious reason that an enlarged gland naturally crowds the nerve roots of the hypogastric and lumbar plexus. The glandular swellings may be the outcome of tubercular or lympho-granulomatous, of leucemic or aleucemic or in rare instances of syphilitic origin. Their presence is betrayed by these very pains in the sacrum as well as by the entire clinical aspect of the disease. Palpation is only possible when a number of the affected glands are bunched together into a lumpy mass (tuberculosis, leucemia). We may be also dealing with a metastatic neoplasm (sarcoma, carcinoma or a lymphosarcoma). In the latter case the diagnosis will be surrounded by difficulties unless palpation is rendered possible for the reason aforesaid, or a splenic tumor or a lymphosarcomatous disease of some other internal organ is a concomitant issue (tonsils, stomach, gastrointestinal tract). The initial sacral pains are here a leading symptom, the same as in lymphogranuloma of the retroperitoneal glands. In aleucemia and in leucemia these pains rarely manifest themselves in the beginning of the disease, which is also the case in chronic lymphatic leucemia.

The observation of sacro- or median-lumbar pains will prove a powerful adjuvant in the diagnosis of metastatic growths in the retroperitoneal (paravertebral) cellular tissue following a primary carcinoma of the stomach, the intestine, the uterus, the esophagus or the mamma. If these pains arrive now in successive attacks and then again are of a protracted character (not at all uncommon in *carcinoma of the stomach*) there can be no doubt about the diagnosis. It is further confirmed by a strong pulsation—both visible and palpable of the abdominal aorta, and no less by a loud stenotic bruit over it, even noticeable to the touch.

The situation is different when the gastric carcinoma has a rearward tendency which by its very nature is productive of sacral pains, or if a companion sickness (metastasis) of the spinal column or an extension of the carcinoma itself into the pancreas put in an appearance. In such a combination, especially in cases of direct *arrosion of the pancreas* through a gastric carcinoma —likewise through an ulcus ventriculi on the posterior wall of the stomach—the most intense continuous pains are unleashed in the sacrolumbar circumference. (See "Abdominal Pain.")

As soon as the causative gastric disease is recognized we shall promptly find the way to the companion affection. But whether the latter is centered in the lymph glands or in the

pancreas itself can only be established by the finding of the other typical signs of the pancreatic function, eventually by the aid of the X-ray.

Sacral pains in gastric carcinoma may also be occasioned by overloading of the stomach, thus causing hypertension. They may also be a diagnostic roadsign when we waver between carcinoma of the pancreas and that of the stomach, pointing, perhaps, with preference to the retroperitoneal space, i.e., the pancreas.

In *enlargements* of the *retroperitoneal glands,* also in *inflammations* thereof, the pains have the same localization, but are of lesser intensity, especially when retroperitoneal phlegmons are a possible additional factor, likewise morbid conditions in the small pelvis or infections of the lower extremities.

The same kind of pains are encountered when there is an unusual strain on the mesenterium of the peritoneum at the point of union with the lumbar vertebræ, e.g., in mesenteric tumors. Likewise, when a tumor embedded in the mesenterium (metastatic glandular tumor) causes hypertension, or when a chronic, inflammatory (tuberculous) disease contracts the mesenterium. Such conditions cannot easily escape a watchful eye.

All morbid processes which effect an acute stretching of the mesenterium are associated with

median-lumbar pains extending sometimes even upwards into the dorsal vertebræ. In cases where we find such sacro-lumbo-dorsal pains associated with obstructive strangulation our first thought must be directed to the presence of an *intestinal axial torsion,* especially of the small intestine or the colon. Aneurysm of the *abdominal aorta* claims attention here, as well as of the aorta proper, and the entire venous system. *Phlebitis,* or *thrombophlebitis* of the *iliac vein* due to inflammation, suppuration or new growths in the small pelvis or in the lower extremities, may be the originators of sacral pains in one or the other or in both sides. Considering this possibility and finding signs of obstruction in the veins and their radicular sphere, a detailed diagnosis should not be difficult. It is different, however, in cases of cellulitis or retroperitoneal lymphadenitis in which the symptoms of congestion do not prevail.

Chronic *ectasy* of the *pelvic veins* in women is another source of these pains, especially when they come to the surface with the patient lying down. The same relates to *thrombosis,* or *thrombophlebitis* of the pelvic vessels, which are frequently the result of inflammatory or neoplastic affections of the pelvic organs, mainly of the male or female inner genitalia. Here hieralgia is often connected with local pains in the rectum, in the perineum and in the buttocks,

also in the groins. They are aggravated by coughing or sneezing, in fact by every intra-abdominal pressure.

In *neuralgia* of the *celiac* plexus they are a partial factor only.

We may add *chronic lead poisoning,* in which pains in the circumference of the navel radiating into the sacrum and into the thighs are notorious.

Affection of *distant organs* may likewise be provocative of pains in the sacrum. In women they suggest morbid changes in the *internal sexual organism.*

During the *menstrual period* complains of sacral pain are ripe, especially in pregnant women, primarily due to the menstrual molimena. In joint hysteria the pains are often of a most distressing, excruciating character, especially when there is a slight uterine retroflexion or a congenital narrowing of the os or cervix uteri. If the retroflexion is very pronounced the pains are continuous.

Sacralgia often plays a part in the climacteric process, and is caused by the contraction of the pelvic connective tissue. We meet here vasomotoric disturbances, heat flushes, congestion in the head, spontaneous perspiration, paresthesia in the arms and legs, bluish tint and coldness of the fingers, palpitation, cardiac pains, adiposity, meteorism, *Heberden's* nodules, and pains in the nerves and bones.

There is a goodly number of diseases of the female genital organs which travel together with sacral pains. If of a very distressing character they speak for some endometric affection, specifically uterine colics. Labor pains affect the sacrum in like manner, and so does the accumulation of blood-clots in uterine hemorrhages. We find these pains also in affections of the walls of the uterus and of the adnexa, in parametritis and perimetritis of acute or chronic duration, in descent or prolapse of the womb, in new growths of the uterus and of the ovaries and in retro-uterine hematocele.

It is evident that the presence of sacral pains in one and the same gynecological disease may be due to quite a number of different causes, for instance in carcinoma of the uterus. There might be a carcinomatous infiltration of the pelvic connective tissue reaching up even into the retroperitoneal cellular tissue, or there might be a metastasis of the retroperitoneal glands or in the field of the sacral vertebræ, or else hemorrhages in the uterine cavity, all quite independent of the primary uterine ailment.

I should like to give mention in this place to a particular kind of sacralgia which occurs in women who wear lingerie which is open in the groins, and in consequence take cold locally in semblance of a rheumatic affection. The symptoms are easily recognized, viz., the pains

are strongly influenced by chilly, damp weather, brisk physical exercise, by running, jumping or stooping, and there is delicacy to touch and pressure in the dorsal muscles and the spinal joints.

Coitus interruptus is another cause of sacralgia in the female. We hear complaints of a dragging downward sensation, strangury and general nervousness, no doubt due to a deficient detumescence of a congested uterus during the sexual act.

In *prostatitis, carcinoma* of the *prostate* and *tuberculosis* and other affections of the male genital organs, sacralgia is of rare occurrence. I refer to this on purpose in order to forestall a mistaken diagnosis of kidney trouble.

The *rectum* is very extensively responsible for sacral pains. *Hemorrhoids* are the principal offenders. Copious bleeding from piles often removes the pains from the sacrum, at any rate for a time. Nevertheless, a thorough examination of the rectum for a possible fissure or carcinoma should be made in every case.

Polypus may also give rise to rectal hemorrhages and pains in the sacrum. Rectoscopy is here in order.

Affections of the *colon,* especially a deep-seated carcinoma, have similar effects on the sacral zone. Complaints about aches in this region often reach us when there is difficulty in

getting rid of intestinal gases, in cases of chronic constipation, or obstruction in the rectal passage, also disturbances in the intestinal circulation or chronic intestinal catarrh.

When a carcinoma of the rectum has been positively diagnosed, our next thought must be directed to every possible form of deep-seated intestinal stenosis. Distension and wind in that part of the colon which lies beyond the stenosis generate pains in the sacrum, which eventually are modified or cleared away, for a period at any rate, by a copious alvine evacuation, or even a satisfactory flatus.

Pellagra is another fertile cause of sacral pains and backache. Its diagnosis is easy, for the following reasons: its prevalence is limited apparently to those countries in which maize is a staple article of food. It occurs in the spring or the autumn, it shows atypical erythema, there are characteristic manifestations in the oral and genital mucous membrane, there are digestive as well as psychic disturbances, and pronounced emaciation.

Pain in the Buttocks

If a patient complains of pains in the coccygeal region, we generally have before us a traumatic surgical disease (a birth or a fall). It may be a periostitis, a subluxation, or a luxation of the coccyx against the sacrum, or a fracture, or tuberculous caries or osteomalacia. If examination per rectum and the X-rays prove the absence of all these affections, and if we are satisfied that no rectal disorders (fissure, fistula, neoplasm) or genital or pelvic defects that might be mistaken for coccygodynia, are present, we are still confronted by disturbances which may be causative factors of pains in the nates.

There is the possibility of neuralgia of the coccygeal plexus, frequently a sequel of masturbation, sometimes the expression of a sexual neurasthenia, or in rare cases the manifestation of gouty diathesis. I have seen two cases of coccygodynia in men—it occurs in women with greater frequency—which were indubitably accompanied by chronic gouty diathesis. Both diseases yielded promptly to therapeutic measures. Coccygodynia in the climacteric period may, perhaps, be explained in this manner. On the other hand, we come across cases in

women in which this disease of the coccyx is only a partial manifestation of a generalized hysteria.

Besides tenderness to pressure of an otherwise quite normal coccyx and localized cutaneous hyperesthesia, we are bound to discover other signs of hysteria, and as the causative element an acute psychic trauma with the elimination of which both coccygodynia and hysteria vanish. Of course, we find in non-complicated hysteria also pain and delicacy to touch, also cutaneous hyperalgesia in the coccygeal region as well as in the sacrum, with deep pains in the pelvis radiating into the corresponding vertebral surroundings.

In females, coccygodynia is frequently more a part of neurasthenia, or a spinal irritation. Here, too, we find the paradoxical condition that the soft touch awakens the pain, whilst a slowly ascending, long-continued pressure gives relief. Hot sponging or a weak electric current may prove very painful. Local and generalized vasomotoric manifestations are not uncommon companions of pains in the breech. High feeding and psychic influences are apt to remove the complaint for good.

Finally, coccygodynia may be a partial symptom of tabes dorsalis or the direct result of flat foot.

Pain in the Shoulder

The term employed here bears a double meaning, viz., pain in the shoulder proper and pain between the shoulder blades, i.e., between the scapula and the spinal column or in the spinal column itself or in the acromion. Pains in the supraclavicular and supraspinous fossa naturally are included.

Pains in the scapular region conjure up thoughts of all kinds of affections of the shoulder joint. (Many particulars regarding this matter will be found in the chapter, "Pains in the Joints.") In many morbid articular conditions the shoulder joint is, as a rule, the only member to be considered, if not throughout, at any rate for a notable period of the malady's course. Traumatic and neurotic affections, pyemic metastasis localized exclusively in the shoulder joint, gonorrhea and syphilis, all are possibilities. But in the front rank I mention the various forms of arthritis, from *acute* to *chronic deforming omarthritis,* and *gout* in the sense of *omagra.*

The diagnosis is built up from the following symptoms: seat of the pain, restricted motility of the joint, especially in an upward and back-

ward direction, the contrast between the relatively free active motility and the pain caused by a jerky movement of the joint by the physician, sensitiveness on pressure or percussion, especially at the articular extremity; changes in the articular outlines, the assistance of the Roentgen ray. For a more detailed diagnosis, I refer the reader again to the chapter on "Articular Pains."

The shoulder joint may also be the seat of arthropathy, especially in *syringomyelia.* Very severe pain may be a reflex of a spinal affection, e.g., *multiple sclerosis.*

When a patient speaks of pains in the shoulder or in the region of the shoulder joint, it is not unwise to think of a different derivation. In acute febrile conditions the nearest point must needs be an *acute osteomyelitis* in the upper arm.

A *chronic inflammation* (*tuberculosis*) or a *neoplasm* in the *osseous substance,* the *humerus,* will always prove to be painful conditions, and really belong in the operating room. The internist is more interested in secondary metastatic new growths of the scapular bones. Many's the time that a patient complains about "rheumatic" pains in the shoulder, bothersome today, missing tomorrow, coming on again the next day, and is treated by his doctor for rheumatism, when a closer examination reveals a primary

malignant neoplasm (prostate, ovary, testicle, mamma, thyreoid, hypernephroma), and the local conditions (pressure and throbbing pains, X-rays), also blood test, leave no doubt of the existence of a metastasis of the bones. (See chapter on "Pains in the Bones.")

An acute painful affection of the muscular plexus of the shoulder joint, of the deltoid, or of the scapular, cucullar, pectoral muscles, an *acute scapular myalgia*, or *omalgia* may very well give ground to complaints of shoulder pains, in which condition the sick seek relief by keeping this articulation as quiet as possible. These pains are in reality caused, as a rule, by overburdening or overtaxing the muscles, e.g., in amateur sportsmen, carrying a heavy valise or wearing a heavy overcoat, etc. The fact that in these cases—not to speak of a Roentgen photograph of the affected joint—the muscles are very tender on pressure, should make the diagnosis easy. When the pains, however, radiate into the forearm, waist or even the fingers, a neuritic condition might be a good guess. But the lack of the other classic neuritic manifestations (sensibility disturbances, tenderness on pressure in the nerves, nodules, vasomotoric and trophic derangements) should be a decisive factor.

In a similar fashion, *brachial neuritis* is in error often taken for omarthritis. This is quite

excusable because a primary, inflammatory articular affection is frequently accompanied by secondary neuritic conditions of the nerves which encompass or pass over the articulation. In this case the pains radiate along the corresponding nerve trunks and give rise to paresthesias in the affected zone. On the other hand, there may be an atrophy of the appertaining muscles, but without sensibility disturbance and without denegeration of the electric reaction, the latter being only minimized. Some authors attribute this muscular atrophy to a functional lesion of the ganglion of the anterior columna.

The presence of actual neuritic changes may seriously handicap a concrete diagnosis, because the patient experiences, when moving the upper arm, identically the same intense pains as if the joint itself were affected. The differential diagnosis between neuritis on the one hand and omarthritis on the other may in consequence be gravely influenced. In favor of neuritis speak the following facts: the joint itself is not the most sensitive part, pressure upon it from any direction does not materially exacerbate the pain, which is rather localized in the nerve trunks and in the muscles of the upper arm, the presence of paresthesias, sensibility disturbances and muscular, truly neuritic, atrophies. But even this consideration will leave us in perplexity if both anarthritis and neuritis run

a joined course. Yet if the anamnesis establishes the fact that the articular affection came first and the neuritic conditions only as secondary manifestations, then Roentgen photography will promptly make the differential diagnosis positive.

On the other hand, there are patients who locate the pain not only in the shoulder, but also in the nape of the neck, or in the upper arm alone, or again in the whole arm from the apex of the shoulder down to the very fingertips, and these pains are aggravated with every movement. We find the nerve trunks hypersensitive on pressure, also exaggerated tenderness of the skin, muscles and bones, but no disturbances of sensibility or noteworthy muscular atrophy. A great temptation, indeed, to attribute the cause of the pains to neuritis. And yet there is anarthritis with secondary brachialgia. The diagnosis is based here on the X-ray and another point of great moment, viz., if we find that pressure upon the shoulder joint, especially forward in the intertubercular sulcus, is particularly painful, we have definite proof that the motility of the shoulder joint is essentially impaired. The head of the humerus is firmly set in the joint by muscular contraction so that the joint can no longer be moved by fixation of the scapula. Otherwise the scapula will involuntarily follow every passive movement of the arm,

giving the patient intense pains as far down as the fingers. In this fixation of the upper arm we have a definite proof of an existing primary arthritis.

And yet there are patients who, contrariwise, locate the pain in the shoulder blade when it really is in the cervicobrachial plexus. It will occur in restricted morbid conditions of the supraclavicular fossa, e.g., of the superior pulmonary lobe (tuberculosis, tumors, glandular swellings), also of the mediastinum, with direct or irradiating stimulation of the appropriate nerve plexus. The diagnosis is simple enough, for the reason that careful probing reveals a strong sensitiveness to pressure in the plexus above the clavicle, whilst there is no such reaction in the joint itself.

Sometimes an articular affection seems to exist, when in reality the pains are due to an inflammatory process in a *synovial sac,* chiefly behind the deltoid, especially in resorption of tuberculous pleural exudates. The diagnosis in this case is beset with great difficulties when coarser configuration anomalies are lacking. If there is no pressure pain anteriorly between the acromion and the coracoid process, and also in the direction of the axilla, if the shoulder joint moves freely, if there is only localized tenderness in the upper arm about three inches below the acromium, a visible swelling in the bicepital

sulcus, pronounced reactionary inhibition, and distressing painfulness when the patient is abed or resting, the chances are that we are dealing with a case of bursitis. A test for narcosis is indicated when the morbid condition is of long standing. Free articular motility should here also point to bursitis. The X-ray picture will be of service.

A *subpectoral phlegmon* will claim our attention when we find upon closer scrutiny a digital lesion, high fever, malaise, septic conditions, a feeling of tension when the arm is moved, or else lymphangitis and lymphadenitis.

Pains in the shoulder radiating into the upper extremity are in all probability of arterial origin; a surmise of *intermittent dyspragia* arising from arteriosclerosis or arteritis is here a safe conductor. The deciding factor is that the pain sets in with the movement of the arm and ceases when the member is at rest. But it may also come on on a march or during a brisk walk, only to disappear again when a halt is made. Evanescence of the arterial pulse and the Roentgenogram are typical proofs of this painful affection, which is, however, sometimes also recognized in the lower extremities.

Retrosternal pains in the chest that radiate into the apex of the shoulder and thence into the arm, nape of the neck, and with preference into the left lower jaw, intimate an existing

angina pectoris or *aortalgia,* even though this irradiation does not in all cases extend beyond the shoulder. It may likewise take the direction of the right instead of the left shoulder.

Pains in the shoulder associated with aches in the back and sacrum are often of "psychogenous" origin in such diseases as *hysteria, neurasthenia,* and *psychopathies.* The diagnosis in these cases may be at times somewhat puzzling, particularly when the patient attributes the pain to a cold, or an overexertion or an unexpected drenching. Negative clinical findings, proper consideration of the general physical conditions and radiology should grant a satisfactory solution.

Rheumatic pains in the shoulder may be irradiations of nervous origin, f.i., of the phrenic nerve, and extend from the abdominal region as far as the cervical nerves. If the pains are in the right shoulder (or both) we should be reminded of rupture of the liver, cholelithiasis, abscess, gumma, tuberculosis of the liver; if in the left shoulder (or both), rupture of the stomach, ulcus ventriculi with or without perigastritis, a ventricular carcinoma in the pyloric region, or rupture of the duodenum as a sequel to duodenal ulcer, even of the stomach due to a parapyloric ulcer, or of the jejunum after a peptic ulcer thereof. Furthermore, we may expect affections of the left hepatic lobe (gumma,

carcinoma), with perigastritis, or splenic diseases, e.g., perisplenitis, splenic infarcts or abscesses, or rupture or echinococcus of the spleen, or myelogenous leucemia, paranephritic abscess or an acute pancreatic affection (acute pancreatitis, necrosis of the pancreas) (left shoulder). In retroperitoneal growths of the retroperitoneal cellular tissue (glandular metastases, acute inflammation, calosities, metastatic neoplastic infiltrations) they appear mostly on the left side, likewise in all possible neoplasms within the retromediastinal space, not to forget hemorrhages flooding the peritoneal cavity below the diaphragm (tubal pregnancy).

Naturally the patient complains of pain in the affected part ranging as far as the shoulder. But he exhibits also other local signs, e.g., localized tension of the abdominal muscles (rupture of the stomach, of the duodenum, etc.), absence of abdominal breathing or increased pain during the act. Coughing or walking aggravate these pains, which also possess the peculiarity that local pressure over the seat of the tumor influences more the pain in the shoulder than that in the abdomen. But I make the point that a ventricular ulcer or carcinoma will throw the pains to the right shoulder, when it and the liver have grown together; likewise, that shoulder pains in appendicitis indicate a high location of the vermiform appendix or an upward ex-

tension of the morbid condition, i.e., towards the liver or diaphragm. Sometimes this shoulder pain is still further marked by the circumstance that it is very much aggravated when the right leg is somewhat overstretched in the hip joint.

In affections of the pleura (pleuritis) or contractions of the phrenico-costal sinus, in *empyema, pneumothorax,* in severances of *pleural adhesions,* affections of the *lungs* or *pericardium,* these self-same irradiating shoulder pains are very annoying, just as they are in an artificial pneumothorax or a sudden hemorrhage in the pleural cavity from, for instance, a ruptured aortic aneurysm.

I wish to lay emphasis on the fact that when the patient complains exclusively of shoulder pain, the seat of the trouble is for a certainty to be found in the liver. There are two possibilities: *liver abscess* following amebic dysentery, but after a long interval, *echinococcus* of the *liver.* The enlargement of the liver—proved by pain upon lateral pressure against the hypochondria—the presence of a cystic bulging of the liver towards the lung, and other individual symptoms (color of the face, attitude of the patient, leucocytosis, rise in temperature) and the anamnesis should definitely settle the diagnosis of hepatic abscess.

We must always think of that when the sick man who has had dysentery or any other in-

flammatory or suppurative process in the radicular area of the portal vein, begins to worry about pains in the right shoulder. In echinococcus there is pain in the right shoulder only when the parasite is embedded immediately under the diaphragm. In this case there is also a domelike axillary bulging upwards of the diaphragm. With the aid of the other characteristic symptoms of this cystic parasite and of subdiaphragmatic liver abscess, especially the X-ray, the proper diagnosis cannot be missed.

Sarcoma of the liver is likewise associated with pains in the right shoulder. This affection forms one or more nodes on the surface of the liver which arch the diaphragm upwards. So long as there is no dullness of sound over this arch, a diagnosis of hepatic carcinoma can only be made when we find also in other parts of the liver carcinomatous eminences which change the shape of this organ so far as it is accessible to the touch. Our suspicion should be aroused when an elderly cachectic patient complains of pains in the right shoulder, although we can find no pathological changes either here or in the vicinity (neck, lung, mediastinum).

Febrile gummata of the liver, associated with cholelithiasiform, intensive, colicky attacks, generally start with severe pains in the right shoulder several days in advance of the colicky spells, run a parallel course, and disappear with them.

Cholelithiasis itself does not, as a rule, bring these pains in the right shoulder with it; but I once saw a case in which this did happen, with the result of an erroneous diagnosis of neuritis.

In *perihepatitis* in those parts which incline toward the diaphragm the same pains may be expected, and we must keep a sharp lookout for other morbid processes in the diaphragm from which pains might travel via the phrenic and by transmission to the fourth cervical nerve, and thence to the cutaneous nerves of the shoulder.

In *subphrenic abscesses* this happens principally when the subphrenic suppuration proceeds from the liver or the spleen.

More often is it advisable to think of *diaphragmatic pleuritis* when these shoulder pains have come suddenly to the surface and have continued for days as the most prominent morbid symptom. The diagnosis may safely lean here upon the other well-known signs of pleuritis, which, by the way, may also harbor a *primary subpleural* tubercular condition of the lung.

Diaphragmatic hernias and *eventration* are the homes of pain that radiates from the chest to the shoulder, a sign of diagnostic value especially when also the stomach has passed through the hernial breach in the pectoral space. With

the intake of food the pains are naturally very much exacerbated.

But shoulder pain may be the only and exclusive complaint in any form of *pleuritis* which leads to an irritation of the phrenic nerve. It has even the peculiar habit of becoming more severe with any movement of the right shoulder joint, thus leading to a false diagnosis of omarthritis. The presence of phrenic pressure points (those in the neck, sternum and spinal column may miss and only the abdominal points may react!), the non-participation of the affected thoracic portion in the act of breathing and the accelerated respiration will no doubt facilitate the diagnosis even when no pleural friction is perceptible.

The same applies to *pericarditis* and *mediastinitis.*

Of course, shoulder pains are always a sign of any possible irritation of the phrenic nerve, also of *foreign bodies* in the *diaphragm* itself—even a projectile—often associated with hiccough.

In *splenic affections,* e.g., *perisplenitis,* dominating pains in the left shoulder make the abdominal hypochrondral pains of secondary consideration to such an extent that the patient only complains of the former. This often leads to a mistaken diagnosis of rheumatism of the shoulder joint.

When we can find no clinical or radiologic evidence of changes or motoric defects in the joint, nor typical pressure points, but hepatic or lienic hyperesthesia of the skin or tenderness in the trapezius region and respiratory differences, we must look for other abdominal symptoms if we wish to find the correct diagnosis.

In rare cases only exclusive pains in the shoulder are a sign of *nephrolithiasis*. The lumbar and hypochondral pains, however, sometimes radiate as far as the scapula. Only long experience will prevent mistakes in these cases.

It will be seen that in most of the cases quoted here tenderness in the trapezius muscle is a usable diagnostic sign. I will mention here that this delicacy is often the only available symptom, shoulder pain being absent, and that its discovery is an essential advantage for the diagnosis of the subdiaphragmatic organic diseases in question.

In all these diseases the shoulder pain, whether it is isolated or irradiated, emanates beyond from the phrenic nerve through a symptomatic neuritis (neuralgia) thereof.

Insofar as *idiopathic neuralgia* of the *phrenic nerve* is concerned, I speak with reserve.

In another chain of cases the patient does not point to the shoulder joint or the space between the shoulder blades, but rather to that **part**

of the back which is occupied by the scapula as the seat of pain. Cholelithiasis, for instance, when associated with an ulcer, irradiates its pain into or against the shoulder blade, or else between the two blades.

Sometimes the movement of the scapula is accompanied by a harsh, crackling sound which is noticeable to the patient as well as to the bystander. This noise is apparently due to the formation of an accessory synovial sac and a proliferating hygroma within it between the anterior scapular plane and the posterior thoracic wall, or, perhaps, by bony spurs on the ribs or in the scapula, the work of pleuritis deformans, or due to exostosis, a fracture or to syphilis.

Howsomever, a similar crepitus in the scapula—mostly discernible only in auscultation—also happens in very lean or emaciated persons, no doubt caused by friction between the shoulder blades and the ribs; it may also be due to some muscular action and is most frequently heard in pulmonary tuberculosis. The patient himself is not always aware of its existence. A differential sign between the two forms is the fact that in the latter case it proceeds with a symmetrical rhythm and is painless except in unilateral atrophy of the dorsal muscles.

And yet we come across patients who complain of the annoyance occasioned by this noise, as well as of the pain that accompanies it. Fur-

ther questioning may elicit the fact that the patient experiences the same sensation also in other joints of the body. We are safe in attributing the whole trouble to hysterical causes. Emaciation, often enough superinduced by voluntary action, is evidently the originating factor of the existing neurosis. The diagnosis can well be based upon the claim that the crackling sound prevails in diverse places, and also on the other hysterical manifestations.

Pains in the scapular region may also be the result of some anatomical defect in the shoulder-blades, e.g., in caries or acute osteomyelitis, a fact which is of interest to the surgeon.

We are strongly reminded here of morbid conditions that may occur in the adjacent thoracic organs, especially of the *pleura* in a primary *affection of the lungs.* Not only tuberculosis, or any form of acute or chronic infection, abscess or tumor of the lungs may cause these pains by way of encroachment on the pleura or in combination with pleural affections, but we must also look for *primary* inflammations or neoplasms of the pleura itself as the originating factors. In interlobar pleuritis (empyema), for instance, we are conscious of severe pains in the region of the shoulder blades, proximal to the spina scapulæ. In suppurative, inflammatory processes of the bronchial glands

these pains are rather of an interscapular nature.

Matterstock mentions a peculiar kind of shoulder pain which deserves mention. When we glide the hand with firm pressure along the crista scapulæ, the patient who is suffering from lobar pulmonary tuberculosis speaks of severe pain in the affected section.

Not counting any of the previously enumerated ailments and leaving out of consideration even a possible *subscapular bursitis* as sources of pain, we are not falling into an error when we accept a painful affection of the shoulder blades as a manifestation of *intercostal* neuralgia. The characteristic signs are: the seat of the pain corresponds with the anterior angle of the scapula, and the presence of pressure points, especially in the vertebræ.

Bilateral shoulder pains should direct us to recognize deuteropathic intercostal neuralgia due to a primary affection of the vertebræ or of the vertebral joints, of the ribs or of the spinal contents.

One-sided pains of great intensity in one shoulder blade only may also be the initial symptom of an extramedullary tumor of the spinal cord. Another possibility is a morbid process within the chest, especially of the posterior mediastinum in the form of intercostal neuralgia,

or an aneurysm of the aorta or a broncho-sarcoma.

There is a special form of pain at the *apex* of the *scapular angle.* All morbid processes that possibly may occur in the thoracic cavity or its wall may here be involved, and must be taken into consideration whenever these pains come under our observation.

I strongly emphasize the fact that these pains may, when coupled with local pressure sensitiveness, figure in a number of subdiaphragmatic affections either as the most distinctive irradiation pains or, at any rate for a time, as the only and exclusive pains of the fundamental disease. This is the case in cholelithiasis, in cholecystitis, in ventricular ulcer or carcinoma. I shall refer to this again later on.

Pains *between the shoulder blades,* at the apex of the scapula, are of varying genesis according to their localization either *between the scapula and spinal column* or *along the spinal column.* In the latter case the differential diagnosis must follow the same course as outlined in the chapter on "Pains in the Sacrum."

Affections of the spinal column (bones, joints and cartilages) and its contents (membranes of the spinal cord, nerve roots and the spinal cord proper) claim our attention. Pains in the spinal column at this level may originate from an attack of functional neurosis (spinalgia, spinal

irritation). The diagnosis must proceed from the fact that the pain radiates from the sacral region upwards into the shoulder blade, that the skin is here exquisitely hyperesthetic, and that the other typical symptoms are present.

Other morbid conditions in the *posterior mediastinum* loom up as possible causes of these pains. *Arrosion* of the *spinal column, aneurysm* in the *arc* of the *descending aorta,* carcinoma of the esophagus involving one or several of the vertebræ, are all possibilities. The diagnosis can only meet with an obstacle when the patient has no other complaint to make beyond these local pains, as may very well be the case in carcinoma of the esophagus or in the earlier stage of aneurysm of the descending pectoral aorta. If there is no pulsation or bruit laterally to the spinal column, we are certain to find a retardation in the crural, as compared with the radial pulse, also a very conspicuous smallness of the former as compared with the latter, and above all the Roentgen picture will assist in clearing away doubts that may exist in the diagnosis.

There are certain affections of the posterior mediastinum which do not directly involve the vertebral column, and yet are associated with pains in that part of the shoulder blade which is adjacent to the spine. If we find here interscapular sensitiveness to pressure and percussion

in the spinous processes of the first to fourth vertebra, we may be sure of an affection in the mediastinum, or in the heart and the larger vessels. Hyperesthesia between the fourth and eighth vertebra, or even below the latter, points to affections in the stomach, lower esophagus, liver, ventricular ulcer or gastric carcinoma.

Pains *between the shoulder blades,* or between the *scapula and the vertebral column,* give us the impression that there is some primary disease of the spinal column or its contents which exerts a pressure on the neighboring nerve roots. There is quite a list of such possible ills, viz., caries, spondilitis, new growths, divers affections of the vertebræ, meningitis, meningeal apoplexy, hypertrophic cervical pachymeningitis (syphilitic), tumors of the spinal cord or the membranes thereof, inflammatory conditions of the cord with lateral meningitis and hematomyelia, not to forget foreign bodies in the intervertebral foramina and the massing together of neoplasmatic metastases or leucemic pseudoleucemic or lymphogranulomatous infiltrations.

Intercostal neuralgia between the shoulder blades is another font of pain. It may be occasioned by local conditions, e.g., by disease of the neighboring ribs (neoplasms, syphilis, etc.) or by some malformation in the spine (scoliosis) or a bronchial or pulmonary affection (bronchial carcinoma) or of the mediastinum (aortic aneu-

rysm, lymphosarcoma, etc.), or it may be merely a pain caused by fatigue or exhaustion, or it may be the forerunner of herpes zoster or its companion.

Pains which are due to overtaxing or overtiring of the spinal column in the sense of *vertebral insufficiency,* belong to this class. The patient does not of his own accord generally complain of backache, but rather of weariness and languidness. But upon closer scrutiny we find a decided tenderness on pressure and tapping in the spine, exquisitely so between the shoulder blades and laterally from the corresponding vertebræ, also in the lumbar vertebræ when tapped or pressed through the abdominal wall. We also find complicating gastric disturbances. This form of insufficiency may be observed in almost any morbid condition of the spinal column, such as scoliosis, osteomalacia, chronic ankylosing articular inflammation of the spine, after injuries (carrying heavy burdens), in anomalies of the spine, especially at the age when the normal bearing power of the column is on the wane (fortieth to fiftieth year of age), not any the less after wasting diseases or abnormal physical exercises or overexertions. The *costal region* is, of course, included in this section. There may be tuberculous caries, syphilitic periostitis, arrosion of one or several ribs caused by an aneurysm or similar materies morbi.

Next in order are the morbid affections of the muscular plexus. Foremost among them is *rheumatism of the interscapular muscles.*

Rheumatic myalgia, myositis in syphilis (predominantly nocturnal pains!), polymyositis ossificans, and also trichinosis are harbingers of heavy interscapular pains. We find them in many vocational pursuits, e.g., among tailors, cobblers, seamstresses and miners, and others who have to lean over their work most of the time.

Pains in the back count among the primary signs of *tetanus.* The diagnosis may be disturbed when the attack comes on top of a severe cold thus misleading to the erroneous classification of "a rheumatic cold." A study of the other tetanic symptoms should correct the mistake.

In *chlorosis, anemia, asthenia* and *orthotic albuminuria* we get plenty of grumbling about backaches and bodily weariness. The fact that rest in bed brings relief is a strong point in favor of the correct diagnosis. The claim made by some patients that leaning the shoulders against the back of a high chair brings relief, I do not consider of much value in this connection.

Back-shoulder pains due to some *myocardial weakness* must be included in this paragraph. General fatigue, pains in the loins or calves, ache and dullness in the head, dyspeptic troubles,

dyspnea, cyanosis even after minimal physical exertion, nycturia or oliguria and changes in the heart itself, are all good pointers for the diagnosis.

The dragging, cutting pains in the interscapular area can always be recognized as the earliest symptoms of pulmonary tuberculosis, unless they are due to a localized dry pleuritis or to hyperesthesia of the skin. Such initial symptoms as painful palpation of the musculus cucullaris (also of the sterno-cleido-mastoid) and sensitiveness on pressure in the cervico-brachial plexus above the clavicle should give a sound foundation to the diagnosis.

In various diseases of the mediastinum the pains lay a preferential claim to the region between the left scapula and the spine. If they come of a sudden they signal an inflammation of the cellular tissue of the posterior mediastinum or its glands. *Acute mediastinitis* is generally of a phlegmonous nature, though it may be also in the form of a localized mediastinal abscess. It issues from some morbid process of an adjacent organ, inflammations or new growths of the esophagus, pericardium, the pleura or lungs, or some subdiaphragmatic phlegmon which has extended through the esophageal hiatus into the posterior mediastinum, or from a primary vertebral or costal lesion, tuberculous caries being the most common among these.

Prevertebral actinomycosis may develop from the oral cavity through the cellular tissue of the neck or also from the esophagus. The diagnosis should not be difficult if the primary port of entry of the actinomyces is found in the oral cavity (dental actinomycosis) or in the neck or in the respiratory tract. (Remember, too, that pulmonary actinomycosis may also be the secondary outcome of a prevertebral actinomycotic phlegmon.) Examination of pus and sputum and the Roentgen-ray are important adjuvants.

Acute inflammations of the *anterior mediastinum* also give rise to pains between the shoulder blades. The diagnosis in this as well as in the former instance is furthered by the fact that the pains are interscapular and behind the sternum, and by the symptoms of mediastinal irritation or displacement of any kind (trachea, esophagus, bloodvessels, nerves, glands). Febrile conditions are of a septic character. A cutaneous edema in the jugulum on one side of the sternum or above the clavicle and a swelling in these sections or in the supraclavicular fossa must be taken as warning signals of a possible perforation of the mediastinal abscess. The X-ray should not be neglected in all these cases.

Dark field radiology is of special import in *chronic fibrinous mediastinitis* which so often

follows in the wake of indurated pleuritis, mediastinal lymphadenitis and inflammatory affections of the lungs (phthisis, diseases of the esophagus). The pains are retrosternal, or interscapular with possible dysphagia or hoarseness. Additional symptoms are a descending laryngeal pulse (*Oliver-Cardarelli* symptom), aneurysm and dilatation of the aorta, mediastinal and intrathoracic glandular tumors, enteroptosis and unusually accelerated heart action. However, the pains between the shoulder plates may in some cases be the only perceptible sign.

Inflammatory conditions in the *mediastinal cellular tissue* as well as in the *retromediastinal glands* are bound to give rise to interscapular pains localized sometimes only on one side of, but mostly within the spine itself and generally on the level of the 2. to 4. dorsal vertebra. Angina with secondary infectious symptoms and secondary hemorrhagic nephritis are often the precursors of this situation. After the angina has run its course and only nephritic residues are left behind, we will often enough be able to observe moderate pains between the shoulder blades, not infrequently accompanied by difficulty in swallowing, but always by a perceptible rise in the temperature, to disappear gradually within seven or eight days. I incline to the belief that these pains are attributable to a receding retromediastinal lymphadenitis originating in the

anginose condition, but I have no definite proof for it.

More often, however, these pains are basic in a tuberculous mediastinal lymphadenitis, primary as well as that which runs parallel with a fully developed pulmonary tuberculosis. Of other causes it is meet that I mention here, muscular affections, hyperalgesia of the skin, affections of the bones and mediastinal pleuritis (more about the latter later on) and in some rare cases an acute phlegmon of the mediastinal cellular tissue proceding from a tuberculous cavity in the lung. Chronic indurated mediastenitis and plexus pains belong here also.

We can safely run the risk of looking upon these lymphadenitic conditions as an early symptom of phthisis. They are disclosed by spasmodic coughs, cyanosis, dyspnea, interscapular smothering, the Roentgen-ray, sensitiveness on pressure either on the side of the 2. to 7. dorsal vertebra or the corresponding spinous processes, still more so by the pressure of enlargements in the lymphatic glands, palpable in the median axillary line of the thorax, chiefly in the 4. intercostal space. But, be it said, the last named condition does not in many cases came into evidence until the tubercular state is fully developed in the lungs. If pains are evinced in the aforementioned location during esophageal probing (*Neisser*) we have definite proof of

glandular enlargements. A trial injection of tuberculin followed by very pronounced shoulder pains is an important sign for the diagnosis.

Lymphogranuloma of the mediastinal glands and *anthracoid conditions* of the peribronchial glands claim our attention also. The very nature of the latter affection is bound to cause pain in the indicated place, and so do also any chronic inflammatory or indurated processes which may originate from it in the periglandular mediastinal cellular tissue (around the aorta, esophagus or trachea). More frequently interscapular pains are induced by *neoplastic diseases* of the *mediastinum* and foremost of the mediastinal glands, be they of a primary (lymphoscarcoma) or metastatic character. As a rule the pains are not severe or at any rate are of a negligible quality even when the Roentgen plate shows already deep shadows. Radiology has the same diagnostic value also in *bronchial carcinoma.* Additional diagnostic signs are: hemoptysis in persons of advanced age with negative tubercular conditions, bronchial stenosis, fetid bronchitis, abscess or gangrene of the lungs, apyrexia or subfebrile temperature and cachexia.

Fresh localized pleuritis and pleural adhesions also give rise to such pains which are either of a lasting or only of a temporary tenure but are often exacerbated by local pressure, coughing or deep breathing, etc. Such a localized pleuri-

tis is apt to involve the *posterior mediastinal* or the *interlobar visceral pleura,* or may degenerate into an interlobar tuberculosis or also non-tuberculous pleuritis.

Patients afflicted with *bronchial asthma* or asthmatic bronchitis feel these same pains between the shoulder blades. The diagnosis can here always fall back on the peculiarly singing character in bronchitis, the increased volume of the lungs, the typical attacks, examination of the blood and sputum (*Charcot's* crystals, *Curschmann's* spirals, massive eosinophile cells, bacterial deficiency).

Little attention has been given in this connection to chronic *tracheitis* and chronic asthmatic tracheobronchitis. In these cases the interscapular pains are generally superceded by the parallel painfulness felt behind the sternum. I have observed this in patients suffering from recurrent hay fever (conjunctivitis, rhinitis, bronchitis and asthma) when there were no traces of neurotic stigmata.

In *acute bronchitis,* especially of the right large bronchus, a dull, burning, stitching pain between the scapula and spine is at times a matter of complaint, possibly due to consensual peribronchial lymphadenitis.

In *lobar pneumonia* the patient is likely to complain of backache even before the clinical symptoms are definitely developed. This is

particularly so when the inflammation has been restricted for some time to the hilum, that is to say before the parietal pleura has been affected. These pains would in such an event come from the morbid spot in the mediastinal pleura as has already been pointed out in a previous passage. This fact coupled with the manifestations of compression and enlargement in the posterior mediastinum should enable the shrewd observer to recognize the true nature of the disease, especially if he calls the X-ray into service.

More frequently the causative element is found in *diseases of the esophagus.* If it is a case of carcinoma the osseous spine need not be implicated, but the immediate surroundings of the esophagus are bound to be affected. The diagnosis should result from the collateral symptoms. In the first place note difficulty in deglutition, then signs of congestion or enlargement of the esophagus, the verdict of the probe and the evidence of the X-ray plate. When the pain manifests itself only during the act of swallowing or is rendered more acute by it or by the introduction of the stomach sound we have additional proof of the existence of an esophageal carcinoma.

Among the other painful diseases belonging here I will mention peptic ulcer, syphilitic and tuberculous tumors, suppurative conditions of the mucous membrane due to acetic corrosion, sten-

osis or dilatation of the esophagus associated with pressure and oppression. The patient often complains also of synchronous pains behind the sternum, but at the lower end of it, in peptic ulcer. Still I have seen cases in which no companion pains were observed and the interscapular pains were quite independent of the act of swallowing but set in during the night time, being of an intensive, tearing, drawing nature, especially in carcinoma or sarcoma of the esophagus.

Rupture of an *aortic aneurysm* into the gullet or of an esophageal carcinoma into the trachea will always provoke interscapular pains, although they are, as a rule, located by the patient in the breast.

In abdominal diseases the pains extend frequently into the interscapular district, though in bilious colic and duodenal ulcer they prefer the right, and in ventricular ulcer, rupture or perforation of the stomach and splenic affecting the left shoulder. In ventricular pneumatosis they also radiate into the retrosternal region.

In subdiaphragmatic diseases pains between the shoulder blades are the most prominent, at times even the only symptom that presents itself to the observing mind. Cholelithiasis, for instance, is one of these affections. But existing doubts will be dispelled if we find that the cucullaris pressure pain is unilateral and that pressure

in the gallbladder region increases the interscapular pain.

Pains between the shoulders combined with sacralgia are not uncommon in patients who suffer from abnormal flatulence or defective flatus due to chronic intestinal catarrh, or from obstinate constipation, or also from abdominal plethora, sclerosis of the intestinal arteries, portal congestion, phlebitis or phlebosclerosis of the visceral veins, (partly due to nervous conditions or only to chronic obstipation, or also to swallowing air). Complaint of abnormal gas production, visible distension of the belly and if needs be the Roentgen pictures are the foundations of a true diagnostic finding. Appropriate therapeutic measures will confirm it.

Diseases of the gallbladder, of the stomach (ventricular ulcer or carcinoma), in rare cases affections of the spleen, likewise morbid conditions of the pancreas, in fact, of the entire peritoneal cavity, belong in this category.

The interpretation of interscapular pains following upon *diseases of the aorta* is rather surrounded by difficulties. The pains prefer the left side, although at times they invest both sides with a slight punctuation in the left. Of course, I am speaking here of an aneurysm situated at the spot where the pectoral aorta arches off into the descending aorta. The cogent points are: visible and palpable pulsation in the left painful

zone, systolic murmur, mediastinal engorgement and compression, distinct retardation of the crural pulse and that of the abdominal aorta as compared with the pulse rates in the upper thoracic sections, especially the apex impulse, and the radiological finding.

Another peculiarity is that the pains are more acute when the patient lies on his back, but diminish when he turns around to rest on his abdomen. And again they are sharpened by physical exertion such as walking, running or exercising the arms, whereby the aneurysmal sac becomes extended and calls the accompanying aortitis into action.

Similar conditions prevail, however, in any other form of aortic disease, even without the formation of an aneurysm, e.g., in *syphilis* or *sclerosis* of the *pectoral aorta.* There is still another resemblance between the pains in the shoulders arising from aortic sclerosis and those due to aneurysm of the pectoral aorta, but not in a pathognomic sense. It is this: the patient in either case will tell you that the pains are materially softened when he presses the back against some solid object, e.g., the back of a chair, or under vigorous, punching massage in the left interscapular region.

The fixation of sclerosis of the pectoral aorta can be credibly established upon these symptoms even when arteriosclerotic manifestations in the

other aortic and peripheral vessels are wanting. A distinctly punctuated, or ringing second aorta tonus above the descending pectoral aorta, i.e., in the left interscapular space, is a leading sign.

A certain anatomical affection, chiefly the outcome of a true angina pectoris, must be mentioned here. I mean *sclerosis* (syphilitic) of the *coronary arteries,* i.e., a constriction at the point of exit from the ascending aorta. In other words: when a patient tells us that he is molested by rather intensive — sometimes only dull — pains between the shoulder blades (perhaps for months) we should always think of a possible true angina pectoris. The question to decide here is whether we are dealing with a sclerosis of the ascending pectoral aorta or of the coronary arteries, i.e., the ascending portion of the supravalvular aorta. We shall arrive at a decision when we find definite proof of myocardial changes such as chronic interstitial myocarditis, or myofibrosis, or degeneration of the heart muscle, or, maybe, an aneurysm of the heart together with typical attacks of angina pectoris, which latter may come on just as well when the body is in motion or completely at rest.

If these conditions are still further aggravated by acute weakness of the heart, unusual physical debility, failing pulse, pallor of the face, cerebral symptoms, syncopal incidents, we have a

strong intimation of the sudden thrombotic or embolic occlusion of a coronary artery.

A *rupture* of the pectoral aorta generally announces its arrival by a sudden most violent pain between the left scapula and the spine, though ordinarily it is preceded by a series of minor pains. The rupture may find its way into the left pleura, or into the left lung or into the pericardium. There may be present a pre-existing sclerosis of the aorta, an ordinary or a dissecting aneurysm of the aorta. At times we find a pronounced hyperesthesia of the skin in the affected area, but always the typical signs of internal hemorrhage. The outcome is fatal.

We should ever bear in mind that two of the aforementioned diseases may simultaneously inhabit the same body. It is by no means an unusual occurrence that aortic or coronary sclerosis travels side by side with a carcinoma of the stomach or of the esophagus, or a gastric tumor with an arteriosclerotic basis.

Pains in the apex of the shoulder—not always localized by the patient with exactness—ever point to the possibility of a *spinal* or *intracerebral source.* A cerebellar tumor or any morbid condition that encroaches upon the occipital space will cause such pains. The proper diagnosis can, of course, be made from the observation of other typical symptoms. In *migraine*

with an occiptal base the pains travel via the nape of the neck to the shoulders.

"Pains within the range of the scapula," or rather within the circumference of the *supra-clavicular* and *supraspinous fossa* require special attention in this place. I do not refer to the pains which originate in any of the organs that lie within the supraclavicular fossa itself—they are dealt with in the chapter on "Pains in the Neck"—but rather to those which radiate from the nuchal muscles and from the cervico-brachial plexus into the shoulders.

The proof that certain pains are basic in the nuchal musculature, chiefly in the cucullaris muscle, is furnished by the fact that they are felt only, or at any rate are exacerbated, when the individual muscle comes into action. Pressure, stroking or tapping of the muscle creates a painful sensation. This is frequently the case after unwonted physical, gymnastic exercise, in which case the anamnesis should be sufficient ground for a proper diagnosis. Gout and rheumatism and certain infectious diseases, e.g., plague, are other causes that must be considered.

It is somewhat difficult to separate the initial stages of multiple ossifying myositis from those of the acute suppurative form. In both we find local pain, swelling of a solid consistence, moderate rise of temperature, and subsequent muscular induration. When bone tissue sets up, the diag-

nosis will be clear. All the other forms of myositis show no specific tendency to attack the muscles of the neck or back, except acute dermatomyositis in which disease the pains eventually radiate from the brachial muscles into the nuchal and dorsal plexus.

In *angina pectoris* and other cardiac affections (*paroxysmal tachycardia* with nodal heart rhythm, *acute dilatation* of *right ventricle* with venous engorgement), right-sided *trapezius hyperalgesia* in *periappendicitis,* pains in the shoulder muscles, especially in the cucullaris and in the sternocleidomastoid are experienced. The patient rarely complains of them, but we find hyperalgesia when kneading or roughly stroking the affected muscles.

In true *angina pectoris* and in *aortalgia* subsequent to sclerosis of the aorta or of the coronary arteries the patient often complains about pains in the left shoulder. They are localized either in the supraclavicular or supraspinous fossa and are felt with physical movement or exercise, but also at times when the body is at rest. Muscular hyperesthesia is not in evidence. In some cases the patient does not seem to notice the pains at all, but when asked will speak of a slight pressure in the chest but without painful sensation. This is an important danger signal not to be ignored in sclerosis of the aorta or the coronary arteries.

That the *cervicobrachial plexus* is the causative factor of predominant pains in the shoulder is demonstrated when the patient complains of a sensation of weight, rigidity and acute painfulness in the affected part. He inclines his head backwards and away from the tender side (shaving posture). The nerve fibres above the clavicle are sore to the touch, the pains in the periphery of the involved plexus radiate into the arms, paresthesias and motoric weakness are noticeable in many cases.

These pains are often due to new growths which are forming in this region, especially in the supraclavicular fossa, but may also branch out from some morbid process in an organ which has only a subordinate anatomical relation to the supraclavicular group, or none at all. They may be in part the manifestation of a *polyneuritis* or a *mononeuritis* with a local cause perhaps in the spine or in the spinal canal or in some morbid process that has already reached the corresponding supraclavicular fossa. There is the possibility of a swelling or a tumor developing in the clavicle (osteomyelitis, caries, gummata), a disease or enlargement of the supraclavicular lymphatic glands, the formation of an aneurysm of the subclavian artery—all easy to recognize—or the elongation of the costiform process. In the latter case the X-ray will furnish the proof where palpation fails.

In *omarthritis* the pains are not felt so much, if at all, in the joint itself, but rather in the zone of the cervico-brachial plexus above as well as below the clavicle. (See also chapter on "Pains in the Extremities.")

These plexal pains go, however, together also with other quite independent diseases. It is by no means uncommon that a patient who is suffering from an *aneurysm of the aortic arc* complains solely of pains in the right shoulder if not in both shoulders, whence they radiate first into the right and later on into both arms. The same may be said of simple *sclerosis* or *dilatation* of the aorta. A proper consideration of the typical symptoms of these diseases should reveal a correct diagnosis.

We shall find pains of a minor degree in the chest but such of decided acuteness in the left supraclavicular fossa in every attack of *angina pectoris* based upon sclerosis of the aorta.

Aneurysm of the *anonymous artery* carries with it very severe pains in the right shoulder and arm. (See "Cardiac Pains.")

Solid *tumors* of the *mediastinum* produce shoulder pains as an initial symptom. The diagnosis can be made from the Roentgen picture, other typical symptoms such as mediastinal sensitiveness to pressure, localized lack of resonance in percussion, tympanitic dullness. Bronchial sarcoma is worthy of mention here also.

The sudden appearance of a pain in one of the shoulders or in the supraclavicular fossa should arouse our suspicion of a lesion in the corresponding *apex of the lung* or its pleural coating. *Pneumonia* of the *apex* accompanied by pleuritis of the apex pleura tends to an acute perineuritis of the cervico-brachial plexus and thus gives rise to considerable pain in the shoulder. We should recognize this condition from the characteristic symptoms of the disease described elsewhere. If the pains have been present for some time we should look for some chronic disease, especially *tuberculosis* of the pulmonary apex. The pain combined with tenderness in the cervical plexus when the pressing finger glides slowly in a forward movement over it, will give proof of an indurated pleuritis and subsequent chronic perineuritis (eventually neuritis) of the cervico-brachial plexus.

The corresponding primary symptom of this apex disease manifests itself in the shape of a painful sensation in the cucullaris muscle when it is rolled about or tapped with the finger. The patient is not always aware of its presence until the sore spot is touched which may also be the seat of an amyotrophic condition.

Interstitial or chronic pneumonia, apex pleuritis either fibrinous or exudative (suppurative), also superior lobular tumors are generators of scapular pains, which become a typical diag-

nostic symptom when aggravated by moving or raising the arm up high.

Pains in the fossa supraspinata emanate also from an *interlobar empyema.* They disappear when the pus has been drained off.

Proliferating growths on the superior pulmonary lobe frequently cause similar pains when extending upwards they encroach upon the plexus that lies above the apex. I refer to malignant and cystic (echinococcic) neoplasms. The accompanying pain is of importance for diagnostic purposes.

Shoulder pains awakened by tapping or rotating the cucullaris muscle may also be due to some inflammatory process in the diaphragmatic region such as a *local pleuritis,* or also a *perihepatitis,* or any kind of inflammatory *subphrenic lesion,* or perigastritis or perisplenitis. In these cases a rearward pressure of the fingers will elicit pain within the borders of the outer and median third of the right cucullaris muscle.

Analogous conditions may prevail in *acute appendicitis* even when the liver is intact and the subdiaphragmatic, subhepatic and pleural cavities are not involved (subhepatic suppurations may give rise to pains in the right shoulder). The patient complains of pain in the iliocecal region when drawing a deep breath. In *chronic appendicitis* similar pains at times make their appearance, especially in the right

supraspinate fossa and below the right clavicle. The long continued fever and the emaciating effect on the patient are apt to lead to an erroneous diagnosis of tuberculosis of the right apex.

But the pains may be also localized in the left side when the primary affection is in the *left lobe of the liver* (gumma, carcinoma) with subsequent perihepatitis.

I include here diseases of the *pancreas* and of the *neck*.

Backache

The term "backache" carries a double meaning. In the language of the patient it may mean a pain which is felt either along the whole or nearly whole of the vertebral column, or only in a limited zone or on one side or the other of the spine, that is to say in the region that lies between the loins and the neck. As this book deals with nuchal and scapular pains in separate chapters, the reader will know that the pains described in this chapter affect the district which lies above the lumbar and below the interscapular region.

Some patients will complain of pains in the back which by other patients are described as pains in the chest. We can easily guess that that really refers to retrosternal pains which belong to the chapter on "Pectoral Pains."

With these limitations in view we will first of all consider the spine as the seat of pain claiming our attention. This includes all the acute and chronic diseases of the osseous part of the column as well as the diffuse affections of the spinal contents, viz., the cord, and its substance and the meninges.

So far as the acute affections of the different vertebræ themselves are concerned full details are given in the chapter on "Pains in the Sacrum."

As a preliminary remark I mention here that a syphilitic vertebral periostitis may well involve the entire spinal column even in an acute fashion thus simulating acute articular rheumatism. So far as the chronic vertebral affections are concerned I wish to emphasize the fact that if the whole of the spinal column is comprised in the articular process a very characteristic picture is framed: the physiologic spinal curvatures are missing, the entire column has the appearance of a rigid, straight tube, the patient bends his knee- and hip-joints in order to maintain an upright posture, and the head is pointedly inclined in a frontal direction. Such a diffuse, chronic articular inflammation of the spine makes us think of a possible ankylosing vertebral inflammation, of rhizomelia, or arthritis deformans, such as we have already discussed in the chapter on "Pains in the Sacrum," but, be it said, identical conditions may also arise from a gonorrhoic source. The fact that the gonorrhea happened some time, perhaps some years ago, that the patient is still of youthful age, that the erstwhile gonorrhoic attack was associated with gonorrhoic arthritis in one joint or another, e.g., in the knee, should always prompt

a positive diagnosis. Perhaps, the day is not far off when the Roentgen-ray will be an adjunct in such cases. That syphilis is another etiological factor has already been mentioned in the chapter on "Pains in the Sacrum."

The vertebrae themselves are also a possible focus of pains. Habitual *scoliosis* claims here our attention, and so do quite a number of morbid conditions in the entire *osseous skeletal frame.* For details see the chapter on "Pains in the Bones." I only mention here passingly, pains in the bony spine or in any part thereof may be occasioned by osteomalacia—especially senile—by multiple or diffuse neoplasia of the vertebrae as a manifestation ex parte of diffuse osseous neoplasia of primary or secondary origin, also by *Kahler's* multiple myeloma, but rarely by a late tracheitis or by leucemia. I make these remarks here because primary and at times exclusive complaints of these pains are ripe in these cases and may even be accepted as early symptoms of a disease confined wholly to the spinal region. Remember also that backache combined with pain in the head and in the extremities—often of a lancinating character—are not infrequent companions of acromegaly.

Local pains in the *bony spine* may be recognized from divers objective symptoms, such as deformities in the sense of angular or arcual kyphosis, local painfulness aroused by percus-

sion or digital tapping, radiating pains in the waistline or in the extremities, pains by sudden pressure on or impaired motility of the spine, the secondary reaction of the disease on the spinal cord and nerve roots, and, last but not least, from the X-ray picture. Upon these foundations we can readily build up the special diagnosis of appurtenant diseases such as tuberculous spondylitis, neoplasms and syphilis of the vertebrae.

In *chronic tuberculous spondylitis* the pains are mostly localized between the dorsal and lumbar or between the cervical and dorsal vertebrae. But they may be felt in any other place of the spine and come on when the patient has been standing on his feet or been walking for some time. In the initial stages of the disease we may find very acute local painfulness when we ask the patient to lie down in an arched position, i.e., resting the body on the back of the head and on the heels. Often enough the patient does not succeed in assuming this posture owing to the exceeding painfulness caused by the attempt. Furthermore there is local sensitiveness on percussion and pressure especially in the spinous processes of the affected vertebrae, also when touched with a hot sponge or the electrode; there is distinct motoric restriction, exquisite accentuation of pain by sudden brisk pressure on the spine from above, e.g., a jolt on the

shoulder or on the top of the head, and angular kyphosis. The latter does not so clearly manifest itself in some, particularly in the lumbar vertebrae and generally only in the form of a diminished physiologic convexity. The X-ray findings and the manifestation of compression of the spinal cord and its membranes and also the intraspinal nerve roots complete the directions for the diagnosis.

The same symptom complex applies to every other form of *spondilitis* or *caries* of the spine. There is a chronic form of spondylitis which reacts painfully to the aforementioned downward jolt on the shoulder, is accompanied by fever and easily mistaken for Pott's disease. It is due to staphylococcus infection, is of a gummatous, typhoid, posttraumatic or metastatic character, distinguished by central necrosis or abscess of the bones, but rarely of a gonorrhoico-metastatic or actinomycotic nature. Similarly a chronic form of caries with formation of angular kyphosis the special diagnosis of which can only be made from the combined clinical picture, with the aid of the Roentgen-ray, from the serological reactions and, so far as the actinomycotic state is concerned, through the proof of the latter's pressure in the respiratory tract. Echinococcus and an aortic aneurysm may also attack the spine and cause atrophy or kyphosis in several vertebrae. The X-ray is here "first aid."

Spondilitis may set in as an acute affection and run its whole course as such, e.g., staphylococcic osteomyelitis, strepto-staphylo-mycosis, or other acute infectious spondylitic conditions often coupled with chills and high fever, local stiffness, local symptoms in the spinal cord and its meninges or nerve roots, or local edema. The diagnosis will be guided by the X-ray, the evidence of previous infection (in acute vertebral osteomyelitis, for instance, antecedent angina or other diseases leading to suppurative metastases) and the co-existence of an acute suppurative osteomyelitis in some other bones.

But let us remember that a tuberculous spondylitis may be, as is often the case, the immediate successor of some acute infectious disease. The chronic syphilitic and the actinomycotic forms have the same pernicious habit. But this is also the case in other morbid processes, e.g., in aortic aneurysm which, though it be chronic in its nature, will yet at times suddenly and without apparent provocation arouse very distressing pains in the vertebral bodies. The same conditions prevail also in metastatic vertebral carcinoma.

It may do no harm to remind the reader that tuberculous spondylitis is capable of attacking the spine in several distinct places at one and the same time, and that *arthropathic vertebral inflammations* may be formed, e.g., in tabes.

In so far as *neoplasms of the spine* are concerned the internist must know that in sarcomatous conditions of this organ the primary forms prevail, while the secondary forms are more in evidence in carcinomata of the vertebrae. Myeloma and lymphogranuloma are also possibilities, but the diagnosis of these two primary neoplasms is more difficult to make than that of the secondary malignant neoplasms (nearly always carcinomatous) for the simple reason that in the latter case the primary neoplasm is capable of definite proof no matter whether the secondary vertebral neoplasm has arisen from a carcinomatous organism adjacent to the spine or originates from a metastatic condition. If the former is the case it may be rather hard in the beginning of the disease to determine whether the pains are really due to a co-affection of the spine itself or only to a compression of the peripheral nerve trunks. I think a satisfactory solution is to be found in radiological examination and with the aid of a correct clinical differentiation between a neuritis of the nerve roots and that of the nerve trunks.

The local manifestations in *vertebral neoplasia* are principally the following: marked intensity of the local pains. They are of a boring character and never cease even when the patient is at rest (it is otherwise in caries). The irradiating pains are often accompanied by

herpes zoster (this does not seem to happen in caries) or by paraplegia dolorosa (an important diagnostic factor). The local pains in the dorsal vertebrae are felt in neoplasms on the side of the spine, but in caries more laterally in the thorax. In caries and tuberculous spondylitis pressure on and tapping of the spinous processes or a brisk jolt from above stimulate the pain, while in neoplasia deep pressure laterally from the spinous processes has this effect. In caries there is angular kyphosis, in neoplasia it is—if any at all—of an arcuary form, that is to say several vertebrae become evenly prominent.

It is not necessary to consider here other primary tuberculous affections such as enlargements or scars of the glands, abscesses in dependent parts, or the age of the patient, all of which are factors of interest in caries.

The metastatic vertebral carcinomata are by far the most common forms among the neoplasms of the spine and are from the diagnostic standpoint of interest insofar as they, like the primary new growths, escape recognition, especially in the thyreoid glands, in the ovaries, in the kidneys (hypernephroma) and the suprarenal capsules, in the prostate, sometimes also in the mamma or in the testicles. Unless we find additional affections of the bones (multiple primary tumors, e.g., myeloma, or multiple metastatases) the diagnosis is ever restricted to

the aforementioned manifestations and to the X-ray.

In these cases errors are bound to slip in in the identification of neoplasms of the spine and arthritic affections of the vertebrae, the *ankylopoietic spondylarthritis* or *deforming arthritis.* And this is more likely to happen when the patient is suffering also from a deforming osteoarthritis of some standing and complains of aches in the back and spine. We shall be enlightened by the milder intensity of the pains, by the predominancy of the rigidity over the pains, and above all by the X-ray picture. The blood test and the more rapid appearance and progress of the vertebral neoplasm are also telling factors. This is preeminently so when an apparent vertebral tumor arises from a chloroma.

Uric arthritis in the appropriate vertebral joints must not be forgotten. It may be preceded by *gouty* changes in other joints (podagra, etc.), yet this disease, may under circumstances, be the first manifestation of the morbid condition. Look for retarded nucleic metabolism as the final means for a correct diagnosis.

Diseases of the adjoining ribs and of the spinal contents are further causes of backache. Pains which occupy the whole or at least the largest part of the back are not infrequently the initial symptom of acute as well as chronic diffuse *intraspinal morbid conditions,* not only

of the meninges but also of the very substance of the spinal cord, for instance, in acute poliomyelitis or multiple sclerosis. In *pellagra* the same observation can be made, although in this ailment the pains are sometimes only felt in the scapular region, no doubt as a reflex action of the toxin on the central nervous system.

Acute spinal meningitis belongs to this series. The *chronic,* above all the postraumatic, serous forms, it must be mentioned, often lack entirely all the typical meningitic symptoms and travel under the mask of neurasthenia; but the diagnosis can secure evidence from the complaint of headache, backache, vertigo, abnormal excitability and psychic moodiness, coupled with the proof of increased pressure in the spinal fluid.

Encroaching diseases of the brain that raise the intraspinal pressure of the fluid, especially when situated in the occipital fossa, produce pains within the nape of the neck and in the back. *Acromegaly* has the same failing.

Myxedema arouses pain in the sense of rachialgia: extreme tired feeling, especially in the early morning hours, heaviness and pain in the extremities, menstrual disturbances (menorrhagia, amenorrhea), chills, subnormal temperature, hoarseness and obstinate constipation are the predominant signs.

Any kind of irritation of the *posterior roots of the spinal cord* is bound to react with a pain-

ful effect on the back. These pains are of an encircling nature such as is described in the chapter of "Pains in the Chest." If the pains are confined to the level of a certain vertebra we must bear in mind that they may originate just as well from the bony part as from the intervertebral foramina, the spinal canal or the spinal cord itself, or may also be due to a primary affection of the spinal ganglion (herpes zoster). The pains may set in in an acute fashion (poliomyelitis, myelitis, multiple sclerosis, hematomyelia, thrombosis or emboly), or may also take a decidedly chronic course. They are felt as a rule on both sides, but with exceptions. We must be guided by the seat and development of the causative factor.

A purely functional *neurosis* provokes pain in any isolated place or in several places or also in the entire spinal column (in the whole of the back). Such a rachialgia or spinal irritation is simply a manifestation in part of an hysterical or neurasthenic disease of the whole nervous system. Insofar as the spinal irritation is concerned we must not only expect a feeling of painful fatigue, but also an affection of several vertebrae and a strongly marked, often excessive hyperesthesia or a hyperalgesia of the skin over the stricken vertebral zone. The softest touch is extremely painful, much more so than a prolonged, increasing, deep pressure. The

slightest movement of a cold or hot sponge or the application of an ever so weak electric current over the affected part produces a most violently painful sensation.

The rachialgic pain in hysteria is characterized by the fact that, when the attention of the patient is diverted, it abates or vanishes altogether. Yet, for practical purposes this symptom is not always applicable, for the reason that there are many other anatomical lesions which respond in a similar fashion, especially when associated with neurotic conditions.

Owing to the abnormal vascular irritability in spinal irritation we often find in the painful zone a marked vasodilatatation to even a slight mechanical stimulus (vasomotoric paresis!) and vice versa also generalized vasomotoric manifestations in the sense of swooning seizures, in rachialgia and hysteria, also cerebral convulsions. In accordance with the individual neurotic constitution of the patient we may also observe other concomitant vaso-sympathetic manifestations such as abnormal pallor, dizziness, palpitation of the heart, nausea, vomiting, polyuria, etc. The neurasthenic and hysteric stigmata of the patient are further adjuvants of a correct diagnosis.

Nevertheless, it is sometimes rather difficult to separate such a purely functional disturbance from an initial anatomical lesion, principally

from an incipient vertebral caries, because rachialgia, or else spinal irritation may under circumstances be the only symptom of the existing neurosis, despite the fact that it is confined to one solitary vertebra. In such doubtful cases, especially when a deformity is not yet apparent in the spine, much help will come to us from the consideration of the following points: in neurosis the superficial touch is more painful than deep pressure, in the anatomical lesion (spondylitis) the intensity of the pain is in proportion to that of the pressure; in spinal irritation the attack affects several vertebrae and pressure and tapping are felt in like measure in each of them, in spondylitis and new growths only one vertebra is sensitive (but not necessarily so). In spinal irritation and in rachialgia there is no locomotoric spinal restriction, on the contrary movement and diverting of attention ease the pain, although this may be the case also in rachialgia in which even cutaneous hyperalgesia is at times missing. This naturally complicates the differential diagnosis again. But if the patient finds momentary relief from pain when lying flat on his back despite spinal motoric impairment, we can safely decide in favor of rachialgia. Nevertheless, the same phenomenon is apt to occur in tuberculous spondylitis. Otherwise only protracted observation and the Roentgen-ray will furnish the necessary evidence.

There is still another point which we must bear in mind, viz., that it is by no means impossible for tuberculous spondylitis developing from hysteria; likewise that the presence of an abscess in a dependent part indicates vertebral caries, although in the latter instance even a clever diagnostician may be misled when dealing with a case of hysterical rachialgia in which a reflex muscular contraction simulates an abscess in a dependent part.

Kümmel's disease is sometimes erroneously taken for a traumatic neurosis. The nosological status of this *traumatic spondylopathia* is not yet quite clear to me. Some authors refer to it as a rarefying spondylitis, others as a fracture of the vertebra, or an infarction or a softening of the intervertebral cartilage. Perhaps the X-ray will eventually clear up the situation.

Pains in the back and also in the sacrum which interfere with occupational pursuits are frequently the initial symptoms of *dementia precox* in youthful persons. Observation by an experienced psychiatrist is here called for.

Of course, backache is the logical sequel of scapular affections which in their turn may be merely an extension of any acute or chronic inflammatory process in the ribs or spine. I refer to acute osteomyelitis, typhoid or tuberculous osteoperiostitis and to primary or metastatic neoplasmata of the shoulder blades. Their

symptoms are fully described under the headings of diseases of the ribs and spine and in the chapter "Pains in the Bones."

Diseases of the dorsal muscles — see also "Pains in the Shoulder"—are either of rheumatic or infectious toxic (influenza, *Weil's* disease) or myositic (nocturnal backache in syphilitic myositis) or metastatic neoplastic origin. That backaches are caused by them goes without saying. These local pains are also due to fatigue in chlorosis, anemia, asthenia and orthotic albuminuria, or to overexertion in emphysema (recurrent, exasperating coughing). We must add trichinosis and tetanus when the dorsal muscles, especially the extensor dorsi, are involved. Epidemic cerebro-spinal meningitis belongs here also.

I have already mentioned under "Pains in the Neck" that backache and nuchal pains may be occasioned by *septic infections* or *toxic injuries* of the *muscles*.

In the chapter on "Lumbalgia" I have likewise referred to the painful affection of the dorsal muscles caused by sclerosis of the arteries of the lumbar muscles. When resting, the patient is at ease, but when he goes into action the pains in the lumbar region set in either on one or both sides, coupled with stiffness and weakness in the muscular tract. These pains radiate in a frontal direction towards the twelfth rib and the iliac crest, but there is no local

sensitiveness on percussion, neither is there motoric inhibition in the spine. The cause for this lies in sclerosis of the arteries of the lumbar muscles or that of the abdominal aorta, or an aneurysm of the latter.

There is a form of *spinal intermittent lameness,* which is due to sclerosis of the arteries of the spinal cord. We can distinguish it from the commoner peripheral form of intermittent claudication by its peripheral arterial pulse and by the infrequency of pain in the extremities. The patient will, but not of necessity, complain of pains in the back, which are felt in walking but disappear when the limbs are at rest. The legs feel weak and show increased tendon reflex. The patient shows the *Babinski* sign, later spastic paresis and bladder and rectal troubles.

In order not to repeat myself, I refer the reader to the sections on "Pains in the Sacrum, in the Extremities, Shoulders and Muscles," insofar as the *intercostal nerves, lymphatic glands, synovial sacs,* and also the *skin of the back* are here concerned. I will only mention tuberculous diseases of the bronchial glands as factors causing backache and interscapular pains. Localized *erythromelalgia,* although a rare disease, is another link in this chain.

In *posterior mediastinal pleuritis* the pain is felt along the whole length of the dorsal spine, including the interscapular region, no matter

whether it is of a suppurating, serous or fibrinous nature. The latter may be recognized by a pleural friction noise, the other two by tenderness in the spinous processes of the upper and median dorsal column and by an ascending streaky dullness in the left transverse processes. In further extension of the disease the breathing is diminished, suppressed or stertorous, sometimes there is egophony; we also find symptoms of mediastinal compression, e.g., of the trachea, dislocation of the trachea and of the larynx to the right, paralysis of the recurrent nerve, difficulty in swallowing, convulsive coughing, dyspnea, constriction in the branchial portion of the vena cava or the azygos vein, inspiratory constriction of the intercostal spaces and of the thoracic fossæ, chills and high fever, and later on expectoration of fetid sputum due to perforation of the pleural abscesses in the larger air passages. The Roentgenogram shows a distinct dark line along the spinal column.

Posterior acute purulent mediastinitis produces lateral pains of the spinal column. The general septic aspect, mediastinal irritation, consideration of the causal element and the X-ray are the essential requisites for establishing a proper diagnosis. In pulmonary tuberculosis this disease runs a rather insidious course. We notice pains in the back and chest, subfebrile temperature, anemia, emaciation and profuse

sweating. The X-ray can be applied in the fibrinous form only.

Indurated mediastinitis seems to confine itself to dorsal pains. (See chapter on "Shoulder Pains.")

Backache of lesser intensity, but coupled with preponderating retrosternal pains, may be observed occasionally in *bronchial asthma.*

Backaches in the region of the *dorsal spine,* with irradiations in the armpit and nipple, and in the arm itself, should remind us of a morbid condition in the *thoracic aorta* such as arteriosclerosis, chronic aortitis, especially syphilitic, or an aneurysm; also of a deep-seated, painful affection of the *esophagus,* chiefly of a carcinomatous character.

A *rupture* of the *thoracic aorta* is heralded by a most violent pain in the back in the zone of the dorsal spine. It comes on very suddenly and early death ensues.

If the pain is located between the tenth and twelfth dorsal vertebra, it is definitely due to some trouble in the *thoracic descending aorta,* i.e., aneurysm.

Pain to the left or right side of the lower dorsal column is a frequent manifestation of a *recent basal pleuritis,* and therefore also indirectly a sign of various diseases of the lungs and other subdiaphragmatic or even more distant organs which give rise to such a pleuritis.

But there is a form of dry pleuritis which is strictly localized and essentially insidious in its nature and for that reason very hard to recognize. Only a post mortem will reveal the originating cause which may be a small abscess in the lung or a *bronchial carcinoma.*

Both *basal adhesive* and *diaphragmatic* pleuritis are pregnant with pains in the back. (Cf. my book on "Abdominal Pain," Rebman Company, New York.)

These pains are also a common sign, indeed the initial sign of some *pulmonary infarct* preceding by hours or even days the characteristic bloody sputum and the other physical local manifestations. Only *Mahler's* sign, the presence of slight and brief rises in the temperature after a chill and the finding of the source from which the infarction arises can make the diagnosis positive.

Gastric affections make their presence known by pains *in the level of the lower dorsal vertebrae,* specially on the left side of the spine even before the patient begins to complain of the usual dyspeptic or gastric troubles.

But when these pains reach up to the eighth vertebra they constitute the one and only absolute —though not always reliable—sign of an existing carcinoma or ulcer in the posterior gastric parietes. They are by no means dependent on the quality or quantity of the food consumed, but rather influenced by the position or physical

movements of the body. There is no tenderness to touch in the epigastric region, but frequently we find hyperesthesia of the skin or of the spinous processes—sometimes only one vertebra is involved—in the affected area. The pains resemble at times in kind and intensity those observed in caries. In some cases of ulcer in the pit of the stomach they make the impression of intercostal neuralgia emanating from behind the lower ribs to the left of the median line. A genuine case of ulcus or carcinoma ventriculi! A combination of pains in the back and in the epigastrium strengthens the diagnosis which can be made positive only by chemical and bacteriological examination of the gastric contents, of the feces for occult hemorrhage, and by the Roentgen-ray.

There is still a different kind of backache which occurs in gastric ulcer, but even more so in carcinoma of the stomach. It may set in at the very beginning of, or develop gradually during the run of the disease. We hear the patient complain of periodic attacks of very severe pains, similar to tabetic crises. They persist for hours especially in the dorsal position and are apt to radiate into one or more of the lower left intercostal spaces or to spread fanlike over the lower abdominal region. Practically speaking we are justified in accepting these pains as a symptom of a progressive carcinomatous metastasis in the retroperitoneal glands, a carcinomatous prolifer-

ation in the paravertebral tissues, a constriction of the local intercostal nerves, or a direct invasion of the pancreas by an ulcus or carcinoma ventriculi (ulcus penetrans). There is also the possibility of an indurated growth expanding into the retroperitoneal region. Similar conditions may be occasioned by other localized primary diseases of the duodenum or the pancreas, etc. Vertebral affections, however, such as metastasis, do not occur. This fact combined with a careful X-ray examination should lead to definite conclusions.

Periodic or constant backache, though variable in intensity, in the region of the last dorsal vertebra to the right of the spinal column is a definite sign of liver complaint, principally *cholelithiasis* of the gallbladder and acute as well as chronic *cholecystitis,* in rare cases also of a gallstone that has been lodged in the ductus choledochus above the ampulla of Vater. The patient complains of pains in the back when leaning over or wearing a tight belt (sword belt), when standing erect for a while, or when sitting for sometime in which latter position he finds relief by pressing the body hard against the back of the chair. We find tenderness to pressure and percussion in the affected zone, very likely also in the spinous processes of the 8.-10. dorsal vertebra, hyperesthesia and hyperalgesia of the skin, tenderness in the region of the hepatic fissure, palpable changes in the liver or the gallbladder,

demonstrable urobiligenuria (urobilinuria) and itching of the skin. The proper contemplation of this symptom complex together with the anamnesis ought to forestall any possible error in the diagnosis.

The same means for recognizing the disease are applicable in other forms of cholelithiasis (cholecystitis) except that the pains follow a track which is the reverse of that indicated above. The patient tells you that the pains start in the nape on a level with and also in the shoulder sometimes on the right, sometimes on both sides; thence they travel down the back along the dorsal spine as far as the apex of the liver where they branch off into the gastric region. In some cases they are continuous, in others they come in isolated attacks, and may also be accompanied by the feeling of weight and fullness in the stomach. I saw a case in which these attacks occurred every second night. In another patient, a woman, the pains arose from the level of the liver, ascended on both sides of the spine into the shoulders and deflected thence either into both breasts, or by change into the nape, the arms and the chin. How difficult to separate from a stenocardia!

If certain conditions such as described in "Abdominal Pain" prevail, the pains arise in the left side of the spine at the hepatic level. In other words: the irradiation is erratic in its action.

This caste of pains should put us in mind of a possible *duodenal ulcer* in the male, or of a *duodenal carcinoma* in either sex. The intake of cold food or drink arouses or increases them. All the other diseases which exhibit themselves at the gate of the liver belong here, e.g. appendicitis when the appendix is deflected in this direction, for in all of them this particular portion of the retroperitoneal space is involved.

With the same force all this applies to every *painful liver* complaint as well as to the whole category of *retroperitoneal growths* below the diaphragm (pancreas, abdominal and celiac aorta, retroperitoneal glands, every form of subdiaphragmatic pleuritis, in which we must also look out for tenderness in the spinous processes. For particulars consult "Abdominal Pain," also for *mesenteric* diseases and *volvulus* of the small intestine.

When sudden, very vicious pains in the back with collapse are witnessed, look for a *perforation* of the *stomach* or of the *small* intestine, even though the abdominal symptoms (pain and vomiting) should be missing altogether or be of an uncertain nature (e.g. vomiting occurs also in diseases of the pectoral organs). Howsomeever, true guides are found in the tension of the abdominal muscles, the indrawn abdomen, the exclusively thoracic breathing and the final collapse.

Pains in the Neck

This chapter is devoted to the pains occurring in the restricted portion of the body which connects the head with the trunk. This includes the region of the inferior maxilla as far as the jugular and supraclavicular fossa.

The first disease that engages our attention is *cervical myalgia* localized in the sternocleidomastoid and also in the deeper cervical muscles. When sudden pains set in after taking cold or an unexpected drenching, we more than likely are dealing with rheumatic myalgia and its after-effects, i.e. caput obstipum (wry-neck, torticollis) easily recognized by the etiology and the local tenderness of the superficial muscles, especially of the sternocleidomastoid. However, pain may be also reflected in the latter by a morbid process in the cervical lymphatic glands in which case a false diagnosis can easily deploy. This may be obviated by ascertaining whether the local tenderness is in the muscle itself or rather lies in the deeper tissues beyond it. Moreover, we should bear in mind that stiffness in the neck is often the reflex action of an irritation in the sensible roots of the cervical muscles due to some morbid process in the vertebrae, e.g.

neoplasm or spondylitis, or else of the spinal contents.

Polymyositis and *dermatomyositis* deserve mention here as originators of pain in the nuchal, dorsal and cervical muscles, causing also serious trouble in deglutition. In trichinosis the presence of eosinophiles in the blood should clear the view. In muscular gummata we find an indolent, typically nocturnal pain. The differential diagnosis from carcinoma depends on the outcome of the iodide test and on serological reaction.

Pains in the cervical and nuchal muscles very often follow an attack of influenza, the same as they appear also in the thighs and calves, no doubt due to myalgia or myositis. Fever with leucocytosis and pains in the eye muscles are typical companions.

The commonest source of pains in the neck we find in diseases of the lymphatic glands and vessels, in fact, in all acute affections of the face, the oral and faucial cavities, the pyriform sinus, the upper esophagus and the cervical spine, no matter whether they are of infectious, traumatic, toxic or thermic origin. These glandular enlargements are present in all forms of cynanche, as an indication of scarlatina but a contraindication of diphtheria. The plague infects the skin of the face, the oral and pharyngeal cavities, the mucous membrane of the nose, covers the in-

ferior maxilla and the whole of the neck with buboes which fuse into a solid edematous mass very painful to pressure so long as the patient retains consciousness. We find the same glandular swellings as accompanying signs in all forms of periostitis and stomatitis (mercurial) with pains in the neck as the logical result.

In acute leucemia, glandular enlargements in the inferior maxilla and in the neck should be looked for as a common but not regular initial symptom. But more about this in another place.

What seems to me of moment is the fact that these acute glandular swellings play in some cases a quite independent role, because the primary port of entry of the infection has either not yet been discovered or only comes into evidence after the enlargement of the glands is already fully developed. If the former is the case, we should be on the lookout for acute swellings of the whole glandular complex which belongs to the periphery of the inferior maxilla, the neck, behind the sternocleidomastoid and in front of the cucullaris muscle, all the way from the occiput to the clavicle. This totality of symptoms combined with high temperature and a general feeling of illness is recognized by some authors as a disease per se and is yclept *"glandular fever."* I do not share this opinion, but incline rather to the belief that we are dealing with an infection that emanates from the phar-

yngeal or tonsillar region and escapes our attention for the want of closer inspection. While these conditions prevail principally in children, they may be also observed in adults.

There is a number of tonsillar affections which radiate into the adjacent lymphatic system of the neck. They come under our notice only when we suddenly discover sensitive dilatations in the cervical glands, while the primary *tonsilitis* remains in hiding until we question the patient about difficulty in swallowing. In fact, there are cases in which the causative factor has simply passed through the lymphatic apparatus without leaving a trace of infection in it. If we bear this well in mind we may often enough find the key to the origin of many a septic affection, even of an etiologically enigmatical endocarditis, of acute nephritis or apparently acute articular rheumatism—without reaction to salicylic drugs—with or without purpura or erythema nodosum.

Analogous conditions are prevalent in acute appendicitis. I have seen cases in which the typical symptoms of appendicitis were accompanied by slightly sensitive enlargements of the cervical glands with a reddish hue in the tonsils, and dysphagia. In such instances not only the lateral cervical glands are involved, but also those which are situated between the margin of the sternocleidomastoid and the hyoid bone.

On the other hand, it is also possible for the primary disease to become demonstrable only two to three days after the swelling in the appurtenant glands has already manifested itself. We hear the patient complain of pain in the neck when he turns his head to one side, thus inviting the diagnosis of rheumatism in the local muscles. A careful scrutiny leads to the discovery that the seat of the pain is really in the muscular processes and a true diagnosis of tonsillitis or lymphadenitis is the result.

Of similar importance is that acute glandular swellings, no matter whether they be spontaneously painful or only sensitive on pressure, under the inferior maxilla or in the neck, ever remind us of the possible existence of erysipelas, especially in the rhinitic zone. I have seen patients in whom such an apparently independent glandular swelling existed for twenty-four hours, with fluctuating temperature, light headache and scarcely noticeable disturbance in the general conditions. But on the following day the outer surface of the ala nasi betrayed the existence of erysipelas. Rhinoscopy is the proper adjuvant in such cases.

If we are confronted by *chronic enlargements* of the *lymphatic glands* of the neck and the inferior maxilla we must make the same careful examination as described above. If this leaves us still in doubt, we must inspect the oral

cavities and the scalp (eczema, pediculosis) for the causative factor. These glandular swellings are sometimes the first and most useful symptom of a tumor at the base of the skull.

On the other hand, they may form a part of the manifestations of a multiple or universal glandular infection, such as occurs in syphilis, scrofulous tuberculosis, strumous buboes, leucemia, aleucemia and in lymphogranulomatous and lymphosarcomatous conditions. Strictly speaking, these affections do not belong here, for as a rule they are not of a painful nature. But I will add to the foregoing that the enlargement of the cervical and inferior maxillary glands may be a pathological condition per se, and thus present a primary and independent disease. If that is the case the diagnosis will waver between lymphosarcoma or lymphogranuloma and tuberculous lymphoma. In any case, the patient complains of an unpleasant feeling of tension, slight pain in the glands, and—in tuberculous lymphoma—of tenderness to touch. However, more about this in the passage dealing with "Glandular Swellings."

Phlegmons of the *cervical cellular tissue* are by their very nature of a painful character. They are by far the commonest results of infectious inflammatory processes in the cervical glands, for which reason a painstaking scrutiny must be made of the oral, pharyngeal and nasal

(accessory) cavities, of the ear, the upper region of the larynx and esophagus and the thyreoid glands. The diagnosis should be obvious, even if, owing to adenitis or periadenitis, we find a diffuse swelling in the affected cervical region, together with reddening or a livid appearance of the skin, or perhaps fluctuation. Nevertheless, the manifestations are not always so plain. In fact, the most severe cases of diffuse extension of the phlegmon do not show them at all. Here the existing pain must guide the diagnostician. His practised eye may be able to detect a slight swelling and a scarcely perceptible change in the skin. But it is the manifest general symptoms, such as local fever with almost normal body temperature, that conduct us to the discernment of the phlegmonous conditions which are chiefly due to streptococcic influences.

Angina Ludovici is a subordinate form of cervical phlegmon, and owing to its proximity to the larynx (edema of the glottis) is of a dangerous character. It concerns the surgeon.

There is another form of cervical phlegmon which also belongs in the domain of surgery, but nevertheless is of interest to the internist. I mean the *actinomycotic phlegmon.* It is easily recognized if it is due to a carious tooth or some morbid affection of the jaws. But it may also originate in the adjacent cellular tissue of

the neck. In both cases it is characterized by a subacute or chronic course, trifling painfulness and sensitiveness in the phlegmonous infiltration, which is rigid and of irregular shape. Bluish-red abscesses with a characteristic serous or sero-purulent secretion and fistulous perforations are formed. In acute attacks, which are not so uncommon, the formation of granulating tumors and pus proceeds rapidly. This is significant for the internist, as it indicates from which direction the disease is descending into the mediastinum or ascending into the cerebral region.

Just the opposite direction is taken by the *diplococcus infection* in *pneumonia,* i.e., the process travels from the thoracic focus upwards into the lymphatic vessels and cellular tissues of the neck until it reaches the meninges. In croupous pneumonia the diplococcus develops a purulent meningitis. Few patients complain of spontaneous pain in the neck, although some tenderness in the intersternocleidomastoid region is always observable.

It goes without saying that every form of *acute ascending mediastinitis* is ripe with pains in the neck. The inflammatory, suppurative process generally deploys from the left supraclavicular fossa, although the incissura sterni jugularis sometimes forms another gate. The diagnosis is plainly staked out by the following

tokens: the pains are felt behind the sternum, whence they radiate into the back between the shoulder blades and into the nape of the neck; there is dysphagia, fever and generalized sepsis; we find a primary affection in the adjacent bones (ribs, vertebræ) or in the neighboring organs, such as the esophagus, trachea, lungs, pleura, or in the abdomen (inflammatory subdiaphragmatic growths), or an edema in the skin of the neck or above the sternum, and gas formation in subcutaneous emphysema.

An acute *inflammation* of the *submaxillary glands* is likewise a frequent cause of pains in the neck which may reach a very high degree of intensity when pus forms in the affected glands occasioned by leucemia or leucemic stomatitis.

There is hardly any appreciable pain in *epidemic parotitis,* though the swollen submaxillary glands are sensitive to touch. When the enlargement of the parotid—as is usually the case—antedates that of the submaxillary glands the diagnosis is self-evident. In this connection it is worth while to remember that the infection at times reaches the submaxillary ahead of the auricular salivary glands, or may not even implicate the latter at all. When this happens at the beginning of an epidemic before typical cases of *mumps* have developed the diagnosis may be subject to errors, especially so if only

one of the thyreoids is as yet involved. The presence of fever or a general indisposition are not much of help. But the diagnosis can be made positive when the ovaries or testicles evince tenderness on pressure. A blood test for eosinophiles is also advised.

Inflammation of the thyreoid glands, purulent as well as non-purulent (*thyreoiditis* or *strumitis*), is heralded by pains in the neck and when swallowing. Casually chills and fever are initial symptoms. But the determinating signs are swelling and painfulness in the thyreoid, or in a part thereof, the fact that the pains radiate towards the head and the ascending swelling causes dysphagia. The inflammation soon establishes itself as a genuine disease, supposedly through an infection of the trachea or fauces—the lymphatic and blood vessels acting as carriers—or it may be (when afebrile) the drug reaction of iodide. It may also be the expression of a generalized infection which has centered in the thyreoids, the causative factors being the bacillus coli or other pus-producing bacteria.

On the other hand, an inflamed thyreoid is often merely a partial symptom of some fully developed infectious disease; in other words, a reflex of typhoid, pneumonia, erysipelas, sepsis, pyemia, influenza, dysentery, malaria, cholera, tuberculosis or syphilis. In a series of cases

the inflammation and casual suppuration established themselves in some section of the hitherto normal tissue of the thyreoid gland in the nature of an acute thyreoiditis. But when under this condition the gland has already undergone goitrous changes, the pains are due to acute strumitis. In rare cases a local hematoma of long standing may be the exciting element, and lead to a false diagnosis of carcinoma.

To the internist the secondary forms of thyreoiditis and strumitis are of great interest. As they are mainly after-effects of some acute disease, for instance typhoid, a sharp eye must be kept on the thyreoid glands which are the favorite place for the colonization of the typhoid bacillus.

Owing to the great similarity of the initial symptoms in both diseases, these thyreoid affections are often mistaken for angina. Only a very thorough examination of the patient can prevent such an error. I mention here also that *Basedow's* disease may be the upshot of acute thyreoiditis or strumitis.

Whenever we find in a patient an enlarged, goiterlike and painful thyreoid gland, we must look for a neoplasm, mainly a carcinoma. Rapid extension and hardening of the growth, emaciation and the anemic look of the patient point to sarcoma in younger people, but to carcinoma in older folks. The diagnosis will be confirmed by

the rapid spread of the tumor, the fixation of the thyreoid gland, when it cannot be moved about by the examining hand, when the lateral cervical nerve and vessels appear compressed, and when dyspnea and dysphagia are present.

Nevertheless, it is sometimes difficult to differentiate between strumitis and hemorrhage in the struma or neoplasm, because high temperature, leucocytosis, pains, swelling, dyspnea and dysphagia are all common symptoms. Yet it seems to me that in strumitis the fever is higher and the secondary character of the disease as well as the local and general inflammatory changes are demonstrable. For the diagnosis of hemorrhage is of importance that the tumor spreads with phenomenal rapidity for several hours, comes to a standstill, and then gradually recedes.

The Bloodvessels and Nerves as Irritating Factors

Under this heading I mention first of all the arteries. We can hardly speak here of real pain. It is rather an aching tension or pressure, an uncomfortable feeling which is produced by any sclerotic or arteritic change in the coating of the arteries, especially when the cardiac action is accelerated. Attacks of genuine, sometimes very intensive pain along the line of the carotids are rather attributable to some primary

disease such as *angina pectoris.* The patient generally complains of an irradiation of retrosternal pains along the left side of the neck into the nuchal or inferior maxillary or dental zone. These pains in the neck and teeth are at times almost unbearable. A demonstrable arteriosclerosis, periodic anginose attack, hyperalgesia during the intervals, and tenderness in the carotids on palpation should furnish sufficient proof of the anginose nature of this painful occurrence.

Spontaneous pains due to an affection of the veins or nerves in the neck, I have never been able to observe. But sensitiveness on pressure in the vena jugularis interna or in the vagus is not uncommon, and I consider this symptom of high diagnostic value, especially in *phlebitis of the internal jugular vein.* Such a phlebitis may be the sequel of an otitis media or else of an *abscess in a dependent part* descending from the carotid triangle.

Moreover, tenderness in the lateral upper region of the neck between the ramus of the inferior maxillary bone and the mastoid process is a serviceable early symptom of *meningitis* or any other pressure on the brain.

In a similar fashion, a *thrombosis* of the *jugular vein,* generally as a continuation of a thrombosis of the superior vena cava, rarely as a primary disease, may give rise to a slight feeling of pain, or rather to an aching, oppressive

sensation in the region behind the sternocleidomastoid muscle. The diagnosis can be made from the swollen appearance of the face and its bluish tint, or from a local edema in the region of the parotis, of the skull or of the right arm as well as on the neck or in the mucous membrane of the oral cavities and the ectatic condition of the cutaneous veins.

Furthermore, tenderness at the inner margin of the sternocleidomastoid must direct our attention to some possible disorder of the vagus nerve. When young people complain of dyspepsia and gastric troubles and in consequence have a haggard appearance, and I find tenderness at this spot, I am always prepared for the beginning of a tuberculous affection in the apex of the lung.

With gastric carcinoma the patient generally suffers from want of appetite and nausea if food is placed before him; but there are cases in which the very opposite happens, i.e., intense, constant craving for something to eat. In both these cases the vagus itself need not be at all involved, but remain absolutely intact.

But not only affections of the vagus nerve, but also a neuritis or neuralgia of the phrenic nerve (a concomitant in diseases of the heart, of the pericardium, of the aorta, of the diaphragm or of the subdiaphragmatic organs), leads to extreme local tenderness and even to

spontaneous pains in the side of the neck, either between the posterior border of the sternocleidomastoid and the anterior margin of the scalinus anticus muscle or between the two processes of the first named muscle. The diagnosis of the phrenic affection is notably based on the aforementioned painful points lateral to the sternum and spinal column, and also upon the abdominal pressure point (bouton diaphragmatique) at the crossing of the continued sternal line and the connective line of the two 10. osseous ribs.

This involvement of the phrenic nerves the fibres of which do not proceed only from the 4., but also in part from the 3. cervical nerve, explains the presence of pains in the neck or in the cervico-nuchal region which we have occasion to observe in affections of the diaphragmatic peritoneum and in liver complaints. The shoulder pains seem to be, however, the preponderating element in these conditions.

In *chronic lymphadenitis of the neck* the pains along the inner border of the sternocleidomastoid and also the local tenderness are of a milder form. It generally originates as an upshot of chronic tonsillitis from behind the anterior process of the sternocleidomastoid and in the retromandibular region (pain in the ear) above the membranous hypothyreoid at the entrance of the laryngeus superior nerve and at the port of entry of the facial into the internal

jugular vein and the paratracheal glands. This disease is frequently mistaken for chronic articular rheumatism.

When pains in the neck are combined with trouble in swallowing, we are led in the direction of an irregularity in the *esophagus.*

An acute inflammation of the esophageal mucous membrane (*esophagitis*) due to some mechanical, thermic or chemical injury or as the concomitant of an acute infection, will infest the cervical portion of the esophagus with pain, which radiates at times downwards behind the sternum.

Deglutition increases the pain, especially in cases of acid poisoning, movements of the cervical spine have the same effect, food gushes back, there is a copious flow of phlegm and sputum, and tenderness at the side of the throat. With these symptoms, with a careful examination of the oral cavities and the aid of a proper anamnesis a mistake in the diagnosis is impossible.

In chronic esophagitis these symptoms are generally missing, but not so in pharyngo-esophageal diverticulum. But even in the latter pain as a symptom is of lesser value, if we except that connected with deglutition and stenosis. Nevertheless, there are cases of diverticulum in which the patient suffers from very intensive pains, no doubt caused by some inflammatory

condition in the wall of the diverticulum itself. The diagnosis should offer no difficulties as all the symptoms are of a strictly characteristic form. I mention: marked stenotic conditions, tumor formation with a peculiar creaking sound above it in the throat, the fetid breath and flow of saliva. The X-ray should do the rest.

Globus Hystericus

It consists of an unpleasant, at times very painful, choking sensation, partly due to hyperesthesia of the mucous membrane of the esophagus, and partly caused by a spasmodic contraction of the pharyngeal and esophageal muscles, commonly called "lump in the throat." The patient has the feeling of a lump rising from the esophageal orifice of the stomach to the throat, where it stops. If other hysterical symptoms are present, the diagnosis is plain enough, but when there are no signs of hysteria we meet with difficulties, for there are other diseases which produce the same effect.

We all know what terrible spasms grip the throats of patients affected with *hydrophobia,* or *tetanus,* so there is no need for me to dwell on this subject; but I deem if necessary to say something about the differentiation between lyssa and lissophobia. A thorough anamnesis and the incubation period (6 weeks to 3 months) coupled with the typical symptoms should leave

no doubts in our mind so far as the diagnosis of lyssa is concerned. If the patient does not show spasmodic conditions in the throat or in the respiratory passages when we blow hard upon the skin—a most valuable, in fact a veritable pathognomic sign for rabies—if there is no excess of the reflex action, if he drinks water freely and without untoward result, we may ease his mind and our own and decide in favor of lyssophobia, especially if the bite of a dog or other mad animal is denied. By the way, lyssophobia is catching, and may be transferred from one person to another by mere suggestion.

It is different in tetanus. Here the spasmodic contractions are the first sign of the disease, preceded, perhaps, by lockjaw (trismus).

Spasmodic contractions in the throat similar to globus hystericus may constitute an important accompanying symptom in other diseases; for instance, in sclerosis of the aorta or of the coronary arteries. *Anginoid* and *anginose conditions* also manifest themselves in this form. But they are of minor importance, because there we have the evidence of the originally retrosternal localization of pressure and pain, their irradiation, the fact that they are provoked by bodily movements and the accompanying uneasiness. Nevertheless, there is a suprasternal form of angina pectoris in which the patient complains exclusively and only of this painful,

constricting sensation. Still an erroneous diagnosis of globus hystericus may slip in if the patient is a female who claims that the spasms in the throat follow some psychic emotion. We should then endeavor to ascertain whether they do not also follow in the wake of some physical exertion, whether they do not also come in the night time, whether they are not felt in the shape of an oppressive feeling in the chest or in the epigastrium. If these questions are answered in the affirmative, all doubts should dwindle away.

Insufficiency and debility of the heart (especially *Basedow's* disease) give rise to similar episodes. They crop up chiefly after an unusually heavy meal, especially when the patient has made a long speech or smoked to excess. The ordinary symptoms of cardiac insufficiency should tell us the truth.

These pains are particularly disturbing in attacks of *essential* or *symptomatic paroxysmal tachycardia.* The diagnostic conditions are discussed on another page.

If in a mediastinal disease the *pectoral vagus* is involved, these pains are turned into real deglutition spasms, reaching sometimes into the deeper registers of the esophagus. The experienced observer will find the key to the solution in the recognition of the primary affection, and in the accompanying manifestations of dysphagia,

bradycardia, arhythmia, occasional anginose attacks, vagus asthma, pain in the nervous vagus of the neck, and intermittent meteorism.

Spasmodic contractions in the throat caused by *flatulency* or preceding the act of *vomiting* are always pure and simple reactions of an irritated nervus vagus.

In *asthmatic attacks* and in hay fever these selfsame spastic pains are molesting elements.

But they are also at times partial symptoms of pharyngeal crises in *tabes,* and may be observed in botulism, in *atropine poisoning* and in *uremia.*

Pains in the Nape of the Neck

In order to present a clear survey of this subject, I am taking each layer of tissue that constitutes the nape by itself as a possible seat of pain.

I begin with the muscular system, for the reason that local pains to some extent originate in the nuchal skin through *furuncles* and *anthrax,* that they are produced by the movements of the head in *scleroderma* on account of the characteristic stiffness and hardening of the integument which is also the case in *myxedema.* All those *muscular affections* which are mentioned in other sections of this bok as sources of pain in the sacrum, in the shoulders, in the back and in the muscular system are mutatis mutandis discussed here also.

When the nuchal pains are only partial manifestations of other synchronous muscular pains, the diagnosis cannot be imperilled. But they may be just as well the primary signal of some generalized muscular disease. Such is, for instance, the case in *tetanus, trichinosis;* and because in *Weil's disease* the muscles of the nape are so often primarily attacked with the most

intensive pains the existence of an epidemic "stiff-neck" has been variously mooted.

In *Wolhynian* and *Pappataci fever* severe nuchal pains have been observed. Painful tenderness in the cucullaris points to *chronic mitigated sepsis.* Pains in the nape are also occasioned by *chronic traumatic myositis.*

Diseases of the occipital fossa (tumor in the cerebellum) or of the meninges may affect the *sensible posterior nerve roots* and produce stiffness in the nape connected with pain which impairs the motility of the head.

The effect may also result from ever so many morbid conditions in the spinal column, especially in the region of intervertebral foramina (arthritis, rhizomyelia, tumors, infiltrations, i.e., leucemic, interspinal canals, aneurysms, etc.), or in the spinal contents; furthermore in all possible expanding extramedullary diseases (tumors, cervical hypertrophic or luetic or tuberculous pachymeningitis), likewise in intermedullary affections (tabes, syringomyelia, multiple sclerosis, myelitis, tumors). The pains are often associated with local stiffness.

Of course, pain and stiffness in the nape are very much in evidence in all forms of *meningitis* and *pseudomeningitis.* The differentiation between these two diseases depends upon the results of lumbar puncture. The same may be said of abortive forms of epidemic meningitis.

Although the presence of pain in the nape, in the head, the extremities, the throat coupled with coughing and occasional vomiting arouse suspicions which gain in strength by the addition of stiffness in the nucha, and the evidence of *Kernig's* sign, yet it is always lumbar puncture that makes the diagnosis positive, especially when also meningococci are found in the nasal and pharyngeal secretions.

In acute *poliomyelitis* intensive pains in the nape are oftentimes undesirable guests, whilst in superior *myelitis* without meningitic complications they may be looked upon as an associated symptom.

Of the various spinal diseases in which these pains constitute a predominant sign, I will mention *Pott's* disease, *malum Rustii* and tuberculous inflammations of the nuchal vertebræ. Distinguishing symptoms are: when the patient is lying on his side and wishes to raise the head he is likely to support it with both hands; there is local deformity in the spine; we find local tenderness upon pressure or palpation, painful reaction to the touch of a hot sponge or an electrode, or to downward pressure on the head. All the other affections which are discussed in the section dealing with the spinal column belong to this category.

It may be of interest to point out that the first four vertebræ are the privileged quarters

for syphilitic affections of the spine. The Roentgenogram, the anamnesis and serological tests should make the differentiation from tuberculous or rheumatic conditions clear.

So far as the malum *Rustii* is concerned, I will add that it may spring not only from a tuberculous state in the two superior cervical vertebræ, but also from a neoplasm, a gumma or a fracture in that locality, not to forget arthritis, rheumatism, gout or syphilitic gummata.

Acute *articular rheumatism* and *gonorrhoic affections* of the *nuchal vertebræ* are members of this cotery. . .

Cervico-occipital neuralgia is the next item before us. It is typified by the paroxysmal character of the pains which radiate from the nape into the occipital region, and also by tenderness between the cervical vertebræ and the mastoid process. Nevertheless, we should not be too rash in making a positive diagnosis from these symptoms, because similar pains may be caused by some preceding primary disease, e.g., by a tumor or by an ankylopoietic spondylarthritis. The differentiation will be found in the fact that the movements of the head are free from pain during the intervals between the attacks, while in anatomical lesions of the posterior nerve roots there is continuous motoric impairment together with other signs pointing

to an affection located at the place of origin of the nerves.

The *lymphatic glands* of the nuchal region are another field in which local pains may arise from some morbid condition, e.g., from lymphadenitis. The diagnosis will materialize from the finding of glandular swellings and their origin (primary infection in the head or trachea), or, perhaps, from enlargements of the glands in general, not uncommon, for instance, in cases of the *plague,* although in this last-named disease the pains in the nape and also in the shoulders are more likely of muscular genesis.

In *occipital migraine,* pains in the nape and headaches are steady companions.

Primary affections of the posterior oral cavities show at times painful reflexes in the nape. In some cases of *acute angina* and *tonsilitis* the patient complains of pains in the corresponding nuchal zone. This happens particularly when the tonsils are affected by an existing *chloroma.* The greenish appearance of the tonsils and the leucemic, sub- or a-leucemic condition of the blood should prevent an error in the diagnosis. It is always advisable to examine the oral cavities thoroughly whenever complaints of pain in the neck are made by a patient.

Any disease of the *pleural* or *pulmonary apices,* especially in the posterior section thereof, in fact all acute or chronic inflammatory

processes or neoplasms in that region, are connected with pains in the nape of the neck, and may constitute a symptom of impending phthisis. In *apical* pneumonia these pains are of great diagnostic worth, as also in *syphilitic aortitis.*

In *diaphragmatic pleuritis* pains in the nape are, no doubt, reflex actions emanating from the 3rd-4th cervical segment.

That an affection of the *subdiaphragmatic peritoneum* or of the *serous coating of the liver* often causes pain in the nape, in the neck, and also in the shoulders, is not hard to understand, because the fibres of the phrenic nerve originate not only from the 4th, but also from the 3rd cervical nerves.

In the majority of cases, however, the pains caused by these diseases, i.e., subdiaphragmatic pleuritis and peritonitis (perihepatitis, perisplenitis), are not spontaneous in their nature, but are rather evinced in a certain part of the muscle when we gently press the trapezius between our fingers or tap the supraspinate fossa. The middle portion of the trapezius seems to be most susceptible to this reflex action. This tenderness disappears with the decline of the serositis, but returns with a fresh attack. That explains also why the pain in the nuchal muscles is an appreciable symptom of apical pulmonary tuberculosis, and is otherwise a useful guide in the diagnosis of subdiaphragmatic morbid conditions.

Pain in the Chest

Pain in the chest may be caused either by morbid conditions of the thoracic frame or by diseases of the internal organs of the pectoral cavity, if not by complications involving both regions.

Insofar as the internal organs are concerned, the pains due to diseases of the heart are discussed in a separate chapter.

The pains originating in the wall of the thorax are the result of affections of the skin, the muscles, the intercostal nerves, the fascia, the subpleural connective tissue, the ribs and the mammæ.

Among the *cutaneous* diseases there are not many that are of direct interest to the internist, excepting, of course, those which occur in the zones of *Head,* and these will be discussed in the sections dealing with the corresponding primary organic troubles.

But medical writers report cases in which these zones become so painful that the patient instinctively avoids every contact with the affected portion of the skin.

Cholelithiasis and hyperesthesia of the right upper abdominal and lower thoracic integument

will cause the patient to keep his right arm away from the affected part for fear of severe pains elicited by contact with the skin.

If these pains are merely symptomatic of an isolated skin disease, they belong in the province of the dermatologist. But if they are a partial manifestation of a general disease, e.g., of adipositas dolorosa (*Dercum's* disease), of "symmetrical" lipoma, or of neurofibromatosis, the internist must take notice. I refer to this subject more fully in the chapter on "Pains in the Extremities." And again, a skin disease may be the superficial sign of a deep-seated morbid condition. In this sense I include here only those cutaneous and subcutaneous inflammatory changes which are the reflex actions of internal organs of the thorax. The internist will keep here a sharp lookout for subcutaneous abscesses which originate from an empyema necessitatis, or, if situated in the precordial region, are connected with the mediastinum, but not with the pleura, or also may be a derivation from some primary disease of the lungs. Peripleuritic abscesses and pulmonary hernias belong here, too. A carcinoma in the pleura is just as likely to find its way to the surface of the thoracic integument, thus forming an unmistakable symptom of the primary affection.

Empyema necessitatis, cysts with or without involvement of the mediastinum, perforation of

a bronchiectatic pocket, peripheritic abscess, all are subject to inflammatory conditions. This makes it so easy to separate them from abscesses due to perforation of a tuberculous sac (cold abscess) or from a pulmonary hernia in which there is febrile reaction of the skin.

The differentiation between the first three aforementioned diseases should not offer any difficulties. It is found in the condition of the lungs or pleura (empyema or bronchiectasis). Doubt can arise only when an abscess is formed by some anaërobic bacteria which reach the mediastinum. Where such a communication does exist the diagnosis should not be difficult. Where it does not exist, however, we run the danger of being misled by the fluctuation and crepitation over the tumor with additional gurgling râles due to oscillations of intrathoraic pressure. That the lung itself is apparently sound does not prove anything to the contrary. But in my opinion absence of the tympanitic percussion sound which one might expect if the lungs were involved, and also absence of typical respiratory inflation of the tumor by coughing are definite contraindications. No doubt the Roentgen ray will on some not so very distant day come to our assistance in this matter.

If no aërobic abscess is in evidence the differential diagnosis between *peripleuritis* and a per-

forating pleural abscess will be rather puzzling. It is easy enough to spot a perforation of the thoracic wall by a pulmonary process when we have definite proof of a primary lung disease and unmistakable pulmonary symptoms before us. But to use a strict localization of pains and swellings for diagnosing a purulent peripleuritis I do not consider practicable, because we find these same conditions also in perforating empyema, and pain is always associated with a non-perforating pocket empyema. To my mind the points of importance are: the absence of all pulmonary symptoms, especially coughing—early severe dyspnea occurs in both diseases; it is due to the painful breathing—and there is no dislocation of the mediastinal organs in spite of obstruction and dilatation. In empyema necessitatis I have never been able to see more than one perforation point. The X-ray should be very helpful in the diagnosis.

Local acute inflammation is not noticeable in the *perforation* of a *tuberculous sac* into the softer parts of the thorax, neither is there pain. The skin does not seem to react to pain, although it is bulgy and livid in color, crepitation, tympanitic sounds and râles are perceptible and the tumor has a squashy, doughy consistence. The condition may easily be mistaken for *pulmonary hernia.* Still the diagnosis should yield positive results from the observations of pocket symp-

toms, the presence of râles, the squashy consistence of the tumor—in pulmonary hernia the tumor makes a creaky sound when squeezed with the fingers—the severe involvement of the lungs and the favorite spot for the perforation in the 1.-3. anterior intercostal space in pulmonary tuberculosis, while pulmonary hernia prefers the upper clavicular region or 6. to 9. anterior intercostal space.

This applies with equal force to those cases of pulmonary hernia which are not of traumatic orgin, but arrive spontaneously owing to extraordinary intrathoracic pressure on an otherwise abnormally fragile tissue.

The recognition of *actinomycosis* arising from the air passages and perforating the thoracic tissue should offer no dilemma, because this disease is chiefly a chronic progressive infiltration making its way to the surface from the deeper tissues; it is the formation of multiple subpleural phlegmonous foci from which the pus works up until it perforates the skin in the shape of manifold fistulae.

A *subcutaneous hematoma* generally sets in with sudden severe pains in the chest and dyspnea. When spontaneous in origin it is of particular concern to the internist. As a rule the affected part is tinged with a bluish red color by which it can readily be recognized.

Aneurysms of the *pectoral aorta* generally

cause only moderate pain as they gradually expand under the skin. The abnormal bulging of the skin and very strong pulsation together with the Roentgenogram can leave no room for an erroneous diagnosis.

Female patients often have occasion to complain about *pain in the Mamma.*

Aside from mastitis and carcinoma, the only organic disease that claims the attention of the internist is *hypertrophy* which causes a dragging pain in the affected mamma. Similar painful feelings are caused by any infiltration of the mammary glands in pregnancy or during the lactation period, a sort of muscular fatigue. They may assume a permanent form in leucemic or aleucemic adiposity. A proper support for the mammary glands relieves the pain or removes it.

Mammary carcinoma, by the way, at times causes periodic attacks of, if not continuous lancinating pains which radiate into the arm, especially when the seat of the tumor is in the exterior upper quadrant of the mammary gland. A hard, slightly sensitive, almost immovable and irregularly formed node and the indrawn nipple which exudes a sticky, serous moisture are most reliable symptoms for a correct diagnosis. Such a carcinoma may also occur in the adult male, when it manifests itself in the lymphatics in the form of nodules the size of a pinhead or pea

which produce a peculiarly painful sensation in the superficial nerves of the chest.

Presenile or *senile involvement* of the mammary glands, it is said, also is connected with local pain. Of course, there are other appurtinant diseases but they belong in the domain of surgery. What, however, is of concern to the internist is pain in the mammary glands occasioned by neurosis, neurasthenia and predominently by *hysteria.* In these conditions the mamma, sometimes the nipple only, constitutes a very strongly marked hyperesthetic zone. The slightest friction of the clothing, even of the flimsiest underwear evokes most intensive pains. The presence of typical stigmata and particularly that of psychic symptoms should guarantee a positive diagnosis.

In *hysteria* and pointedly so in *neurasthenia* the mammary pains may be due to mastodymia, which after all is merely the expression of an intermammary (at times even very stubborn) intercostal neuralgia. The pains are of a shooting, burning, boring, tearing character and come in periodic attacks; we find pronounced hyperesthesia of the skin, pressure points (external, axillary, vertebral), an additional anatomical lesion of the glands that by itself is a possible originator of a deuteropathic mastodynia, all of which combined form a proper basis for the diagnosis.

There are cases of what I may call "imitation hysteria" in which the patient complains of all the typical symptoms of such a mastodynia. The condition is brought about by self-delusion —auto-hypnotism—after the patient has visited a friend who is suffering from the actual disease and now imagines she has it, too.

Pains in the breasts are not uncommon during the *menstrual period,* or in the early stages of *pregnancy,* also during the *climacterium,* with actual swelling of the mammary glands. These are no doubt a reflex action of the genital glands on the mammary glandular system.

Burning pains in the mamma which radiate into the adjacent arm have also been observed in cases of *sclerosis* of the *mammary arteries.* I cannot speak here from personal experience.

So far as *intercostal neuralgia* is concerned, I wish to point out that it is always risky to judge from a bilateral condition of the existence of a deuteropathic affection caused by a lesion of the nerve roots either in the spine itself or its joints or in the contents of the spinal canal. Even in a case of stubborn unilateral intercostal neuralgia there is always the possibility of an existing tumor in the extramedullary spinal cord or in one of the vertebrae. Chronic meningitis, a serious mechanical injury of a nerve trunk caused by a diseased rib (gummata) or by an affection of the pleura (neoplasmata) or of the

mediastinal organs, e.g., anurysm of the descending thoracic aorta or a bronchus carcinoma, all are included in this list of eventualities. Important for the diagnosis is the fact that in just such cases the characteristic pressure points are frequently missing. But we should remember that every form of neuralgia may originate from a diseased condition of the central nervous system as well as from general causes such as diabetes, gout, malaria, chronic constipation, anemia or chlorosis.

In cholelithiasis pain in the right eleventh rib often resembles an attack of intercostal neuralgia.

In some cases of true *angina pectoris* the typical retrosternal pains are accompanied by pains in the superior intercostal spaces. When these are combined with deeper intercostal neuralgias we may well take them as the signal for an aneurysm of the pectoral aorta.

Pain in the intercostal nerves is a constant companion of local *herpes zoster* during the entire course of the disease, the symptoms of which should be easily recognized. The pain extends, belt like, over the whole affected intercostal space and the eruption consists of typically grouped vesicles and subsequent scars. We must not forget that herpes zoster, especially in its bilateral form, is often a partial manifestation of a secondary neuritis of the nerve roots, or of

some mediastinal morbid condition, e.g., of an aneurysm of the aorta. It may also be provoked by arsenic or carbon dioxid poisoning.

If herpes zoster is the result of a morbid process in the intervertebral ganglia it may give rise to severe, continuous or only spasmodic pains in the chest, especially in the posterior or lateral portion parallel with the seat of the lesion. But these pains are likely to prevail even when the attack is not accompanied by herpes zoster. This refers to all expanding growths in the spinal canal or in the vertebral bones.

Such an irritation of the spinal roots is no doubt the causating factor of those intense intercostal neuralgias which in company of headache, neuralgic pains in other parts of the body, and articular pains constitute the initial symptoms of *multiple sclerosis.*

In *tabes dorsalis* lightning pains in the intercostal nerves are sometimes experienced with preference in the lower hypochondriac intercostal spaces.

The *tabetic girdle sense* is associated with unpleasant, slightly painful sensations in the chest. When this symptom is the first and perhaps the only complaint made by the patient the diagnosis may not be so easy, especially so in cases where the pain is confined to one side of the chest only.

A similar *girdle sense* crops up not infre-

quently also in *neurasthenia.* But when this happens it is advisable to make a thorough search for some anatomical affection of the central nervous system. If this proves fruitless and the typical neurasthenic symptoms are demonstrable the diagnosis may be made positive.

Furthermore, girdle-like paresthesias involving pain are likewise possible in many other spinal diseases such as *meningitis* in all its forms, *syphilis* of the *spine,* or a *tumor, syringomyelia, chronic multiple sclerosis,* etc. *Syphilitic neuritis* and also *poliomyelitis* are often ripe with pains including the girdle variety in the back as well as in the chest.

If in poliomyelitis girdle pains set in all of a sudden and with great intensity we should seriously look upon this condition as an initial warning of acute *multiple sclerosis,* or *hematomyelia,* acute *myelitis, embolism* or *thrombosis* of the arteries of the spinal cord. As a rule we shall find a concomitant acute paresis if not paralysis of the two lower extremities, or some ailment of the bladder or rectum. In that case the diagnosis would be patent.

This is also true of diseases of the spinal column proper, e.g., *spondylarthritis ankylopoietica,* and furthermore of diseases of the bones which cause a contraction of the dorsal vertebrae e.g., *osteomalacia.* I mention also *arteriosclerosis* of the vessels in the posterior column of the

spinal cord. The diagnosis in this case must be established on the following symptoms: abnormal fatigue, weakness and stiffness in the lower extremities when walking (in the absence of painful intermittent dysbasia), pulsation in the arteries of the foot, the *Babinski sign* during or after walking but absent when at rest, increase of the tendon reflexes in demonstrable arteriosclerosis of the peripheral or inner vessels, presence of other symptoms pointing to arteriosclerosis such as angina pectoris, gastralgias and enteralgias, etc.

These girdle pains are sometimes a very important manifestation of some *retromediastinal process.* An *aneurysm* for instance of the descending *thoracic aorta* at the point where the latter passes through the diaphragm frequently betrays its presence there by such girdle pains in the diaphragmatic region. They are felt during physical exertions, or, and mostly so, when the patient lies on his back after the intake of food.

Some patients suffering from *syphilitic aortitis* complain of an "uncanny feeling" or girdle sensation and even genuine pain in the region of the asternal ribs. The cause for this seems to me to be presence of an arteritis of the intercostal arteries, I mean a constriction at the point where they branch off from the pectoral aorta. The pains may be felt on both sides or on the

left side alone. Analogous observations have been made in typical attacks of *angina pectoris* and also in *cervical tabes.* In tabes dorsalis some patients complain of a feeling as if the skin over the cardiac region were being painfully pinched.

The girdle pains due to *clonic* or *tonic spasms* of the diaphragm are generally confined to the region of union between the diaphragm and the thoracic wall. The chronic form of these spasms manifests itself by singultus which may endure for several days under very severe girdle as well as epigastric pains. We observe this, in part at any rate, in abdominal diseases which have a reflex action on the phrenic nerve, e.g., in the bladder when obnormally distented by a surcharge of urine, in prostatitis, in uterine affections; likewise in central irritation of the phrenic nerve due to anatomical lesions of the brain or spinal cord (encephalitis, epidermic singultus) or caused by toxic influences (spastic singultus in uremia, sepsis, chronic alcoholism), in neuroses (hysteria), in anemic and cachectic conditions, in anatomical lesions of the trunk of the phrenic nerve due to indurated mediastino-pericarditis, possibly also following an aneurysm of the pectoral aorta accompanied as a rule, by indurated or gummatous mediastinitis. We find this same spastic singultus in direct irritations of the serous coating of the diaphragm, for instance as a

manifestation of a primary or more or less diffuse peritonitis in the lower surface of the diaphragm, and also in rare cases of diaphragmatic pleuritis.

It is more difficult, and perhaps only with the aid of the Roentgen-ray, to recognize the tonic form of diaphragmatic cramps. It occurs as an accompanying sign of tetanus, also of tetany, and in articular and muscular rheumatism due to exposure to cold, and preëminently so in hysteria. The patient is likely to be attacked by sudden, very intensive girdle-pains in the diaphragmatic circumference; dyspnea and cyanosis are very pronounced, the abdomen is distorted and the pectoral organs are pushed backwards. Minor attacks of this nature seem to be not uncommon in hysteria.

In *acute diaphragmatitis* arising from pneumonia, pleuritis, peritonitis, perihepatitis or perisplenitis we are often called upon to witness attacks of intermittent or also long continued pains in the diaphragmatic zone. The patient complains of girdle-like pains in that region, also in the chest and back, seriously interfering with deep breathing. This condition may easily escape observation in a case of pneumonia or cholecystitis; the more so when we are dealing with a case of acute diaphragmatic pleuritis, especially of a tuberculous nature. Under these circumstances cholecystitis or ulcus ventriculi are

the predominant errors in the diagnosis. Proper search for pressure points in the phrenicus and the service of the X-ray are the only correctives.

Difformities in the thorax, chiefly *scoliosis* of the spinal column, are common causes of pains in the chest. A chronic affection of the intercostal nerves is possibly the originating factor of these painful sensations which may be unilateral only, but often enough are felt on both sides.

If they are unilateral in the left inferior intercostal spaces and of a neuralgic character, they point to the existence of an *ulcus ventriculi.* It is always advisable in such cases to look most diligently for gastric symptoms such as periodic pressure in the stomach, pyrosis and indigestion lest we be surprised by a sudden gastric hemorrhage. The gastric contents should be carefully analyzed, a steady watch for gastric or intestinal hemorrhages must be kept, also for hyperalgesia of the epigastric skin, for *Boas'* sign, and for deep pressure sensibility.

The larger superficial as well as the deeper lying muscles of the chest may be the habitat of local pains. By way of preliminary mention I wish to point out that periodic attacks of pain in the pectoralis muscle should always remind us of a possible angina pectoris, and also that *subpectoral suppuration* should always engage the attention of the internist although as a rule

its consideration and treatment is left to the surgeon.

I have repeatedly seen in my clinic cases in which the patient complained of intensive pains in the chest and difficult breathing with chills and fever which were taken by the intern as manifestations of an acute attack in the respiratory tract. The examination gave negative results so far as the pulmonary conditions were concerned, but there were indications of a generalized sepsis, enlargements in the anterior section of the thorax, especially in the region of one major pectoralis muscle, a slight edema of the skin, very intensive local tenderness, and a movement of the arm which contracted the pectoralis major elicited most intensive pains in that muscle. A diagnosis of subpectoral suppuration was made and confirmed by the attending surgeon by way of incision.

The diagnosis may be even a more difficult problem when an empyema and a subpectoral phlegmon run side by side.

That the muscles themselves are the carriers of the localized pain, is demonstrated by local tenderness and aggravated painful sensations which accompany an attempt to activate the appurtenant muscles.

After all, muscular pains in the chest are generally only a reflex action of morbid conditions in other muscles of the body. The charac-

teristic symptoms of the primary disease should facilitate a true diagnosis. But due regard must be had of the fact that a disease which involves so large a district as the muscular complex of the chest must needs carry with it respiratory impairments (suffocation) and diaphragmatic complications. If pulmonary troubles (pneumonia) accede or the examination of the patient is carelessly made the diagnosis is apt to take an erroneous trend.

In acute *polyomyositis* or *trichinosis,* in *Weil's disease* and also in *gout* such a muscular involment is ever a contingency.

Independent muscular pains in the chest are often due to athletic or gymnastic sports, likewise to violent and protracted fits of coughing (pertussis, bronchitis, pulmonary emphysema). They are also observed in occupational pursuits —seamstresses, cobblers, tailors, dentists—and are due to the constant strain on the muscles, bones and nerves of the chest, not to forget *overfatigue.*

Exquisite muscular pains in the chest are seemingly the prerogative of *pleurodynia* and *rheumatism of the pectoral muscles.* If the superficial muscles are the site, the diagnosis should result promptly, for we find in both affections muscular tenderness and motoric pains, in protracted cases also muscular twitching and formation of nodes.

In more severe muscular diseases the patient complains not only of pains when raising the arm but also of such which accompany coughing, sneezing, swallowing and deep breathing. The latter is naturally and instinctively avoided and leads to congestion of the bronchial secretions with complicating catarrhal conditions. I raise the question: Is a secondary catarrh the sequel of a primary rheumatism of the pectoral muscles or is it a primary bronchitis with secondary dry pleuritis?

If in overfatigue the pains persist for a considerable period of time we should remember that pains in the pectoralis muscles are specifically the symptom of a chronic overtax of the muscles due to abnormal growth of the pectoral glands.

When the deeper muscles alone are involved (pleurodynia) the differential diagnosis will be concerned only with *pleuritis sicca* in which sharp pains in the intercostal region are also a prevailing symptom. Intercostal neuralgia is barred owing to the peculiar characteristic of the pain and tenderness along the intercostal space.

In the differential diagnosis between pleurodynia and pleuritis sicca in the primary as well as in the secondary form which latter may arise from a morbid condition in the thoracic wall, the lungs or the bronchi, we must be circumspect and not place too much stress on what the

patient tells us. He is wont to complain of increased painfulness when drawing a deep breath or during coughing or sneezing. He will instinctively put his hand to that part of the thorax in which the pain is felt when the urge for such respiratory excursions comes on and he will take in the air in short draughts to forestall pain. However, all these symptoms may be observed in every form of thoracic neuralgia, in every form of intercostal neuralgia as well as in pleurodynia and pleuritis sicca. He will also tell us that every more or less vigorous movement of the thorax engenders the same kind of pain—also common to all the diseases aforesaid. But when a deliberate, slow movement provokes the pain more so than deep breathing, we may take it as a pointer in favor of intercostal myalgia or neuralgia. If the thoracic movement towards the healthy side of the body strongly exacerbate the pain we are dealing with a tension of the pleura and may incline to a diagnosis of pleuritis. In intercostal neuralgia the same kind of movement towards the affected side contracts the nerves and pain follows, whilst in pleurodynia (myalgia) this is not the case. In some cases the faradic current passing through a moist sponge materially assists the diagnosis of myalgia, because under electric treatment the pain in the pectoral muscles disappears.

The presence of local tenderness carries no weight, excepting the characteristic pressure points of neuralgia. Local tenderness exists in intercostal myalgia as well as in pleuritis sicca, but if it extend over several intercostal spaces it points to pleuritis. Fever exists both in muscular rheumatism and in fibrinous pleuritis, and both may have the same etiological genesis, i.e., either of them may be the signal of acute rheumatism. Thus, there remains only one more distinguishing mark, i.e., friction fremitus in the pleura, and even this must be taken with caution unless it is unmistakably perceptible in its typical form.

We can hear in the affected part a fine crepitant, crackling sound with inspiration as well as expiration. It is of the same constancy no matter whether the patient breathes deeply in the regular fashion or coughs. It's nature is that of an atelectatic crepitus caused by continued superficial breathing. The patient could not draw a deep breath if he wanted to. Erroneously we call it pleural friction fremitus. I had such a case under observation. Crepitus was distinct and constant. The patient was much relieved by the application of a compress. I diagnosed fibrinous pleuritis surmising a pulmonary infarct due to demonstrable arteriosclerosis and myofibrosis, but future events proved clearly that it was a case of intercostal

neuralgia without pressure points and caused by a carcinoma of the spinal column. This shows that even a definite audible crepitus is not a reliable criterion and it is always advisable in such cases to resort promptly and at the earliest possible moment to the electric current for assistance.

It follows that, whenever we are called upon to decide whether a case before us is one of pleuritis sicca or of unilateral intercostal neuralgia with pressure points and local tenderness, we should make a thorough examination of the spinal column and of the ribs and ascertain whether pain reacts more intensely to thoracic movement than to deep breathing, and avoid the palpable error of a diagnosis of pleuritis sicca.

Muscular pains in the chest are likewise a sign of *neurasthenia.* The patient is in fear of a heart disease. If the pain is aggravated by movements of the affected muscle but is mitigated by walking the diagnosis is readily made.

In *tetanus* these pains are often erroneously taken to be of a pleuritic nature, while in reality they are due to morbid contractions of the pectoral muscles.

The pains released in the chest by a beginning *grippous affection* and at times felt in the initial stages of *scurvy* are at least in part due to myalgia. In scurvy they are also caused by hemorrhagic condition at the finals between the cartilage and the osseous section of the ribs.

Pectoral when combined with nuchal pains (also in part at least as constituents of myalgia) may be furthermore signs of a *chronic septic infection.*

These pains in the chest occasionally come from an overexertion of the *abdominal muscles* in severe fits of coughing because their terminals are situated in the bony frame of the thoracic wall.

It is self-evident that a fibrinous, in fact any inflammatory process of the *pleura* necessarily leads to pains in the chest. An acute tuberculous pleuritis, it is true, may set in with very mild local pains, even without any at all, but the purulent form is characterized by the very intensity and constancy of these molesting conditions. Exceptions, of course, are parapneumonic and metapneumonic *empyema.*

The only remaining part of the thoracic wall in which pectoral pains may originate are the *bones themselves,* mainly the ribs and the *sternum,* both of which I shall now proceed to discuss.

May it suffice to just merely mention that these two skeletal parts may be affected by an acute periostitis or osteomyelitis, by a chronic tuberculous (caries), luetic or actinomycotic osteoperiostitis, by primary or secondary neoplasms, because they have already been fully discussed in the chapter in "Diseases of the Bones."

But I will apprise the reader here of the fact that pains in the chest caused by primary or secondary neoplasms of the bones come on at times most abruptly and with such intensity that deep breathing becomes impossible and that the patient apprehensively avoids every kind of movement.

In *vertebral insufficiency* pains in the chest are, in part at least, attributable to the spinal column. For diagnosis and symptoms the reader is referred to the volume dealing with "Abdominal Pain."

Pains in the sternum and in the ribs, the same as those in the spinal column, may be only partial manifestations of a generalized affection of the bones, e.g., osteomalacia, osteoporosis, rachitis, multiple primary or secondary neoplasm or myeloma. (Details will be found in the chapter on "Pains in the Bones.") For differential diagnostic purposes it is important to say that these pains, similarly to those in the muscles and pleura, are aggravated by coughing and sneezing, etc., in fact coughing frequently starts them.

In chronic myeloid leucemia we find exquisite tenderness especially in the thoracic bones which are so intense that the patient has the utmost difficulty in dressing.

There are cases, however, of this last-named disease in which the pains appear rather sub-

dued, or may be missing altogether until provoked by pressure and palpation. The sternum, the ribs and the lower extremities are by preference the seat of these pains. But this is not unusual in other diseases such as severe anemias of primary or secondary origin, chlorosis, *Basedow's* disease, in acute phosphoric poisoning, in septic and pyemic affections, septic endocarditis and acute leucemia. A valuable hint for the diagnostician ever.

Malignant neoplasms, cold abscesses or gummata, also arrosion of the sternum due to aneurysm of the aorta, etc., are diseases localized in the *posterior section of the sternum.* The clinical examination shows no visible or palpable changes, but local dullness of a higher degree may be noticeable. Sternal pain and local tenderness are present, i.e., conditions which strongly resemble those enumerated in the preceding paragraphs and for that reason deserve mention in this place.

A special form of sternal pain has its abode in the *region* of the *ensiform appendix* and is likely to remind us of an affection of the synchondrosis of the ensiform cartilage with the corpus sterni often enough witnessed as a partial manifestation of gonorrhoic articular rheumatism or of a gouty diathesis. A fracture or separation of these two bodies due to some trouble in the bony skeleton, e.g., osteoporosis is

also a possibility. Chondrosis and perichondrosis of the aforesaid cartilage, with local tenderness or even enlargement (perhaps of syphilitic origin) may also be at the bottom of these pains. Furthermore, we must not forget that girdle-pains arising from any irritation or inflammation of the posterior roots of the upper and median dorsal segments may be focused in the region of the ensiform cartilage. The sole complaint the sufferer makes is of pains at the bottom of the sternum, i.e., the ensiform appendix.

Pains behind the xiphoid occur in *aortalgia* and *angina pectoris*. These attacks come on as a rule with bodily exercise and produce a burning, pressing painful sensation behind the ensiform appendix with a tendency to gradually climb upward behind the sternum into the left arm. But if it remains stationary we must be prepared to encounter further symptoms of epigastralgia.

Pains of a like nature supervene also in diseases of the esophagus and cardia. I will speak of this anon.

It is rather remarkable that this investigation of pain is not always a correct indication of the real seat of the disease. The patient who complains of pains thus localized, especially during swallowing, is undoubtedly suffering from carcinoma of the esophagus. But this carcinoma is not located behind the ensiform process, but

higher up on a level with the middle of the chest. The pain, therefore, can only be the reflex action of esophageal spasms. Another patient complains of the same kind of spastic pains, he can pass neither fluid nor solid foods. The causative factor is a tumor in the pyloric region. The painful cardiospasms are the reflex action of the tumor.

Pains behind or close below the xiphoid process are proper to *gastroptosis,* no doubt due to a dragging of the cardia. They come on after eating, or during walking or going upstairs.

Pains around the ensiform appendix are common in diseases of the *diaphragmatic muscular system.* When associated with dragging pains in the masseters, beginning trismus, epigastric pains, pulling pains in the extremities, profuse sweating, and sleeplessness, they are valuable symptoms of *tetanus.* Later on they pair with diaphragmatic shock.

Within a narrow, limited zone around the sternum certain diseases originate which cause very painful local sensations. I refer to the affections of the *sterno-clavicular joint* and that which connects the *sternum with the 7. rib.* The most common among these are acute articular rheumatism and gonorrhoic arthritis. Local swellings, reddening of the skin, the general symptoms and the X-ray will steer the diagnosis into a safe port even in those rare cases in which

the two aforesaid diseases settle either momentarily or even permanently in these joints. I must add, however, *aneurysms* of the *aorta,* of the *anonyma* and the *subclavian arteries,* which are offenders in the same sense.

Pain *behind* the sternum should direct our eye to the *myocardium* and the *aorta.* Acute and chronic aortitis, sclerosis, aneurysm, insufficiency of the aorta, sclerosis of the coronary arteries, acute and chronic pericarditis, acute arteritis of the anonyma, are distinguished by the fact that the spontaneous pains, characteristic in all of them, are provoked by bodily movements, but dormant when the patient is at rest. On the other hand in acute myocarditis, arteritis, aortitis and pericarditis the pains are of a constant nature in the region of the heart, i.e., behind or lateral to the superior sternum, coupled with pronounced local tenderness on pressure and palpation.

In true angina pectoris the pains are likewise localized behind the sternum. (See chapter on "Cardiac Pains.")

I must not neglect to mention *thrombosis* and *thrombophlebitis* of the *vena cava superior* as a possible cause of latero-sternal pains. A similar pain, resembling rather that connected with angina pectoris, may also be provoked by an aneurysm of the anonyma when it expands downwards inwardly. The patient is apt to

complain of pectoral pain in the direction of the right shoulder joint.

Patients suffering from stenosis or obliteration of the superior vena cava complain of constringent pains behind the anterior wall of the chest. The diagnosis should be: cyanosis and edema, and collateral expansion of the superficial thoracic vessels and epigastric veins.

Sensitiveness in the upper sternum and the adjacent intercostal spaces is likely to be induced throughout the intervals between the attacks of angina pectoris. It springs from an aneurysm of the aorta with or without arrosion of the sternum or from any other form of mediastinal disease that gives injury to the posterior sternal portion. I mention morbid conditions of the *retro-sternal* antemediastinal lymphatic *glands*. In *tuberculosis* of these glands painful tenderness chiefly on pressure and palpation exists in a limited zone of the sternum corresponding with the level of the 4.-6. thoracic vertebra. Analogous pains are felt in syphilis of the same glands and are coupled with acute inflammatory or purulent changes. Anthracosis of the *peribronchial glands* with *perforation into the air passages* is not an uncommon incident. Important for the diagnosis is the presence of acute or subacute putrid bronchitis (unilateral) which degenerates into an abscess or gangrene of the lung with expectoration of sputum which shows under

the microscope a free layer of black, crumby pigment; there is dullness over the superior sternum or laterally to it, the X-ray shows a shadowy impression; we find spontaneous painfulness over or laterally to the manubrium sterni with tenderness.

The perforation may also be in the *esophagus*. If it is latent in its character it has, as a rule, been preceded by a traction—diverticulum. If infection supersedes the diagnosis will be materially advanced by the observation of pains behind the sternum and between the shoulder blades, sensitiveness in the spinous processes on palpation, coughing, pain in swallowing behind the manubrium sterni or the xiphoid process, recurrent paresis, pupillary difference and heaving up of evil smelling, caseous, purulent, bloody masses.

This list would be incomplete without the inclusion of benign and madignant tumors of the mediastinum or the appertaining glands, also inflammations thereof (diffuse or localized mediastinal abscesses of an acute, purulent or sanious character, or arising through continuity or metastasis, or of a chronic, indurated or syphilitic nature, likewise actinomycotic mediastinitis), also tumors of the thymus, substernal struma with strumitis, carcinoma of the bronchus, mediastinal pleuritis and pericarditis. The same manifestations of severe, periodically in-

creasing pains behind the upper sternum or within the reach of the intercostal nerves are individual to all these diseases whether they are associated with an affection of the sternum proper or not.

Of course, combinations of these diseases are not impossible and we may well anticipate the presence of severe retrosternal pains when each of them separately is capable of producing them.

Carcinomata of the *lung* or the *bronchus* call forth severe pains which resemble neuralgia very closely and radiate into the arms. They arise, in part at least, from the aforementioned glands, but also from the bronchus, and bear the character of pulmonary infiltrating carcinomata. The clinical picture will be complete for diagnostic purposes when we observe: irritating coughs, want of thoracic participation in the act of breathing, dyspnea, slight involvement of the lungs, general indisposition, absence of respiratory sound or crepitus, sputum tinged with blood, metastasis in the glands, in the supraclavicular fossa and in the skin and sometimes subfebrile temperature. The Roentgen-ray is bound to render good service.

We will turn our attention now to the trachea and the esophagus.

Among the tracheal pains the commonest is that observed in acute as well as in chronic *tracheitis*. There is a feeling of dryness, tick-

ling, burning and soreness behind the sternum. There is cough with copious expectoration, a distinct deficiency of bronchial symptoms, but decided treacheoscopic evidence. A complex of unmistakable symptoms for the diagnostician. Sensitiveness on pressure and palpation is usually absent, but the pains are highly sharpened by paroxysms of coughing which may also be provoked by pressure on the first treacheal ring. The retrosternal pain in *pertussis* may be explained in the same manner, and likewise that experienced in chronic *stenosis of the windpipe.*

It is hardly necessary to mention that *foreign bodies in the treachea* cause local pain. Repeated paroxysms of coughing and attacks of suffocation and gagging indicate their presence as well as the existence of a subsequent purulent or sanious tracheitis, if the anamnesis has not already given us the required information. The arrival of the substance in the windpipe or in one of the bronchi and its expulsion or removal are connected with retrosternal pains, no doubt caused by an injury to the mucous membrane. Similar retrosternal pains are also the companions of other morbid tracheal or bronchial affections, such as syphilis or carcinoma.

A *perforation,* e.g., of an esophageal carcinoma *into the windpipe* also releases retrosternal pains combined with local tenderness owing to mediastinitis produced by the disease.

Bronchial asthma confronts us likewise. The pains come in sudden attacks behind the sternum and are a collateral manifestation of the periodic paroxysms, and are undoubtedly due to spasms in the bronchial and tracheal muscles and to acute hyperemia of the mucous membranes.

In *broncholithiasis* the pains in the chest are very vicious and insufferable. Spasmodic coughing similar to whooping cough, dyspnea, the sensation as if a lump were rising and falling in the windpipe, periodical hemoptysis and the final expulsion of the stone are clear indications.

It is needless to say that the inhalation of *poisonous fumes* irritates the air passages and causes severe local pain. Chlorine, brass, zinc and the terrible gases employed in warfare are striking examples.

Diseases of the esophagus proper are frequent factors in retrosternal pains. Soup, coffee, tea, milk or other beverages that are too hot, or a morsel of food that is too large, etc., may cause severe pain and even injury to the gullet. Analogous painful sensations in the act of swallowing are caused by inflammatory processes in and around the esophagus (esophagitis, mediastinal pleuritis, pericarditis, mediastinitis, etc.); by every possible form of functional or organic stenosis of this organ, or by local tumors. In carcinoma they are accompanied by local tenderness and aggravated by deglutition or when the

patient is in a prone position. In this affection —but not exclusively—these retrosternal pains are not only experienced during the intake of food, but may be present constantly and more so in the night time. Dysphagia is apt to prove a fatal complication. A deep-seated esophegeal diverticulum may also during ravenous eating cause severe pains in the chest, likewise dysphagia and palpitation of the heart. Prompt relief ensues from emptying the contents.

In *peptic ulcer* the pain is generally localized in the epigastrium but often reaches up behind the sternum in the neighborhood of the nipples. They come on in periodic attacks for days or weeks—the same as is the case in ulcus ventriculi or duodeni—and last for a minute or hours with remissions and intermissions and cycles of absolute painlessness. Fairly reliable indications are: when the patient throws up profuse masses of blood, when swallowing of solid food and eventually of liquids becomes more and more difficult. Of course, in such instances the differential diagnosis lies between carcinoma and intermittent esophagospasm. Radioscopic examination should furnish the proof.

In the diagnosis of *esophagism* my advice is to proceed with caution, because it is so frequently the consort of an anatomical lesion, e.g., ulcus or carcinoma of the esophagus.

The retrosternal pains which accompany that

rather rare disease, *dysphagia lusoria,* are localized high up behind the sternum and are due to pressure on the esophagus by the right subclavian artery.

Pyrosis or *heartburn* is another offending member of this family of pains to which belongs also a whole string of *gastric affections,* diseases of the *liver,* the *pancreas* and the *adrenals.* I have already touched on this subject in the chapter on "Cardiac Pains." Our mind turns automatically to the gastric or lower esophageal region when a patient complains of pains behind the lower *third* of the sternum. A carcinoma of the heart or an infiltrating scirrhus of the stomach naturally give rise to such pains, especially when they announce themselves during meal time. But this "warning" localization of pectoral pains is by no means always a requisite sign of gastric trouble. The pains may lie higher up. I saw not long ago a case of hourglass stomach in which the patient placed the pains behind the *middle* of the sternum. Another patient who suffered from gastric carcinoma told me that when going down hill his whole body shook with pain. The same happened in a case of adhesive perigastritis of the small curvature. In stenocardia, on the contrary, this motoric pain arrives with the going up hill.

In peracute engorgement of the liver the

patient likewise experiences a similar motoric pain and also pressure in the retrosternal region when he is going down hill, but epigastric and dextrohypochondral painful sensations are also present. (Cf. volume on "Abdominal Pain.")

Some patients suffering from *liver colic* localize the pain immediately behind the sternum, i.e,, the lower median region, or about the height of the 2. rib, with less intensive pain behind the right costal arch. For a proper diagnosis it is important to note—especially in a differential diagnosis of angina pectoris—that the patient does not quite correctly localize the pain as being behind the sternum, it is more to the right of it on a level with the mamma or nipple, also that he experiences no motoric pains, unless it be during the night, that he finds relief with flatus, that the liver is enlarged, that the urine contains urobilinogen or urobilin, that we find a slight rise in the temperature, pressure pain in the cucullaris on the right side and, perhaps, icterus.

The last three of the aforesaid symptoms suffice sometimes to make the diagnosis positive in those rare cases of *colica hepatica calculosa* (I have seen three of them) in which exclusive pains, and very light at that, are felt behind the sternum or simultaneously also to the right or left of it.

From my own experience I can say that

oppressive pains behind the sternum 2 hours after eating and also after some psychic excitement may well be accepted as a symptom of chronic cholelithiasis.

This selfsame disease may furthermore, run its course with boring, stitching and oppressive pains in the chest and, perhaps, in the back. Sensitiveness on pressure in the region of the fissure of the liver is a valuable hint.

Ulcus duodeni produces the same kind of pain as hepatic colic, but exclusively behind the sternum and to right and left of it in the nipple line. We find minor tension in the upper right rectus, tenderness on deep prodding palpation to the right of the median line near to the gall-bladder, hyperesthesia of the skin, the latter also in the back on the right side of the spinal column over the 12. vertebra; likewise hyperacidity, hypersecretion, gastrectasis, possibly occult hemorrhages in the intestine, and a very instructive Roentgen picture.

Addison's disease harbors pains which are felt oppressively behind the sternum some time after eating. The diagnosis is not difficult to make from the typical symptoms of the ailment.

Similar pains are noticeable in disease or insufficiency of other internal secretory glands, e.g., in *agenitalism* or *hypogenitalism.* They frequently cause sleepless nights. The same may be said of *Basedow's disease,* which should

always remind us of a substantial struma or an enlargement of the thymus as the possible cause of the pain.

We must reckon with an expanding subdiaphragmatic morbid process, especially of the liver (abscess of the liver, echinococcus cyst below the diaphragm, large gummata or carcinomatous growths), when the patient complains of an unpleasant, incommoding feeling behind the sternum.

Neurosis in the form of *nosophobia* is distinguished by pains which are felt only in a very limited part of the sternum. We may observe it in *neurasthenic* people who have suffered an acute psychic shock or are eshausted by overexertion in mental work after a previous attack of syphilis. It is not uncommon among medical men who are liable to make an individual erroneous diagnosis of sclerosis of the coronary arteries, or of an aneurysm of the aorta, or even a retrosternal gumma. The negative Roentgen picture, negative clinical findings, repeated negative *Wassermann* reaction and the proof that the pains are not due to physical overexertion but rather to a mental strain, soon calm the mind of the patient and remove the pain.

In *anginoid conditions* it is not pain so much than a feeling of oppressive constriction behind the sternum, that is felt by the patient. If this sensation combines with a similar feeling in the

throat it bears another meaning for the particulars of which I refer the reader to "Pains in the Neck."

Asthmatic people complain of sensations such as are described above.

In *chronic indurated mediastinitis* the *Oliver-Cardarelli* sign and the Roentgenogram are both of value. Pain is of minor significance because it is very faint, but there is a sort of timid, incommoding feeling behind the sternum together with slight signs of dysphagia.

A similar molesting, oppressive, retrosternal sensation—I agree with *Rosenbach's* theory—is often experienced by people who, for instance, *bend over a desk* for a long time when engaged in strenuous work. They breathe little and superficially, forget it almost, so to speak, and are forced to take in quick and deep draughts every now and then. An oppressive strain makes itself known near the xiphoid process and in the adjacent sternum, in the neighboring parts of the chest, in the muscles of the neck of the lower thorax and the abdomen. Whether the triangular muscle of the sternum is involved is a mere matter of speculation. In fact we have no knowledge of any disease by which this muscle is affected.

When the pains are localized not behind but *at the side of the sternal margins with* local sensitiveness we should be on the lookout for

hysteria. It is well-known that the parasternal pressure points form an important hyperalgesic region for the diagnosis of this disease.

Neuritis, possibly also neuralgia, *of the phrenic nerve* is chiefly the sequel of diaphragmatic pleuritis, or pericarditis or subdiaphragmatic peritonitis or perihepatitis or peripleuritis. It is characterized by a pressure point in the neck between the sternocleidomastoid and the scalenus anticus, by the bouton diaphragmatique, i.e., a pressure point at the crossing of the outer sternal margin and an imaginary 10. rib, also by painfulness on pressure along the 9. and 10. rib on a level with the line of attachment of the diaphragm, and finally by a pressure point at both sides of the sternal margins in the first intercostal spaces and laterally from the spine.

Morbid affections of the *mediastinum* (bronchial carcinoma), *mediastinitis,* also *diseases in the diaphragmatic cavity* (spleen, liver, peritoneum) often cause pain behind and lateral from the sternum with or without involvement of the phrenic nerve. Whether there is a parallel idiopathic neuralgia of the phrenic nerve is uncertain.

Pain on the sides of the sternal margins, sometimes spontaneous, but always on pressure, are often the sign of a *gouty diathesis,* evidently due to gouty changes in the sternocostal joints. The diagnosis must be based on the presence of

tophi, pains in the tendon sheaths and in the articulations (mostly without deformities), skin affections (itching, chronic eczema, etc.), digestive troubles, hemorrhoids, cramps in the calves, renal colic, gouty pains in the feet or in certain nervous zones (ischias) preceding acute articular gout, overrich food, sumptuous living, hereditary taint, and above all on the chemical examination of the blood.

Pain in the chest which lies beyond the sternum directs us to the bronchial and pulmonary regions. We are dealing here chiefly with pleurodynia.

When we are told of stitching pains in the armpit or around the nipple, especially during coughing, etc., we shall have to decide between thoracic myalgia, intercostal neuralgia and *dry pleurisy*. Friction fremitus is one of the characteristic signs of dry pleurisy. But friction fremitus is a tricky symptom and leads to many an erroneous diagnosis. We must know how to distinguish between peritonitic, perihepatic and perisplenitic friction. It is either a primary inflammatory morbid process of the peritoneum, mostly of a tuberculous nature, or it is caused chiefly by primary diseases of the liver, the spleen or other intraperitoneal organs.

In endocarditis (of recent origin or the revival of a former affection of the heart), especially in endocarditis lenta the false diagnosis of dry

pleurisy on the basis of a pulmonary infarct is most frequently made, when in reality it is a case of perisplenitis on the basis of an infarct in the spleen. I have seen a diagnosis of left fibrinous exudative pleuritis made in a case of unmistakable leucemic tumor of the spleen coupled with fibrinous perisplenitis which originated from myeloid leucemia and a large solitary tubercle of the spleen.

The deciding point in such cases is the deep focal seat of the maxinal distinctness of the friction fremitus just beyond the lower margin of the lung. This fremitus is often perceptible away high up in the lungs and cannot always be strictly localized within distinct limits. If that is the case then deep palpation and the presence of maximal sensitiveness in the intercostal space below the inferior pulmonary border will come to our assistance. The absence of irritation to cough is only of problematical value, because this may be observed in fibrinous pleurisy as well.

It is scarcely necessary to mention that dry pleurisy is a complicating element in the majority of pulmonary diseases, and even in bronchitis when this approaches the pleural surface, and that it is often the natural cause of pleurodynia. The latter is also observed (in the 10. to 12. intercostal space) in cases of independent *upper lobar pneumonia,* when the pain is not due to

pleurisy but is rather the reflex action of pain in the pectoral muscles or those of the diaphragm (phrenicus). I wish to point out also that despite the central localization of pneumonia (recognizable only by the aid of the X-ray) pleurodynia is often the sign of a pleural involvement.

Stitching pains in the left side are frequently present at the beginning and also during many *infectious diseases* and are due either to perisplenitis or to an acute swelling of the spleen with painful tension in the splenic capsule. *Chronic malaria* is a striking example.

The same quality of pain adheres to *acute empyema,* especially in the *interlobar* form; likewise to *diaphragmatic pleuritis,* but sharper during deep inspiration and coupled with dysphagia.

When a patient complains of such stitches in the chest, either in the anterior or posterior, especially in the lower sections (in a line with the lower intercostal spaces) or near the borders of the costal arches and sharpened by deep breathing or by coughing, we must look for a fibrinous and exudative inflammation below the diaphragm, especially for a subphrenic abscess. (Fuller particulars will be found in the volume on "Abdominal Pain.")

The diagnosis is based primarily upon the correct differentiation from a basal pleuritis, because both have in common diffuse dullness,

absence of *Litten's* sign, but delicate breathing, weakened fremitus and consonance of the voice, pleural friction at the upper border of the dullness. (Fuller details may be found under "Pleuritis.")

Similar pains reach the side of the trunk in acute inflammatory conditions within the retroperitoneal space, such as acute *pyelitis, peri-* or *para-nephritis.* Respiratory complications are not unusual. It requires the most painstaking examination to arrive at a satisfactory conclusion.

When the pain is localized chiefly in the left side, between the diaphragm and the heart, with a sensation of fullness, chronic constipation is indicated, or congestion in haustris of the colon, distension of the flexura coli linealis (rarely of the flexura hepatica), which severely taxes the left diaphragm, especially when local adhesions are present. Free and copious discharge of gases and intestinal contents generally brings relief and clears the diagnostic aspect.

Also in muscular affections of the diaphragm, we find pain in the chest and sides, particularly in cases of influenza, trichinosis and acute polymyositis. Of course, in trichinosis the pains are essentially due to the invasion of the respiratory muscles by the trichinæ, but the fact that these pains are frequently associated with difficulty in breathing and even attacks of suffocation,

and that the patient has a dry cough, should be sufficient proof of diaphragmatic involvement, especially if the other characteristics of the disease are carefully studied. Moreover, the absence of *Litten's* diaphragmatic phenomenon, and the X-ray picture will be adjuvants.

Overfatigue from gymnastic exercise, running, racing or any kind of sport which requires unusual respiratory action, spasmodic coughing or sneezing or singultus, violent retching or laughing are apt to cause stitching pains in the sides. The diagnosis can be easily made when we know the causative factor, when the pain ceases with rest, and we can find no abnormal conditions in the organs. Nevertheless, we should not forget that the primary cause may lie in hyperemia of the liver or spleen.

If the pains continue and we are unable to attribute them to any of the aforesaid causes, we should turn our attention to subacute or chronic affections of the pleura.

Persons who have had pleurisy, sometimes years ago, may be suddenly attacked by violent pains in the sides when panting heavily after running or some specially strenuous physical exertion, that one is seriously tempted to think of a return of the original disease, possibly due to pleural adhesions. If they are chiefly localized in the lower sections of the lungs, we should look for weaker—sometimes stronger—vesicular

breathing, atelectatic crackling, dullness of sound, change of vocal fremitus and an ever perceptible rasping noise in the bronchi.

Pleural adhesions may also be the after-effect of a former injury to the lung (bullet, bayonet, knife) which healed up with a smooth surface of the skin. The clinician may surmise its presence from the history of the case, but he cannot diagnose it. The Roentgen ray is the only means by which it can be discovered. This is also the case in pleural adhesions which have developed after pleural puncture. The pains in this condition are very severe and associated with dyspnea, agony and oppressive feelings, they are all over the chest, no matter on which side the patient lies. If he turns on the sick side, the pleural sinus contractions push the abdominal organs downwards; if he turn to the other side, they are pushed upwards, and so in either case there is painful tension on the adhesions.

But when these pleural adhesions are localized in other than the lower sections of the lungs, the diagnosis is beyond our ken. Only the Roentgen-ray and, in marginal adhesions of the heart, i.e., in pleuropericardial adhesions, the presence of cystolic and presystolic contractions (of the apex impulse, or of a certain part of the precordia, or of the marginal parts of the heart) can give us information. These contractions, according to my own studies on the sub-

ject, may be explained on the ground that the lung is unable to fill in the place left vacant by the shrinking heart owing to interference by the local adhesions.

But not all pleural adhesions are due to acute fibrinous or fibrinous-exudative pleurisy in which long continued pains are suffered. There is another form which is *not* adhesive, but also associated with persistent pains in the sides, and which, it seems to me, is not sufficiently appreciated by the profession. The pain as well as the audible, sometimes even palpable, friction fremitus is due here to enlargements of contiguous connective tissues which rub against each other. The identification of large pleural adhesions should offer no difficulty when a thoracic contraction cannot be explained on any other basis or in post-pleuritic scoliosis of the spine.

It is much harder to trace to their origin these severe pains in the breast, coupled also with dyspnea, which are due to a chain of interlobar cicatrices. In some cases it is utterly impossible for the patient to lie down. Only the Roentgen-ray can enlighten us in this condition. The cicatricial tissue forms a solid union between the mediastinum and the lateral wall of the thorax, so that, no matter on which side the patient may lie, a painful tension on the cicatricial cord ensues.

In a case of pleurisy—it need be only of a

serous character—without cytologic conditions, in which complaint is made of very severe, increasing pains, be they spontaneous or following a pleural puncture, we should think of a malignant *neoplasm in the pleura.* I know, on account of the slight temporary rise in the temperature and the youthful age of the patient, the diagnosis in these cases generally reads: "Serous, very likely tuberculous pleurisy." But in the course of a few weeks a slight constriction of the posterior and lateral sections of the thorax manifests itself; the intercostal spaces on the affected side seem to have grown narrower. We have either a thick pleural induration, with a possible exudate behind it, or a malignant neoplasm of the pleura before us. Whenever the regular symptoms fail us, i.e., absence of metatastic glands, of cachectic edema, of ectasia in the superficial veins of the trunk, of thoracic edema, or absence of a primary tumor; if we cannot find an hemorrhagic, but do find a serous exudate, and no cytologic conditions of diagnostic value, but a slight upward tendency in the temperature, then, and in that case, a complaint of severe and ever-increasing pains in the affected side of the chest should awaken in us the suspicion of a neoplasm. This thought has often been my guide in making a correct diagnosis. A test puncture in the painful zone, with a careful laboratory diagnosis, will forestall

errors. The use of the X-ray should not be neglected.

Subacute or *chronic actinomycosis* is another condition in which very severe pains in the chest give the impression of a pleuritic affection. Here, also, we find a tendency to the formation of very extensive constricting pleural indurations. With this possibility before us we are already far advanced on the road to a proper diagnosis. A local puncture, bacteriological examination and the complement fixation test will do the rest.

The same may be said of *pleural syphilis.* If the *Wassermann* reaction is more positive in the exudate, if such exist, than in the blood, and the presence of spirochetes in the exudate are the pillars of the diagnosis.

We must not forget that even a feeling of slight oppression or of fullness in the chest or pains of a lesser degree in the side may indicate a pneumothorax that has developed overnight, or, perhaps, a sanious empyema or a pyopneumothorax, all of which are causative factors of severe painful sensations.

Not only the vicious character of the pains, but their characteristic localization, on the right near the fourth rib, on the left in the fifth intercostal space, are powerful indications of a special subordinate form of pleural exudation, i.e., of interlobar pleurisy, particularly interlobar

empyema. In addition to these specific pains, we also find the so-called "suspended" dullness in the interlobar fossa, or in a part thereof, displacement of the heart with slight dullness, and what I call the paravertebral circular segment. With these symptoms and the aid of the Roentgen-ray the correct diagnosis is bound to evolve.

Not only primary affections of the pleura, but also *diseases of the lungs* involving the pleura, give rise to severe stitching pains in the chest. In the presence of such pains our mind's eye is naturally directed to all kinds of acute as well as chronic inflammatory processes in the lungs, including acute active congestion, new formations, parasitic growths, the diagnosis of which is to be found in another chapter of this book. I will only point out that the localization of the painful focus is not always a clinical proof of the seat of the disease. In croupous pneumonia, for instance, the patient generally points to the healthy side as the seat of the pain. Perhaps anastosmosis of the intercostal nerves must bear the blame for this error.

If in the course of such a disease, e.g., *pulmonary echinococcus,* the hitherto only slight painfulness should turn suddenly into an attack of very severe pleurodynia, we must be prepared to find cystic suppuration.

Moreover, a sudden attack of pleurodynia,

localized in a specially limited area, is nearly always the symptom of a small, superficial *pulmonary infarct.* A slight acceleration of the breathing act and the cardiac rhythm, and desultory participation of the affected part of the chest in the respiratory movements, may be noticeable. The localized pleural friction may limp behind or be completely corrected.

In *pulmonary tuberculosis* the pains are mostly felt in the lobar region, and are generally a sign of an aftergrowth which has reached the surface with or without a demonstrable dry pleurisy. These pains may, however, also be due to a complicating attack of adhesive pleurisy or to tension from pleural adhesions.

Morbid conditions of the *mediastinum* or of the *mediastinal pleuræ* are further causes of pectoral pain. We have here acute fibrinous or exudative *mediastinal pleurisy,* not unusual accompaniments of pneumonia or tuberculosis. The latter betrays itself by systolic friction, which dies away at the height of the inspirium.

I have already mentioned that *sclerosis of the aorta* frequently provokes pressure sensitiveness in the upper intercostal spaces. I wish to add here that similar pains occur in *sclerosis of the intercostal arteries* in the form of an intermittent dyspragia.

Aneurysms of the aorta and *mediastinal tumors* generally settle rather in certain parts of

the ribs than in the sternal region. The patient complains of pain in the mamillary line, with irradiations in the back. They are not of a constant, but rather of a piercing, more or less violent, nature, coming on in successive attacks, and last for half an hour or so. Analogous pains between the sternum and the nipples occur in mediastinal pleurisy, and may continue during the whole course of the disease.

The fact of the matter is, that all diseases which cause irritating or inflammatory conditions in the posterior *spinal roots* of the upper or median segment of the thorax will also give origin to girdle feelings around the chest and to pleurodynia.

Insofar as mediastinal tumors are concerned, I animadvert only acute or chronic affections of the *mediastinal lymphatic glands,* particularly the pulmonary hilum gland.

I have repeatedly seen cases of acute tonsillar infections in which the patient complained of piercing and shooting pains either to the right or to the left of the sternum in the second or third intercostal space, and outside the parasternal line, which radiated also in the back. Local pressure and signs of bronchitis were present, but conspicuously often only in the region of the upper middle lobes around the pulmonary hilum. I think there is good reason to attribute these pains to an acute swelling of

this hilum gland. The same, no doubt, is also the case when the patient complains about stitches and pressure around that point in the presence of tracheitis or dry bronchitis, possibly combined with local tenderness. Signs of fibrous mediastinitis were never observable in any of these cases, and I am certain the trouble was due to defective nasal breathing, or chronic tonsilitis, if not to an analogous infection of the lymphatic pharyngeal tissues. The Roentgen-ray proves of extraordinary value in such cases.

A primary *bronchial disease,* a foreign body or stone in the bronchial tube gives rise to similar pains in the chest.

The pain in the chest which is experienced by patients afflicted with a mitral affection during any kind of bodily motation is by some authors attributed to an acute congestion of the bronchial mucous membrane. The claim is made that the bronchial veins resp. the veins of the mediastinum are so congested that the blood, instead of being discharged into the vena cava, runs back into the cardiac chambers, and in this manner produces the severe pains in the chest.

In *hysteria,* spontaneous pains in the chest are hardly ever observable, but pain may be elicited by pressing the finger on the so-called intercostal points. This tenderness is generally found at the lower margin of the ribs, about two inches in front of the anterior axillary line, sometimes,

however, only in the eighth or ninth intercostal space.

Subacute or chronic *diseases of the diaphragm* (hernia, eventration) are frequently the cause of pectoral pains which may be mistaken for a symptom of pleurisy.

Diseases of the trunk, of the intercostal nerves, the muscles and the bones are apt to give rise to stitching pains in the sides. In fact, some forms of metastatic neoplasms announce their presence through them.

A special form of complaint is observed in patients who are suddenly visited by exceedingly boisterous pains in the breast. Sometimes only one side is involved, but in many cases the pain goes right through the whole of the chest, or through the middle field thereof, in the region *behind the sternum* and around the *precordium.* So far as affections of the heart are here concerned, the reader will find a satisfactory account in the chapter on "Cardiac Pains."

I mention only *thrombosis* of the *pulmonary artery* and of the *right ventricle,* diseases of the *mediastinum* and *subdiaphragmatic affections* as causal elements of pectoral pains.

Embolism of the pulmonary artery is still more culpable in this respect than even thrombosis.

When the thrombus settles in the trunk or in the main branch of this artery, the pains come

on most suddenly together with an enormously deep pressure, and with a feeling of suffocation. The diagnosis can be made without fail when we can promptly and properly localize the seat of the thrombus, i.e., in the right ventricle (in all diseases of the mitral valves, sometimes in tricuspid, also in arteriosclerosis of the pulmonary artery), or in peripheral veins of the body (in puerperal phlebitis, phlebitis and phlebothrombosis of the lower extremities, in all kinds of infected peripheral wounds, in anal-resp. hemorrhoidal veins, etc.).

Sudden severe pains behind the sternum, coupled with anxious moments, may indicate a *perforation of the esophagus* (foreign bodies, tumor, scald or burns, overtension, rupture in inveterate drinkers), or a suddenly arising communication between the gullet and the windpipe (trachea, large bronchus). The *bursting in the gullet* of a carcinoma or diverticulum, mediastinitis or purulent pericarditis or an abscess in a dependent part are some of these causal factors readily portrayed by the X-ray. There is a sensation as if "something had given way" in the chest. The patient complains of retrosternal pain when swallowing. Food in its passage causes a fit of coughing, and is regurgitated.

Similar pains may be observed in youthful individuals who have suffered from fever, excessive sweating, emaciation, loss of appetite,

distressing irritating coughs or intrascapular pains. The patient generally recovers after having thrown up some ill-smelling, bloody, purulent phlegm or a clod of blood streaked with pus and a black carbon pigment. In some cases a localized bronchitis, broncho-pneumonic, gangrenous or caseous pneumonic focus is formed, caused by the *perforation of anthracotic or tuberculous mediastinal glands.*

The perforation of (mycotic) *aneurysms of the aorta* into the larger air passages or into the gullet is generally accompanied by pains in the chest, but they are of lesser intensity, sometimes missing altogether or coupled with interscapular pains. Hemoptysis and subsequent death from suffocation are the usual result.

II. Pains Only in One Side of the Trunk

Here the trend of mind is turned to the consideration of various morbid processes that may exist in the pleural space, not to speak of unilateral embolism of the pulmonary arteries or infarction of the lungs.

Not only in recent, but also in old, cases of *fibrous unilateral pleurisy,* sudden severe pains in the chest, with dyspnea, may rise to the surface. They generally go away gradually in the course of a week or so, owing, I think, to the rupture of a pleural adhesion with local rupture of the lung through violent coughing.

Similar pains are frequently a coexisting symptom when a *communication between lung and pleura* is being formed. This may take place when a free or sacculated purulent or purulent-sanious, sometimes serous pleuritis or an echinococcus of the pleura or else some diaphragmatic process enters the lung or bronchus through the pleura. We generally find coexisting acute dyspnea, typical expectoration, and even *pneumothorax.* On the other hand, a pneumothorax is much more frequently the sequel of a perforation from the lung into the pleura. The diagnosis should evolve from the

typical clinical symptoms, supported by the X-ray picture.

When a *pulmonary echinococcus* bursts in the pleural cavity, sudden severe pains with dyspnea, and possibly collapse, are the natural consequence. Of course, if we were not aware of its pre-existence, the diagnosis might be a rather complicated matter. But the Roentgen-ray should reveal the true state of affairs, especially if we look for eosinophilia of the blood and a supervening urticaria.

The perforation of an *aneurysm of the pectoral aorta,* chiefly *into the left pleural cavity,* is always associated with sudden serious pains in the chest which are apt to radiate also into the corresponding shoulder. The general clinical aspect of the hemorrhage should suffice for making a proper diagnosis.

Not only the perforation of a supraphrenic, but also that of a subphrenic organ into the pleural cavity gives rise to such pains, but only when a contraction with the diaphragm and with the diaphragmatic pleura existed, and not with the lung. This happens, for instance, in abscess of the liver or spleen, in gastric tumors and cancer of the stomach. The formation of a pleural empyema, pyopneumothorax, hematothorax is the natural result.

When a trauma, affecting the thorax, has directly preceded the onset of pain, especially

in the left side, we must think of a possible *diaphragmatic hernia.* No matter whether it is congenital or acquired, it will always lead to severe, continuous, though fluctuating, pains, which may even reach up into the shoulders, when stomach or intestines or both protrude through the diaphragm into the thoracic space. If the hernia is acute and due to a traumatic condition, it may be mistaken for a pneumothorax. The Roentgen portrait will clear up the situation, and also show the differentiation between hernia and *eventration.*

It is well to remember that in both these morbid conditions the pains in the chest are relatively often only incidental; I mean to say, after the intake of food when the stomach is distended. The sensation of weight on the chest, digestive troubles, and palpitation of the heart are frequent companions.

The perforation of a subphrenic organ, or the formation of *subphrenic abscesses, pneumothorax* or *hematomata* (of the spleen or adrenals), also a sudden hemorrhage in a subphrenic cyst originate pains in the abdomen as well as in the lower section of the chest (cf. "Abdominal Pains"). The anamnesis, an early examination of the patient and the evidence of internal hemorrhage should remove all doubts so far as the diagnosis is concerned.

If in the course of a *fibrinous* or even a mild

sero-exudative pleurisy the patient complains of sudden severe pain in the chest or in one side thereof, or else and particularly in the region below the left costal arches, we must be prepared to encounter an attack of paresis in the left side of the diaphragm. Here we find sudden pain, periodically labored and rapid breathing, accelerated pulse, inhibition of the left thorax and deficiency of *Litten's* sign. We observe further anteriorly on the left a deep tympanitic, full, sonorous sound (not unlike that in pneumothorax) which ascends to within the fourth rib, extending to the middle of the sternum on the right into the median axillary line on the left. Instrumental percussion is positive, there are no physical defects in the left lower posterior region with the exception of pleural friction and a minor basal pleuritic dullness, unless a parallel infiltration of the left inferior lobe exists or a dull tympanitic sound is heard by stronger percussion over the left lower posterior side of the primary lesion. The heart is shifted upwards toward the right. This clinical picture, which is almost the type of an acute diaphragmatic eventration, presents many features of a sacculated pneumothorax, a subphrenic pneumothorax, a diaphragmatic hernia and an acute dilatation of the stomach. That the Roentgen-ray can give valuable information is self-evident.

Of course, a careful clinical examination paves

the way. Against pneumothorax is the unmistakable tympanitic character of the percussion sound. And, again, the displacement of the heart towards the right and upwards differentiates it from pneumothorax. As for subphrenic pneumothorax and diaphragmatic hernia, it will be observed that the appurtenant etiological factors are missing. Acute dilatation of the stomach is the only other condition that craves closer scrutiny.

The thought of an acute *dilatation of the stomach* forces itself involuntarily on our mind when the patient suddenly complains of an attack of severe pain and weighty oppression in the chest. As a rule, it seems to me, the pain is rather localized in the epigastrium resp. the left hypochondrium. Moreover, the upward tympanitic tendency, the displacement of the heart, the other physical signs, including the pleximeter symptom, are also observed in acute diaphragmatic eventration. But there is one moment which speaks definitely for gastrectasis, viz., dry vomiting. The necessity to remove the stagnating gastric contents with the stomach pump, the etiological factor and the quick relief in the knee-elbow position soon make the diagnosis positive.

Of course, there may be complicating circumstances which render the diagnosis more difficult. If in a case of gastrectasis the patient complains

of sudden oppressive pains in the chest, perhaps in the right side only, we must not be surprised when an attack of *acute cholecystitis* (cholelithiasis) is under way. There is no complaint of hypochondralgia or epigastralgia or of sudden retrosternal stern pain. The sensation is exclusively that of excruciating pain with terrible oppression in the breast. If the reader wishes to get fuller details of the diagnosis he will find them in "Abdominal Pain," under "Hypochondralgia dextra."

When a sudden, most excruciating, splitting pain—the patient frequently speaks of stitches in the side—is experienced in the lower section of the lung near the hypochondria we must not forget that the muscular tissue, and not the serous, pleural or peritoneal membrane of the diaphragm is the most likely causal factor. I have spoken about this on another page of this book.

In conclusion I will say a few words about that apprehensive, oppressive feeling in the chest nearly always coupled with labored breathing, but not with tangible pain. It may be as well to let all the originating causes of this condition pass here before our eyes in a short review. First of all the diseases discussed under "Cardiac Pain" are worthy of mention. These are followed by the morbid conditions which affect the thoracic envelope (skin, muscles, nerves,

bones) and those of the abdomen (elevated position of the diaphragm or painful respiration!). They will be further discussed in the section dealing with dyspnea. A similar sensation is also experienced in cerebral toxic affections. Patients afflicted with uremia or diabetes speak of it as a feeling that comes on before an onset of coma, and so do patients who are distressed by the effects of narcotic poisons without being stupefied (chloroform, morphine); those who suffer from shock or collapse and such as are in a pre-agonal stage and yet are in the full possession of their senses.

I may be allowed a final remark. There are persons who pass painlessly through an illness which is as a rule associated with most intensive pains in the chest. I do not wish to be understood as saying that this is due to a physiologic subnormal quality of sensibility. The patient is rather blessed with an unwonted anesthesia of the pleura due to certain pathological conditions. As an illustration I mention the case of a lady that came under my observation. She was afflicted with tabes dorsalis and passed away. The post mortem showed a severe pleural pneumonia. Another female patient had a sanious pyopneumothorax due to the perforation of a gangrenous tuberculous pulmonary carcinoma. Neither of them ever uttered a single word of complaint about pain.

Pains in the Extremities

If pain in the extremities is properly localized in the muscles, joints or bones the reader will find full particulars in the chapter specially devoted to these three subjects. But if the pain cannot be rubricated under any of these headings, then it is very likely the reflex action of pains that exist in some other part of the anatomy, unless it is exclusively confined to the extremital regions.

For the better understanding of the subject we will first of all turn to the study of pain which is localized in one of the extremities only.

Its field of action may be in the *skin,* a manifestation which is of common occurrence in many internal diseases. Demonstrable skin lesions always facilitate the diagnosis very considerably.

The first in order is *erythema nodosum.* Its favorite place of attack is the skin in front of the tibia to which region it seems to be rigidly confined. Analogous eflorescences may, however, be also observed around the knee joints, the thighs and forearms, even on the trunk of the body and in the face. They consist of mul-

tiple, raised, rosy patches, round or oval, from one-half to three inches in diameter and are exquisitely tender, tense and shiny. The condition chiefly occurs in children and delicate young women.

Sometimes the patient complains of spontaneous pains in these patches and of febrile attacks. When the pains radiate into limbs and joints the disease presents the features of a generalized infection with an acute tumor of the spleen.

This affection is of double interest to the internist, because it frequently follows on the heels of an acute angina, or is the accompanying symptom of articular changes in articular rheumatism, a fact which explains its ready association with inflammatory processes in the endocardium or pericardium. To my mind there is no doubt that erythema nodosum is the expression of a bacterial toxemia or bacteriemia of a different kind altogether, and we must not be surprised to find an analogous exanthema during the career of other septic diseases, for instance, in phthisis or in gonorrhea, etc. The diagnosis should offer no difficulties as it is easily differentiated from erythema exudativum multiforme which is confined to the back of the hands. Moreover, in the latter affection we miss the severity of the general symptoms, but we find a tendency in the patches to run together and

spread out towards the periphery (erythema gyratum, herpes iris), and the fact that they are absolutely painless.

It might be more difficult, perhaps, to distinguish erythema nodosum from the deuteropathic form, especially when the latter sets in after a septic affection. But when we consider the favorite seat of the patches, the benign course so typical of erythema nodosum, the cytologic condition of the blood (always normal with the exception of a possible slight polynucleosis—so far as I am aware bacteria have never been found in the blood in this disease) and the coexistence of septic patches of a different character in the skin, no doubts should arise in our mind as to a correct diagnosis.

In *anasarca* pains in the extremities are not very severe, unless the onset of the disease is very sudden and then only when the affected spot is exposed to pressure or friction of any kind. This is also the case in edema due to thrombosis of the vena cava inferior.

It may be of interest to the reader to mention here that I have seen cases of anasarca in which the skin was treated by drainage. Several quarts of fluid had been drawn off when the patient began to complain of pain in the skin at the affected spot, which molested him day and night for a considerable while. Even paresthesias were observed. The cause for these pains and pares-

thesias is not yet clear to me. Neuritic symptoms? There were none. Perhaps it was the toxic effect of the concentrated detritus which remained in the subcutaneous cellular tissue. Or did physical changes take place in the skin itself? I do not know.

In some rare cases of *subacute hematoma* local pains in the limbs have been observed. They may also be due to *bedsores* on the heels, in the region of the trochanters, and on the knee-joints, likewise to *furuncles* and generalized *furunculosis.*

Glanders is another infectious disease which may produce painful inflammations on one of the lower extremities. This affection is fully discussed in various other sections of this book.

In *erythromelalgia* the pains in the extremities are coupled with certain changes in the skin. The characteristic symptoms of this disease are reddening of the skin of the hands (fingers) and of the feet, principally of the big toe and the heels, severe local burning pains, perceptible swelling in the affected parts and copious sweating towards the end of the sickness. But the pain may also be only an accompanying symptom of other affections such as *Basedow's* disease, or chlorosis, diseases of the posterior or lateral grey substance of the spinal cord, of syringo, tabes, neuritis, or hysteria. It occurs likewise as a combination with *Reynaud's* dis-

ease, for instance, in the foot of nervous individuals who suffer from chronic nicotinism. I speak from personal experience. I have also seen a case of erythromelalgia on the right foot coupled with sclerosis of the arteries of the right leg and foot, also caused by nicotinism. The X-ray confirmed the diagnosis.

Furthermore, diseases of the internal secretory glands are a source of these pains, e.g., *Addison's disease, myxedema* and *periglandular insufficiency.*

Similar conditions prevail in the catatonic form of *dementia precox.* We find here characteristic swellings, reddening and cyanosis accompanied by coldness and dampness of the extremities.

Adipositas dolorosa or *Dercum's disease* is characterized by irregular, sometimes symmetric, deposits of fatty masses in various portions of the body, preceded by and attended with, pain. It is chiefly localized on the exterior side of the upper arm, in the region of the deltoid and on the leg (sometimes on the abdomen but never in the head, in the face, the hands or feet). When the pains are localized only in one leg of a very stout person the diagnosis may be in doubt as similar pains are also caused by phlebectasis. But in the latter case the pains are hardly felt on palpation, if at all. They rather manifest themselves when the patient is standing or walk-

ing, but are modified when he lifts the leg up high or is at rest.

Here is another point. *Dercum's* disease is often attended with neurotic, vasomotoric and trophic disturbances. This is apt to lead to an erroneous diagnosis of "neurosis." Careful palpation of the skin should forestall the mistake. Some authors claim that adipositas dolorosa is further associated with sudden, shooting and cramp-like pains of a very severe character, especially in the night time. I have never been able to notice it.

The features described just now are attributed by some authors to *painful symmetrical lipoma.* Perhaps not without cause. But in lipoma the pains are of a rheumatic, gouty character, change about from one place to another, are not localized in the extremity only, but affect the trunk as well. If palpation does not furnish us with satisfactory evidence for a positive diagnosis, the unusual corpulence of the patient and the attending troubles such as short and labored breathing, palpitation of the heart, etc., should still arouse in us the suspicion of a painful adipositas. Moreover, it seems to me that in many cases of symmetrical lipoma the pains considerably antedate the formation of the fatty tumor.

Neurofibromatosis of the skin is still another source of the pains under consideration. They are caused by the formation of multiple nodes

in the subdermal tissue and along the nerve trunks. At first they feel to the touch like pinheads, but later on may assume the size of a plum, are very painful on palpation and distinguished from those typical of lipoma by their harder consistence. Besides, abnormal pigmentation and thickening of the skin, and trophic, vasomotoric and psychic manifestations are frequent symptoms.

I must not forget to mention *erysipeloid,* a peculiar affection of the palms of the hands and soles of the feet. It is characterized by zones of violaceous—red eruptions with burning and itching and due to wound infection. It is also met in persons who eat meat that is going bad, high game or overripe cheese or any kind of unsound food. The diagnosis offers no obstacles.

These symptoms at once establish the differentiation from *erysipelas* which only in rare cases attacks the upper or lower extremities. If it does the patient complains of heat in the affected portion and the pains are severe. There is local reddening, shiny appearance and swelling of the sensitive skin—the patches are shaped like the wings of a butterfly. We find regional enlargement of the lymphatic glands, general indisposition, chills and fever, constant thirst, loss of appetite and possible complicating conditions in the spleen and kidneys. These symptoms should confirm the diagnosis with ease.

It will be more perplexing, however, when we have to differentiate between erysipelas and *phlegmon.* Of course, plegmonic indurations are harder and the coloring is a deeper red, but the sharp delineation of the patches is the distinguishing feature of erysipelas. We may encounter another difficulty. Erysipelas frequently originates from an old ulcer of the leg, the cutaneous tissue around the sore is very much swollen and indurated, the patient is generally well advanced in years and may have suffered from previous attacks of erysipelas. In consequence the local and general conditions would show different characteristics. The red tint is paler, the local swelling is less pronounced, and the sharp demarcation of the patches and local sensitiveness are almost wanting. The diagnosis must here be guided by the slow progress of the disease which is also confined to a much narrower area, the want of distinctive symptoms and very slight rises in the temperature. In cachectic and anemic patients the reddening of the skin is scarcely perceptible, but local sensitiveness and elevation and demarcation of the cutaneous tissue are typical. The name of *erysipelas pallidum* is here applicable.

Pains in the extremities may also be due to a *thrombosis of the arteries of the skin* on the basis of syphilitic or local endarteritis, or to an

embolism of the same vessels in bacterial endocarditis.

Pains in the fingers and hand, also rheumatoid or neuralgiform pains in all the extremities, coupled with vasomotoric crises (blue coloring of the ends of the limbs, cold, hyperesthesia, sudden sweats) and fever are the initial signs of sclerodermia (sclerodactylia). The patient dozes off easily, gets hard of hearing, feels chilly and is molested with itching in the fingers. Such symptoms should arouse our suspicion. The diagnosis may then be confirmed, when we observe the characteristic thickening and hardening of the skin—it is glossy, like parchment, does not wrinkle and does not slide about over its base. If similar conditions prevail in other parts of the body, the diagnosis is still easier. I may add that in sclerodactylia the formation of painful trophic ulcers is another cause of pain in the fingers.

Affections of the superficial nerves are bound by their very nature to cause neuritic or neuralgic pains in the extremities. Of course, there are forms of neuritis in which a mixed variety of nerves is affected but without motoric symptoms; and again, there are other forms which affect only the sensitive nerves, i.e., they provoke pain with or without paresthesias but no motoric symptoms whatsoever.

Central, chiefly *spinal* disorders which usually

lead to deep, deeper or superficially localized pains in the extremities, may sometimes manifest themselves through cutaneous pains only. *Tabes dorsalis* is an example. The pains in this disease are as a rule of a lancinating, dull, deep-seated character, but I have seen cases with superficially localized pains in the skin alone.

Symptoms similar to those of sclerodermia we find in *acroparesthesia,* in conditions of over-fatigue, in neurasthenia or hysteria and, as additional signs, in various other diseases. The patient falls asleep, feels cold, has creeping, stabbing and burning sensations, suddenly turns pale, one or more fingers, especially the tips, get numb and icy—a typical sign of peripheral vasoconstriction.

What, however, puts the typical stamp of idiopathic neurosis upon acroparesthesia is the relative frequency of the nocturnal attacks due to thermic influences, i.e., heat and cold. They do not play the same predominant rôle in vaso-constricting conditions of neurasthenia and hysteria in which psychic emotions are the most frequent causative factors.

In speaking of *deuteropathic acroparesthesia* I will mention that similar paresthesias in the fingers and toes are often accompanying symptoms of *osteomalacia* or *osteoporosis.*

If these pains are of a tearing, stabbing nature they may be accepted as forerunners of

chronic articular rheumatism. They have a nocturnal and early matutinal habit.

In anemic and wasting diseases they again assume the part of accompanying symptoms. I refer to all forms of secondary, acute and chronic, *anemias,* to *chlorosis* and *pulmonary phthisis.* Cold hands and feet and drowsiness are frequent signs in all of them.

In *arteriosclerosis,* in chronic *myocarditis* and in *nephrosclerosis* they are important additional symptoms and undoubtedly provoked by overstimulation of the vessels.

They are also at times the manifestation in part of anatomical disorders of the central nervous system, e.g., *tabes* or *multiple sclerosis.* Note the contrast between the other characteristic signs of this disease and those of acroparesthesia.

In *diabetes mellitus* these paresthesias in the extremities are an early and unmistakable warning, and based upon some sclerotic vascular disorder or on neuritic conditions.

In *migraine* they are the directing and controlling element. The pains seize as a rule both arms, but may be also confined to one only. The pains in the head promptly arrive with the vasomotoric disturbances in the arms. Of course, other vasomotoric manifestations are by no means excluded, e.g., peripheral angiospasm, sweats and vesicular formations in the skin.

In *epilepsy* pretty well the same conditions prevail. We must make a distinction here between a sensible and vasomotoric, and a vasomotoric and sensible epileptic aura. In the sensible and vasomotoric form the sensible aura is recognized by paresthesias or pains in the extremities which, starting at the distal parts, rise to the center, and the vasomotoric is distinguished by simultaneous vascular spasms coupled with hyperesthesia (hyperesthesia angiospastica). It is not difficult to identify this form because in it the characteristic epileptic convulsions with loss of consciousness develop.

The diagnosis of the so-called vasomotoric epilepsy, on the other hand, is beset with difficulties. The vascular convulsions involve most, if not the whole, of the system. The attack is confined to the limbs only although it may have its starting point there. Sweating is profuse, the temperature high and secretion of urine is reduced; if loss of consciousness also supervenes, the diagnosis is simple enough. Our difficulties begin when these manifestations are accompanied by the so-called psychic epileptic element. The same conditions present themselves in the sensible form of this disease. The patient complains of a sudden creeping sensation as if ants were crawling over him. This always starts in the same extremity gradually extending over the whole side of the body and reaching over to

the other half. The patient does not know for the moment where he is, but in a short time recovers himself and the fit is over. A proper and thorough anamnesis is a wonderful support in making the diagnosis.

It is a matter of common knowledge that paresthesia attended with vasomotoric manifestations in the distal parts of the extremities, especially in the fingers, is the usual initial symptom of oncoming convulsions in *tetany* (gastric tetany included). When the convulsions with the typical obstetric position of the hand follow immediately behind the sensible, vasomotoric disturbances, the diagnosis is assured, particularly so when we observe the other characteristics, i.e., facial phenomena, *Trusseau's* or, perhaps, *Schlesinger's* sign, and mechanical or electric sypersensibility of the nerves. It is these very symptoms which differentiate attacks of tetany from similar pseudotetanic actions in hysteria. Note also, that in hysteria the facial phenomenon may be arrested by giving the facial nerve a hard blow or applying some other harsh mechanical force to it.

In tetany *Trousseau's* sign may produce paresthesias without spasms resulting from the mechanical pressure.

There are also, what I take the liberty of calling frustraneous forms of tetany or tetanoid conditions in which the patient complains of

periodical attacks of paresthesia in the extremities. That they are a rudimentary form of this disease may be confirmed by the fact that the majority of persons who suffer from genuine tetany suffer from these attacks, e.g., cobblers and tailors; also they like tetany itself are usually observed in the springtime of the year and in places in which the malady is endemic. The symptomatology given above applies here in like measure, excepting, however, *Trousseau's* sign.

Acroparesthesias and pains in the extremities especially in the hands may be solitary symptoms of *latent tetany,* in which there are no classic tonic muscular spasms. But *Trousseau's, Chvostek's* and *Schlesinger's* phenomena and mechanical and electric hypersensibility of the nerves are the betraying symbols.

Whether the paresthesias observed in the extremities of pregnant women are the expression of tetany, i.e., a lesion of the epithelial bodies caused by the *gravid state,* is questionable.

In *paroxysmal tachycardia* the same conditions prevail as in tetany and epilepsy, sometimes on one side only—an aura of the attack.

Sudden attacks of paresthesia, preferably in the left, but at times also in the right upper extremity, arising in various cardiac diseases, bear a different meaning. When coupled with an oppressive feeling in the chest, with tachy-

cardia and pallor of the face they are the sign of *angina pectoris,* but there are no pains in the chest or in the left arm. (Cf. "Cardiac Pain.") For the prognosis they are of lesser significance. The patient may suddenly succumb without a sign of a typical attack of stenocardia, while severe anginose attacks may repeatedly come on throughout the period of many years.

Occupation neurosis, writers' and *pianoplayers' cramps* are caused by overexertion of the hands and fingers. There are no motoric disturbances. Pains and paresthesia in the limbs are the only symptoms.

Reynaud's disease (*symmetrical gangrene* and *local asphyxia*) is *characterized* by peripheral vasoconstriction, pallor, coldness, the so-called dead fingers and toes with intensive pains. We find strongly marked sensibility disturbances (hyperalgesia, hypesthesia and anesthesia), a livid blue or bluish-black coloration and symmetrical gangrene of the affected parts. It may exist as an independent disease, but it also may be a secondary or associated symptom of other affections, such as diseases of the nervous system, or of the spinal cord (tabes, syringomyelia, etc.), the individual symptoms of which should materially assist in arriving at a satisfactory conclusion. But note, that in these cases gangrene is not symmetrical. I must add also sclerodermia (sclerodactylia), *Basedow's* disease and hysteria.

Extremity-paresthesia is sometimes the forerunner of *Quincke's edema,* e.g., of the hands.

Chronic hypertrophic acroasphyxia is a disease which has only recently come under observation. Its distinctive features are a slowly progressive asphyxia of the distal ends of the extremities with extension into the softer tissue at the same places. These swellings are symmetrical in form, very seldom unilateral. Paleness, cold hands, at times painless and hardly ever sensitive. Differentiation between hysteria and syringomyelia, and between acromegaly and osteoarthropathy is required for a proper diagnosis.

In organic nervous diseases it is rather the paresthesias accompanied by vasomotoric manifestations, than the pains, that play the predominant rôle. We must not forget, however, that even the slightest mechanical strain or pressure on a nerve trunk is liable to elicit paresthesias, for instance, when sitting we cross one leg over the other. As a rule no significance is attached to them, unless it be, that in neurasthenics they occur more frequently and more easily than in normal people, for instance the hands go to sleep, pins and needles in the fingers, upon the slightest pressure on the nerve trunks. It is a different matter altogether when these manifestations set in whilst the body is kept in one and the same, even quite natural, position for an

unconsciously long time. I have seen cases of aneurysm of the aorta in which the patient never complained of pain or paresthesia in the right upper leg. But as soon as he turned on his right side, they would promptly appear and molest him so that he could not go to sleep. The apparent cause was undoubtedly the shifting of the aneurysm. We may find herein a hint that phenomena of this kind are likely due to the pressure of some organic lesion in the nerves.

These purely functional changes in the peripheral nerves pave the way directly to those lesser grades of *peripheral neuritis* which are generally attended by paresthesia, e.g., alcohol neuritis. And again, pressure on the radialis will arouse the sleeper with a painful sensation. This goes away so long as he keeps awake only to return again with the next nap.

The same condition may be observed in any kind of mechanical lesion of the nerve trunk, or in secondary neuritis through toxic or thermic influences.

These phenomena are of importance to the internist in cases of tumor, e.g., of the supraclavicular glands, or in aneurysm of the subclavian aorta, in mediastinal tumors, in diseases of the pulmonary lobar pleura with a contracted apex pleura and in perineuritis; likewise in paresthesia of the upper arms. Their presence

here may mean the involvement of a sternal rib. At other times they are merely forerunners of a true neuritis. As typical symptoms may be considered: increasing painfulness, local tenderness in the nerves and muscles, sensibility disorders, change in the tendon reflexes, trophic disturbances, muscular paralysis, change in the electric reaction and demonstrable thickening of the affected nerve trunks.

I wish to point out that this "forerunner stadium" in chronic neuritis may endure for a considerable space of time. This is, for instance, the case in alcohol neuritis, which is also attended with vasoconstriction in the fingers. If a traumatic condition has preceded the attack of neuritis, the diagnosis may be somewhat involved, but it will find a firm hold in the facts: that the manifestations are constant, though fluctuating in intensity, that periodical total intermittence is wanting, that they are confined to a definite nervous zone, that there is proof of local sensitiveness in the aforesaid limited nervous area, and the absence of hysterical symptoms.

Certain cases of *chronic poisoning*—carbon-oxysulphid used in vulcanizing india-rubber tubes—cause true toxic neurosis. Paresthesia of the extremities is an essential symptom. Other signs are cyanosis of arms and legs, tremor, giddiness, loss of memory, parosnia and pareugesia, dyspepsia, gastric troubles and con-

stipation. Most of these cases are based upon a functional as well as an anatomical lesion of the peripheral and central nervous system.

A paresthesia of this kind may be a constant solitary symptom of a limited peripheral neuritis or of a multiple cutaneous neuritis. Local sensitiveness in the nervous processes and increased painfulness in motation should indicate the proper course for the diagnosis. The sensation of icy cold, icy draughts, numbness in the nervus cutaneus femoris externus should remind us of *paresthetic meralgia.* For differential diagnostic purposes it is of value to remember that it may be, etiologically speaking, the result of any given form of neuritis (gout, diabetes, cold, trauma, chronic lead poisoning, post-infections, etc.), or it may be in part the manifestation of tabes dorsalis, or may also originate from flat foot.

Equally important is the fact that paresthesia in the inner region of the thigh (down to the knee) coupled with incarceration signs point to an existing obturator hernia.

Intestinal auto-intoxication and *flatulent dyspepsia* also have their paresthesias, such as creeping sensations in the hands, heaviness in the legs, together with a number of vasomotoric manifestations. We observe flushing of the face and heat in the neck, dizziness, headache, psychic changes, anguish, irritability; the patient is dis-

gusted with his work or unable to perform his task, or tired of life, there are symptoms of diaphragmatic elevation, of palpitation of the heart and shortness of breath, especially during the night when the intestinal gases are stagnating. Insufficient stools, flatulency, increase of ethereal sulphuric acid in the urine, regulation of the bowels and checking of the intestinal fermentation by the proper diet are important factors which require close attention. A very strong formation of intestinal gas, of course, is chiefly only a secondary phenomenon of irregular circulation in the intestinal blood vessels, but it may also mean sclerosis of the intestinal arteries, abdominal plethora, polycythemia, the initial stage of insufficiency of the heart, inadequate diaphragmatic rhythm or retarded portal circulation. All these points are worth studying.

Uremia is another endogenous intoxication in which we have occasion to observe the condition of "dead fingers or toes" or syncope of the hands. It may exist as an independent disease or may be the partial manifestation of uremic migraine. I shall refer again to *Raynaud's* disease later on.

Paresthesias in one or several limbs are also known as signs of *spinal affections,* but chiefly as companions of painful sensations. We find them in *tabes* when the patient complains of formication in the extremities, especially in the

ulnar side of the fingers (they feel furry), of a peculiar, indefinite sensation in the soles of the feet as if he were walking on india rubber or on cotton batting, the feet feel as if they were wrapped around with fur, he is not sure whether he is walking on a carpet or on a wooden or stone floor. The hands feel as if they were in woolen gloves. When we find such symptoms it behooves us to look for further signs characteristic of tabes and we must not forget that the incipient manifestations of a tabes superior are to be found in the upper extremities together with patellar reflexes, whilst the usual signs in the lower limbs are totally wanting. I mean lancinating pains and ataxia. In these cases pupillary symptoms, ataxia, hypoatony and sensibility disorders in the upper extremities, symptoms in the optic and cerebral nerves (trigeminus) are not unimportant, but I lay particular stress upon the fact that these paresthesias occur by preference in the region of the ulnaris, and that anesthesia of the triceps tendon, likewise analgesia of the ulnar nerve at the elbow frequently attend.

Some tabetic patients complain of sudden painful itching with goose-flesh formation in the skin of the extremities. But when paresthesias appear in the articular region we must look upon them as the advance agents of a coming tabetic arthropathy.

In *syringomyelia* similar paresthesias are

observed in the extremities (and the trunk) attended with vasomotoric disorders. If the patient is insensitive to heat and pain, but shows symptoms of degenerative muscular atrophy in the distal ends of the upper extremities, trophic disturbances, intensified tendon reflex, scoliosis and bulbar disorders, the diagnosis should be plain.

Multiple *sclerosis* is another affection of the central nervous system in which paresthesias in the arm or in the hand alone are the preceding initial symptom of the disease. They may be associated with pains but exist also without them. We hear of numbness and formication in the limbs. For the early diagnosis I draw attention to impairment of the sense of touch, the patient is unable to judge of depth or position of things and is awkward, clumsy in his actions. The differential diagnosis may be difficult under these circumstances, but if we reflect on the absence of subjective as well as objective pain, the question of neuritis will be eliminated. Moreover, the stronger tendon reflex in the lower extremities, the want of muscular atrophy and the absence of reflexes in one side of the abdominal wall, at any rate, speak decidedly against neuritis.

Another alternative is *cerebro-spinal syphilis.* But this question can easily be settled by the *Wassermann* reaction and by the cytologic and

chemical analysis of the cerebro-spinal fluid. The disturbance of the stereognostic sense might turn our mind also to a cerebral lesion localized in the parietal lobe, the more so as the patient frequently complains of periodic headache and dizziness. The want of cerebral pressure cannot be used without risk against a local expanding lesion in the lobe. A safe diagnosis can only be based on the general course of the disease, the conspicuous fluctuations and the regression of the symptoms, the complete disappearance of the brachial symptoms and the evidence of the classical signs of multiple sclerosis, signs which may not set in for years after we have seen the patient for the first time. Analogous paresthesias in the hand or in the fingers frequently enough are observed during the *progress* of the disease.

In *arteriosclerosis* of the spinal cord with the spinal form of *dysbasia angiosclerotica* paresthesias of the lower extremities are likely to occur. (See "Pains in the Chest.")

Pernicious anemia travels in company with paresthesias in the extremities arising from spinal causes. The same conditions prevail also in other forms of anemia and in severe cases of cachexia (carcinoma, tuberculosis, syphilis, diabetes mellitus, alcoholism).

Paresthesias abound in all diseases of the spinal cord, viz., myelitis, syphilis, tumors, hem-

orrhages, in all expanding diseases of the columnar canal (leucemic infiltrations). In all of them, meningitis included, they are surpassed by the pains.

As prodromal symptoms they figure in acute spinal affections; so in *epidemic cerebrospinal meningitis.*

They play a subordinate rôle in *hydrophobia.* If we are dealing with the mitigated form in persons who have been vaccinated with antirabietic serum we shall find sudden symptoms of debility and paralysis in the lower extremities, bladder and rectal disorders, facial paralysis and salivation. The anamnesis and the consideration of the prodromal manifestations, i.e., restlessness, insomnia, depression, loss of appetite, pains in the head, sacrum and joints should lead the way to a proper diagnosis.

But they may just as well be the prodromal symptoms of true rabies when they appear in the part of the body which has been bitten by a mad animal. In this respect they remind one of the paresthesias and dragging pains which are so often observed in the form of a tetanic aura in *tetanus* in the affected part before muscular contractions and convulsions set in. They are always a most serious warning of an impending outbreak of tetanus when a tetanus (wound) infection is suspected.

From what has been said in the foregoing pages

it will be clear that paresthesias of the extremities may be due to different causes in one and the same disease. If in the legs they may be provoked by the encroachment of a leucemic gland upon the peripheral nerve trunks, or they may be due to pressure of a leucemic infiltration on the nerve roots in the intervertebral foramina or to a leucemic infiltration of the meninges. In some rare cases of accompanying pernicious anemia we may have to decide whether the spinal processes do not in an analogous manner cause the paresthesias.

When the patient complains of paresthesias in one only or in both extremities of the same half of the body we must think of a *cerebral affection*. Diseases of the brain occupy the area of the trigeminal nerve which is the original habitat of paresthesias. I mention here the sensation of numbness in one-half of the face, especially about the angle of the mouth and around the nose; the feeling as if that part of the face were entangled in a cobweb when the patient is suffering from an intrapontine disease or from a basal affection of the posterior cranial fossa, or from a cerebellar tumor, cerebral tabes or syringomyelia. On the other hand we must not forget that paresthesias also occur in the arm or in the arm and leg on the same side of the body either independently or in combination with paresthesias in the trigeminal region as a symp-

tom of a cortico-cerebral lesion. This is obvious, because the sensible cortical centers lie behind the central groove in the posterior central convolution, but overlap only a minimal part of the motoric region. Moreover, the localization in the extremities is often subject to definite laws. For instance, in tabes or in affections of the cauda the favorite place is in the region of the nervus cutaneus exterior.

If the patient complains of sudden numbness in the arm, or hand, or in the arm and corresponding leg and in the same side of the face, but lasting only a short time, and motoric impairment is not noticeable we may take it as a symptom of *cerebral arteriosclerosis* with insufficiency of the hemicerebral circulation. This alone is capable of bringing about recurrent paresthesias in the extremities, which, however, may also be superinduced by multiple minor hemorrhages in the brain or by minimal multiple softening processes (cerebrosclerosis). The coexistence of these with sclerosis of the cerebral arteries can only be confirmed by unremitting cerebral morbid manifestations. It follows that paresthesias in the extremities are a possible, though not unequivocal symptom of a cerebral hemorrhage. The other symptoms of cerebral arteriosclerosis are headache, dizziness, amnesia, psychic changes and frequently a feeling of heat in the top of the head.

Thrombosis or *embolism* in a branch area of the anterior arteries of the brain may produce the same kind of manifestations. The topographical diagnosis offers no difficulties if the unilateral character of the sensible disturbance caused by the paresthesias is demonstrable. This is also the case when the disturbance is confined to one extremity only—generally the arm, especially when the fleeting nature of the manifestation, its recurrent tendency, the advanced age of the patient and the presence of sclerotic conditions in other arteries as well are observed. Often enough the remaining characteristic signs of cerebral arteriosclerosis have already preceded the attack. In some cases even precursors of hemiparesthesia, a transient hemiparesis or hemiplegia or impediments in speech may have been observed. (The temporal intermittence is frequently reversed.)

A similar situation in juvenile individuals should remind us of *syphilitic cerebral endarteritis,* in which we may also find additional signs of weakness in the affected extremity. Of course, in some cases the transient symptoms are entirely due to the endarteritic conditions themselves or to thrombosis of a branch artery, to the latter particularly if the cerebral disturbances persist for a long time.

Essential arterial hypertension is another cause. This condition may prevail for a number of

years without molesting symptoms, yet there comes the time when the patient begins to complain of flatulence, dyspnea after meals, indigestion, headache, dizziness and nycturia; we find a second aorta tonus, cardiac hypertrophy and permanent arterial hypertension. Recent observations attribute the cause to a limited sclerosis of the arteries of the medulla oblongata and subsequent irritation of the vasomotoric center.

A *tumor of the brain* in the motoric or adjacent region may produce paresthesias of the extremities. Sometimes they are of a permanent character and combined with progressive cerebral hemiparesis which at first affects only one, but later on both of the unilateral extremities, and subsequent convulsions. But, the same as in sensible epilepsy, these paresthesias may also come on in the form of regular attacks on the affected portion or cover the entire body, or else in combination with motoric convulsive conditions (*Jackson's* epilepsy). The attacks may also alternate with attacks of cortical epilepsy. This applies to tumors of the parietal lobe with like force.

Of course, the diagnosis cannot be evolved from these symptoms alone. We must look for other important signs, such as headache, cerebral vomiting, psychic changes, infiltration of the

optic nerve and other local motoric and sensible irritating manifestations.

In tumors of the central ganglia, especially of the optic thalamus paresthesias appear in the contralateral extremities. The diagnosis of a tumor of the optic thalamus will not be difficult to make if we bear in mind the characteristic symptoms, which are: pain, objective sensibility disturbances (hyperesthesia and anesthesia); signs of a motoric and paralytic character, i.e., hemiplegia, one-sided convulsions with tremor and position involuntarily assumed, contralateral hemianopsia, hemichorea, hemiathetosis, mimic facial paralysis and cerebral pressure. Tumors of the pons are likewise associated with paresthesias. Alternating hemiplegia is the cardinal symptom of this pontile affection.

A tumor localized in another part of the brain may in the same manner affect the sensible centers and produce contralateral paresthesias, for instance a tumor of the peduncle of the brain, but particularly a tumor of the cerebellum.

Paresthesia of the extremities, hyperesthesia, paralysis of individual groups of muscles and impaired memory frequently survive a heatstroke for a considerable time.

There are other regional affections of the brain in which paresthesias of the extremities also but only occasionally occur. I have, however, in the foregoing section paid more attention to those

cerebral diseases in which paresthesias appear rather as early symptoms or at any rate as momentary guiding manifestations and in consequence are of special significance for the diagnosis. But there are also further morbid processes of the brain which up to the present time have not yet been properly localized, I mean *hemichorea* and *hemiparalysis agitans* (forms of disease which are unilateral in the beginning, but become generalized later on). In these affections paresthesias coupled with pains constitute often enough the incipient stages but may prevail throughout the whole course of the disease.

But if the attack sets in on both sides then the accompanying paresthesias and pains in the extremities are also the predominant initial symptom of the disease (chorea, paralysis agitans). The question is still sub judice whether paralysis agitans is not a disease of the secretory glands rather than an affection of the brain. But we know with certainty that various diseases of the hematopoietic glands begin and progress with paresthesias and pains in the extremities, e.g., *myasthenia,* the nosology of which is likewise not yet clearly established, also *Basedow's disease, myxedema, Addison's disease, acromegaly* and *tetany.*

In *acromegaly* nocturnal pains and even more severe rheumatoidal pains as well as vasomotoric

disorders are significant prodromal symptoms. Unless measurable alterations in the distal parts of the extremities are discernable, the diagnosis may be somewhat intricate. However, irregularities (cessation) of the menstrual flow, or impotence in the male, genital atrophy, pressure symptoms in the brain (hypophysis tumor!) bitemporal contraction of the face (hemianopsia) might prove reliable guides. The course of the disease itself ought to confirm our suspicions. The X-ray should be used early and often.

The *ovaries* are another source of paresthesias and pains in the *climacteric period* (pseudogout). As predominant features of the menopause may be mentioned: a feeling of cold in and blue coloring of the distal parts, heat rushes, congestion in the head, profuse sweating, dizziness, fainting fits, abnormal nervousness, tremor, palpitation and accelerated action of the heart, pains in the small of the back, in the bones, backache, neuralgias in the trigeminus, in the ischiatic and intercostal nerves and *Heberden's* nodes. The anamnesis is the main prop of a correct diagnosis. But we must not disregard the fact that climacteric disorders may put in an appearance years before the menstrual flow ceases and also for a long time afterwards. There exists a very strong interrelationship between the ovaries and the motoric centers, proof of which we

see in the manifestation of various paresthesias during the *menstrual molimina.*

Paresthesias in *osteomalacia*—due to vasoconstriction—are a part of the initial symptoms.

In the *caisson disease* the paresthesias consist at first of formication in the extremities and very annoying itching of the skin to be followed by severe boring, gnawing and lancinating pains with subsequent weakness in and paralysis of the legs. The pains, no doubt, are due to irritation of the spinal roots by gaseous vesicles in the spinal fluid, while the paresthesias may be attributed, if not to the same cause, then to the presence of gas in the cutaneous capillaries.

Very stubborn paresthesias in the extremities, (the finger tips and soles of the feet) are not unusual during the convalescent period in *scarlet fever.* But when coupled with angiospasms or with vasodilation they point to chronic *malaria.* (Cf. "Malaria.")

Chronic ergotine poisoning deserves mention here. The diagnosis of this "creeping sickness" is based on the symptoms of contraction of the flexor muscles in the toes, wrists and fingers, and of the extensor muscles of the big toe, knee and elbow, and, if tonic cramps of the muscular system of the extremities and the thorax and manifestations of peripheral gangrene are wanting, then on the creeping sensation and the anamnesis. When the microscope shows the

presence of ergot in the stools, further proof of ergotism is hardly required.

Hemiparesthesia and hemialgesia are associated with *chronic nicotinism.* We also find diffuse, dragging, lancinating pains in the extremities. Headache (pressure), dizziness, mental sluggishness, neurasthenia, fainting fits, tremor, depression, anguish, cardiac arythmia, anginoid, even anginose conditions and visual disturbances are among the salient features. Of course, it is self-evident that these hemiparesthesias have their genesis in unilateral spasms of the cerebral arteries (intermittent limping!).

Of different origin are the paresthesias in *chronic arsenic poisoning.* They are the first symbol of toxic arsenical neuritis and are soon joined by severe pains in the lower extremities, later on by missing reflexes. As classical warning signals we may consider the existence of certain catarrhal conditions in the mucous membranes (conjunctivitis, rhinitis, pharyngitis, bronchitis, gastroenteritis, colic and diarrhea); likewise generalized neurotic states (fatigue, headache, giddiness, sleeplessness), cutaneous lesions (erythema, papules, herpes, furunculosis, brownish coloration of the skin) and fever. Laboratory tests of the excreta (urine, stools, vomitus) furnish additional evidence.

In *lead poisoning,* paresthesias are of minor importance; but there are exceptions, because in

certain cases paresthesias in the upper extremities spring very prominently into the foreground. The blue line, lead colic, arthralgia, paralysis and punctated erythrocytes should assure a positive diagnosis.

Paresthesias in the most diverse areas of the skin are typical of *pellagra.* We find them in the characteristic erythema itself, but also in other places, such as the arms, shoulders and epigastrium; all no doubt of neuritic origin.

Paresthesias in the upper extremities, especially the upper left, and coupled with vasomotoric disorders, form an essential constituent of *angina pectoris vasomotoria.* But they are no less an accompanying, if not an initial, symptom of true *coronary arteriosclerosis.* This item, however, belongs in the chapter on "Cardiac Pains."

Similar conditions come under our notice in *dyspragia angiosclerotica* (intermittent). Insofar as the lower extremities are concerned, this disease is also known by the name of intermittent claudication, or intermittent dysbasia. Although paresthesias and vasoconstriction usher in and accompany the attack, yet the pain is the preponderating symptom.

I wish to emphasize the fact that in some rare cases of dysbasia intermittens angiosclerotica the subjective troubles consist chiefly in the paresthesias, possibly coupled with paleness

and low temperature in the affected limb. If this happens in the arm, it is rather significant, because it might easily lead to a differential diagnosis of *occupational neurosis.* But the fact that in this disease the paresthesias attack exclusively those parts of the anatomy which come into immediate action by the exercise of the vocational calling (piano players, writers' cramps, cigar and cigarette makers) should prevent a plausible mistake. The more so as in dysbasia the paresthesias closely follow upon every kind of muscular overexertion. Besides, here we also find arteriosclerosis and other arterial constrictions.

On the one hand we know that dysbasia (resp. dyspragia) is conditioned by an anatomical morbid process of the arteries, chiefly sclerosis or arteritis, but not absolutely. On the other hand, we are also aware of the fact that every patient who complains of paresthesias such as described above, i.e., a feeling of numbness and cold in the hands and feet, is the possible victim of *arteriosclerosis* or *endarteritis obliterans* in the affected parts. It is likewise a matter of common knowledge that endarteritic or sclerotic arteries are subject to vasomotoric attacks on the ground of hyperirritation of their own vasomotors through arteriosclerotic or arteritic processes.

Again, the constriction of the arteries caused

by sclerosis in itself, i.e., without the co-efficiency of the vasoconstrictors, prepares the ground for paresthesias. This explains why we find at times paresthesias in the ulnar side of the left upper arm, or still more so in the left forearm and in the left hand, sometimes in union with pains, hyperalgesia, more rarely hyperesthesia, in the paresthetic area as an individual sign of aortic sclerosis. The thought also lies near that every form of restricted circulation in the extremities is a likely cause of local paresthesias. For instance, a constriction of the subclavian artery due to exclusive sclerosis of the aortic arc (i.e., pure aortic sclerosis), or due to an aneurysm of the anonyma, a distortion of the subclavia by a mediastinal induration or by the expansion of a mediastinal tumor, may give rise to paresthesia in the appurtenant region, if only in the fourth or fifth finger.

The diagnosis is not hard to make, especially in the face of diminished pulse beat on the constricted side, the low temperature in the affected extremity, the subjective feeling of cold, paleness, marbling or cyanosis of the skin, hyperesthesia, change in the tendon reflexes, conspicuous difference in the blood pressure between right and left, or swelling of the extremity. Constriction of the abdominal aorta superinduced by an embolus developing in its bifurcation and aggravated by a secondary

thrombosis in the embolus can at all times give rise to paresthesias in the lower extremities.

But not only a constriction, but any other kind of *impairment of the venous circulation* in the extremities, will give rise to local paresthesias (varices, thrombosis).

Phlebectasia is affiliated with sensations of creeping, numbness and heat in the extremities, especially when the patient has been on his feet for a long time.

In *thrombosis,* also, there is a feeling of cold, heaviness, numbness and formication. These may be even the first perceptible symptoms. In chlorosis, for instance, a convalescent, run down by a long spell of illness, will complain of paresthesias in the leg caused by thrombosis of the deeper muscular veins. When these alone are thrombosed and no superficial veins are involved, the diagnosis is not always so easy to make, because the common local symptoms of thrombosis, the formation of an edema in the skin, are frequently wanting, and the thrombotic cord cannot always be felt by the palpating finger. Yet, to speak from my own experience, the cardinal symptom of thrombosis is always sensitiveness on pressure in the deeper muscles (of the calf) when combined with paresthesia and a slight swelling of the glands, at any rate in diseases which tend to marantic

thrombosis, e.g., insufficiency of the cardiac muscle, blood diseases, cachexia, etc.

Paresthesias in the extremities are not only a possible sign of an existing thrombosis, but they are also apt to precede the disease, and thus constitute a premonitory symptom. Of course, numbness, an enfeebled constitution and heaviness in the extremity are not always deciding factors. I lay more stress upon subfebrile temperature, and above all on the finding of a climbing pulse, although some authors deny the significance of the latter.

It is not unimportant for the diagnostician to remember that ever recurring paresthesias in one limb may have their genesis also in other mechanical disorders of the vascular system. I have seen female patients who complained about creeping, cold or heat in one or both lower extremities, without any apparent cause for the trouble. I recommended not to lace the footwear so tight and use more elastic garters. Relief came promptly. Close-fitting boots and crossing the knees are other causes of these tribulations. But it is not only congested circulation, but also the effect on the peripheral and vascular nerves, and, not any the less, cold dampness which produce these anesthesias and painful sensations.

In *hysteria,* the vasoconstricting manifestations are generally wanting and the paresthesias

are less characteristic, but instead we have strongly marked sensibility disorders and other hysterical stigmata, all of which are typically different from genuine acroparesthesias.

In *neurasthenia*, spontaneous paresthesias are relatively uncommon, excepting sexual neurasthenia with its sensations of cold in the extremities. But when they do occur a differentiation from genuine neurosis can be made from the general clinical picture, from the other neurasthenic symptoms, and also from the sex of the patient (neurasthenia prevails in men, acroparesthesia in women). Moreover, in neurasthenia, paresthesia is not spontaneous, but rather due to some minor lesion of the nerves, the sensation persists after the nerve trunk has been pressed for a short time, or it comes on when one foot is resting on the other or the leg is a bit tired out.

Cold hands and feet are often the reflex action of *vasomotoric neurosis* due to *adynemia*. Lank, lean young persons, with a glaring stare or a floating rib, who suffer from enteroptosis or orthotic albuminuria, etc., are good subjects for this form of neurosis.

That complains of cold feet or hands caused by any other form of impaired circulation, such as arteritis, arteriosclerosis, phlebectasy, phlebosclerosis, partial thrombosis of the veins, weakness or paralysis of the vascular nerves (peri-

pheral or central), is an important sign of myxedema resp. hypothyreosis due to excessive use of coffee or tea, is only mentioned.

Raynaud's disease is another deuteropathic acroparesthesia which is of special interest to the diagnostician. The patient complains of cold, paleness or else livid coloration of the fingers or toes (dead fingers, dead toes). Such an "isolated" acroparesthesia (digitus semimortuus) has a double bearing: (1) It is frequently an accompanying, if not an initial, sign of true angina pectoris; (2) it is also a common, if not the most essential, sign of uremic intoxication. Albuminuria, cylindruria, hematuria (all of these may be missing for a long time in contracted kidney), reduction in the urinary output, low specific quantity and light coloring of the urine, nycturia, pollakisuria, strong second aorta tonus, high blood pressure, increased tendon reflex, dyspepsia, uremia, very dry, cardio-renal-edematous skin, changes in the fundus of the eye (retinitis, hemorrhage), congested secretion (chlorine, iodide, sugar of milk) in the kidneys, increased retention of nitrogen, indicanuria, *Ambard's* coefficient and abnormal content of urea in the cerebrospinal fluid, are sufficient indications for a correct diagnosis.

Chlorosis is often distinguished by this manifestation of dead fingers with severe pains during regular intervals. The diagnosis should be

obvious because this disease is almost exclusively confined to young women. The peculiar look and appearance of the patient, menstrual disorders, the condition of the blood, the absence of other anemic signs, the effect of iron and arsenic treatment, are other points of interest.

Some cases of *arteriosclerosis, myocarditis* and *nephrosclerosis* are on record in which acroparesthesia was restricted to one finger.

II. Pains in the Bloodvessels

When vascular lesions are the source of pain, the first disease to think of is *angina pectoris,* with its irradiations into the left, rarely in both, and very seldom in the right arm alone, especially on the side of the little finger, and also on account of its initial and concomitant vasoconstricting symptoms. For particulars I refer the reader to the chapter on "Cardiac Pains."

I will touch, however, on a few facts which really appertain to this section. First of all, it is by no means impossible for these typical irradiations to reach out into the lower extremities or loins, or even the testicles. Secondly, painful attacks in the upper, or also in the lower, extremities are liable to represent the initial symptom of angina pectoris; in fact, manifest, recurrent pains in the left arm, or perhaps only in the left forearm, may very well be the solitary symptom of this disease. I will

go even further and claim that pains in the left wrist, sometimes encircling it like a tight bracelet, at other times settling only in the palmar side, are a grave warning—especially when combined with a terrible feeling of agony—of a fatal anginose attack.

In all these cases of peripheral angina pectoris, it seems to me that the pain is centered in the nerves of the bloodvessels, if not entirely, then in part for certain, whence it radiates into the neighboring nervous fields. The diagnosis cannot go wrong when we observe a simultaneous and continued extension of the pains in the chest accompanied by anguish and the other signs of stenocardia. Analogous irradiations of pain in the arm are observable also in anginoid conditions, companions of acute or chronic myocarditis or pericarditis.

Arterial constriction in the extremities is a very ordinary cause of painful sensations in the legs. I aim here at *dyspragia* resp. *dysbasia intermittens, intermittent claudication,* no matter whether caused by arteriosclerosis or endarteritis obliterans or syphilitic endarteritis with or without vasoconstriction, or even provoked by the latter alone. I have already spoken at length about this affection in a previous section and, to avoid repetition, I refer the patient reader to that part of my book.

The painful attacks connected with this dis-

ease are obviously due to overtaxation of the peripheral vessels. We can find the evidence in the absence of arterial pulsation without claudication, in the missing pulse during the attack; or else, in the typical claudication of the remaining pulsation; and again, in claudication based on vasoconstriction alone (in anemia, neurosis, nicotinism), and finally in unilateral claudication when the trunk of the crural artery is sclerosed on both sides and the arterial pulse is missing in the smaller branches. This bronchial defect will be found on that side in which an old pericolitic (post-dysenteric) exudate pinches the ischiadic nerve (thus producing a onesided ischias) and hyperexcites the vascular nerves by this constriction.

Similar conditions may arise from sclerosis of the trunk of the crural or iliacal arteries or any other local constriction or acute arteritis. It is hardly necessary to add that dysbasia is often followed by gangrene. In chronic nicotinism intermittent dysbasia may be caused by periodic or continuous angiospasm when the arterial walls remain quite normal (the X-ray gives the required information). The pulses are modified during the attack but never entirely missing.

Chronic lead poisoning, ergotism, neurasthenia, chlorosis and juvenile anemia must be rubricated here also. In addition be it said that a

combination of several different etiological factors may produce a focus from which dysbasia is set in motion. Is it necessary to say that there is a form of neuritis which takes its genesis from arteriosclerosis and that in consequence neuritic manifestations are also observed in dysbasia? But this does not justify us to apply the term "neuritic dysbasia" to a form of dysbasia which is conditioned by neuritis.

As changes in the arterial, so may anomalies in the venous passage ways of the blood give rise to intermittent dysbasia, in fact the more so when the arteries have already been attacked. Phlebectasy and phlebosclerosis are the chief offenders. The diagnosis is facilitated by the ectatic state of the veins of the skin which is plainly visible. In women who have offspring or are with child, in persons who are forced to stand most of the time, or in very corpulent individuals the veins are not always very visible. In such cases the bloodvessels can be made prominent if the patient is asked to let the lower limbs hang down loosely for a while. In some cases of phlebectasia the trunks of the veins are not perceptibly enlarged, but we shall always be able to find a bluish network of the smaller vessels on the bridge of the foot or else on the inner surface of the thigh. When the muscular veins are very strongly varicosed while the cutaneous vessels show only slight traces of ectasy we may

with safety ascribe an existing dysbasia to that condition. Errors in the diagnosis can always be avoided by a careful clinical examination. Nevertheless, for safety's sake, I will add a few hints.

It will happen that *ischias* is diagnosed when it is really a case of *dysbasia* and not without reason. There are cases of ischias in which the pain is very much aggravated by walking. But we may be safeguarded when we take into consideration that the ischiatic pain is constant, perhaps only in a milder form, when the leg is at rest. Moreover, we have the typical localization of the pain in the region of the ischiadic nerve, local sensitiveness there, sharpening of the pain when the abdominal press comes into action (during coughing, defecating), tenderness in the lower lumbar vertebræ, *Lasegue's* sign, change in the Achilles tendon reflex, lower temperature in the patellar skin of the affected part. The diagnosis of radicular ischias will be discussed later on.

Peripheral neuritis is another stumbling block, because here also the pains are exacerbated by walking, the affected muscles are stiffened and indurated. But the diagnosis can be made from the typical signs of neuritis, eventually pseudotabes (sensitiveness of the nerve trunks and muscles, muscular atrophy, trophic disorders,

motoric inhibitions, ataxia, changes in the tendon reflexes, reaction to the electric current).

Another source of error consists in a subordinate form of neuritis in the legs, i.e., *arteriosclerotic neuritis* which rests on the same basis as dysbasia, i.e., sclerosis of the arteries. The pains, noticed at first during walking, later on also when at rest, are localized either in the crural or ischiadic zone, or in both together.

Sensibility disturbances and tenderness in the nerve trunks are hardly ever present. But a differentiation is rendered possible by the decline of the tendon reflexes, the presence of muscular paresis and atrophy, and also by the existence of the arterial pulse in arteriosclerotic neuritis. In complications between the two diseases the tendon reflexes and the arterial pulse in the foot are wanting.

Another important point is that both neuritis and arteriosclerotic dysbasia may have a parallel existence, independent of arteriosclerosis as a causative element.

I know full well that despite this apparent parallelism, dysbasia can only exist as a unilateral disease. Still my contention is that wherever the nerves of the extremities are most vulnerable, there and in that place intermittent claudication may also take its exclusive abode. Also, insofar as the development of dysbasia is concerned, we must reckon with two factors,

i.e., stability of the vascular affection and the transitory element of the nervous system, especially of the vasomotors.

Acinesia algera causes pains in the arms or legs. It is easily recognized and distinguished from dysbasia by the fact that the pains set in at once when the patient even attempts the slightest movement, in consequence he remains motionless. There are no morbid changes in the bloodvessels. The pains are felt with the same intensity in other parts of the body, chiefly in the back and in the head. We also find generalized neurotic symptoms of hysteria, neurasthenia or psychosis.

Neurasthenia is sometimes a regular counterfeit of intermittent dysbasia. The patient does complain of rather moderate pains after a long walk, but soon finds rest again. We shall not go astray if we keep a careful eye on the following points: no changes in the bloodvessels, no trace of other causal movements, but presence of other neurasthenic signs, the youthfulness of the patient (generally male), proof of preceding psychic emotion and overexertion. The prognosis is favorable.

In *meralgia paresthetica* the pains come on with walking, at first the exercise modifies but later on aggravates them and they disappear only when the patient comes to rest. The painfulness, however, is clearly confined to the region

of the nervus cutan. femoris externus with hypesthesia or anesthesia of the skin.

Flat foot accounts for another error in the diagnosis of dysbasia. It is one of the commonest causes of pain in the lower extremities and for that very reason so often misunderstood, simply because the patient very rarely complains of pain in the foot. He rather speaks of a painful tension in the calves or knees, of paresthesia in the outer side of the thigh (meralgia), or in the hips, so that an erroneous diagnosis of ischias, or of a painful affection of the knee—or hip joint easily slips in. And yet the diagnosis is plain and simple enough, if we only bear in mind that not every patient who comes to us for advice must of necessity be the carrier of an internal disease. In a way flat foot resembles dysbasia, that is to say, the patient complains in both of pains in the leg, i.e., in the calves, in the nates and in the loins, pains which are awakened by *walking* but disappear during the resting time. If they are mitigated or even vanish during a longer walk we have evidence against dysbasia. We know very well that flat foot patients suffer from severe pains when they have been standing on their feet all day long. In the morning no pain is felt but it comes on and increases in intensity as the hours go by. This is not the case in dysbasia. Here the pains cease so soon as the extremity is put out of

action. Moreover, there are definite external signs by which a pes planus can be recognized, the instep appears inflected, the inner condyle is prominent, a vertical line drawn from the middle of the popliteal space through the achilles tendon deflects outwardly instead of hitting the median line of the heel. The patient walks on the outer border of the foot and wears out the heels of his shoes on the outer side. Flat feet are, as a rule, abnormal in length. Hallux valgus of the big toe and corns on the little toes are other characteristics worthy of notice, and so are pains in the foot itself or when following passive supination of the foot in which case they generally radiate in the knee and hip joints and even into the small of the back as an expression of static arthritis.

A common sequel of flat foot is venous ectasy further complicated by subsequent dysbasia. We must bear this in mind when a patient afflicted with perceptible ectatic veins complains of pains in the legs, especially for the reason that the self-same causes may also originate phebectasy, e.g., preceding pregnancy. Of more common occurrence still is intermittent arteriosclerotic dysbasia combined with flat foot. We shall recognize it when the arterial pulse in the foot is wanting.

Of course, it is understood that all forms of talipes cause pains in the feet and legs. They

belong really in the province of the orthopedic surgeon.

Achillodynia is a symptom complex which forces the patient to complain of intensive pains in the process of the achilles tendon when the feet and legs are in motion. We generally can find a small local tumor about as hard as the tendon itself. It is due to some slight injury, a pinching shoe, periostitis or tendovaginitis and of interest to the internist for the reason that it may also be the effect of gout, gonorrhea, malaria or even syphilis. A purely psychogenous hysterical form of achillodynia seems also to exist.

A long forced march or some other unusual overexertion of the lower extremities is frequently followed by an attack of very painful dysbasia. The patient can walk no further on account of muscular cramps in the calves, or in severer cases owing to a traumatic *rupture of some muscular fascia.* The diagnosis is simple and plain enough.

Myotonia is a muscular affection which owes its origin to some primary disease of the nervous system. Its special symptom is a painless rigidity of the muscles following prolonged physical exercise especially when the patient has been inactive for a considerable space of time previous to the overexertion. Yet there are cases in which painful tension in the thighs and calves

is caused by walking. The pressure of hypertonus in an otherwise well developed muscular system, the fact that the symptoms disappear in continued exercise, the absence of vascular signs, and *Erb's* myotonic reaction facilitate the proper diagnosis.

Resembling myotonia and for that reason often mistaken for dysbasia are certain rare cases of *trichinosis* when the patient complains of cramps and stiffness in both calves, which makes walking difficult but disappear again with rest.

There are also sporadic cases of osteomalacia in which intermittent limping is caused by vasoconstricting cramps in the vessels. The characteristic symptoms (see chapter on "Pains in the Bones") suffice for the diagnosis.

At first sight we may gain the impression of dysbasia in certain cases of arteriosclerosis. The patient complains of pains in the legs which, however, are not at all due to the sclerotic condition but to an entirely different cause. I remember such a case. The patient complained of pains every time he made an attempt to walk and showed all the characteristic symptoms of arteriosclerosis. But the fact that he felt pain in the legs when he turned around in bed and that constant severe pains were present in the sacrum turned my attention to the retroperi-

toneal space. Upon closer examination I found a carcinoma in the pancreas with fatal result.

Dysbasia or rather *dyspragia* in the upper extremity must be suspected when the patient complains of pain provoked by movements of the arm. The cause for this congestion in the circulation may be either sclerosis of the arteries and their branches, or a sclerotic constriction of the subclavian artery with abnormal vasomotoric irritation, or, less frequently simple vasoconstriction.

The differential diagnosis between dyspragia and occupational cramps can be made from the fact that in the latter no anatomical changes in the walls or in the lumen of the vessels are perceptible, that there is no difference in the blood pressure on the affected and healthy side, and also from the quality of the pulsation, all of which are characteristic symptoms of dyspragia, where the arterial pulse may be even entirely wanting in the affected side. Furthermore, in dyspragia the pains come on with any kind of physical activity, e.g., during meal time or when combing the hair and not rarely with a definite occupation, e.g., writing or playing on a musical instrument. Again, dyspragia manifests itself sometimes only when the patient is walking attended with paresthesia, paleness and cold in the affected part. All this is due to sclerosis or dilatation of the appurtenant aorta,

or to pressure of a cervical rib on the underlying cervical plexus and on the subclavian artery. Here, too, paresthesia may manifest itself shortly after motation of the arm. Stitches, cold or hot feeling in the skin, motoric weakness amounting to a quasi paralysis incommode the patient to such an extent that an object he holds in the hand falls to the ground.

Diseases of the vascular system give also rise to other varieties of pain in the extremities.

First of all I mention here that *sclerotic arteries* give rise to moderately painful sensations along their whole length or wall by way of the vascular nerves. Perhaps we find the artery sensitive on pressure, i.e., when we squeeze and roll it between the fingers. We may also detect changes in the arterial wall when we palpate it with the finger nails. This is a valuable hint which I have tried out on my own arteries.

Long continued pains in one or both lower extremities combined with intermittent limping is the natural sequel of arteriosclerosis with secondary vascular cramps superinduced by excessive smoking or a psychic shock or some physical overexertion.

Arteriosclerosis of the extremities—the same as endarteritis—in younger persons is the causative factor of local pains, especially in the distal parts of the extremities owing to defects in the circulation. I have already pointed out that

paresthesia, pain, coldness in these distal parts, marbled, livid, pale skin, smallness of the peripheral pulse are the ruling symptoms for the diagnosis of such arterial affections possibly followed by subsequent gangrene. Arteriosclerotic neuritis and radiculitis are other means by which arteriosclerosis is able to provoke very intensive pains in the extremities. These pains, at times intermittent, may, in fact, constitute the one solitary symptom of the disease, because, as a rule, tenderness of the nerves and muscles and other objective sensibility disturbances are completely missing. The differentiation from arteriosclerotic dysbasia rests on the finding of the arterial pulse.

Arteriosclerosis of the extremities (arteritis obliterans) and *Raynaud's* disease are the originators of the highest degree of pains in the extremities (the distal parts) when they culminate in *gangrene.* It follows that, when we are confronted by gangrenous conditions, we will have to decide to which of these two diseases the present state is due. If due to arteriosclerosis the pains have preceded the attack by several weeks, if *Raynaud's* disease is the cause they have existed for several years. In *Raynaud's* disease, moreover, the gangrene is exquisitely symmetrical and synchronous in both extremities and remains confined to the same locality, whilst in arteriosclerosis it is of a progressive nature

with total loss of pulsation. And yet there are cases in which these points do not afford a convincing differential proof, when even the Roentgen picture fails to come to our assistance. This will happen, for instance, when *Raynaud's* disease—likewise erythromelalgia—runs a parallel course with arteriosclerosis.

Very sudden pain in one of the extremities may at any time be ascribed to some disease of the arteries. *Acute,* toxic, infectious *arteritis* obliterans ranks first in this connection with some rare cases of severe acute infections (typhoid, influenza). In its secondary stage it is apt to give rise to thrombosis of the arterial trunk. The manifestations of arterial ischemia, i.e., the absence of pulsation, pallor and coldness, hyparthesia and anesthesia of the extremities are sufficient guides for the diagnosis. According to recent observations a severe cold in the form of acute rheumatic arteritis is a likely factor when it has directly preceded the attack of arteritis of the trunk. It may be known by the sudden onset of severe, cramp-like pains in the extremity on physical movement or even when the patient is resting, the absence of the peripheral pulse being probably the solitary demonstrable sign. An interesting point which should not be lost sight of, is that pneumonia is apt to supervene in such cases with subsequent spastic hemiplegia in the right side of the body and

motoric aphesia, but without apoplectic conditions.

Arterial pain in the extremity may be the sign of a sudden occlusion of one of the branches, or of the trunk itself. I have in mind here *embolism* of the subclavian or brachial, the femoral or popliteal artery, if not *thrombosis*. If it is an embolism we shall have no difficulty in finding the thrombotic focus nearly always in the left ventricle, likewise local asphyxia, sudden loss of pulsation, icy coldness, hyperthesia and anesthesia with secondary symptoms of gangrene.

Of course, the primary thrombotic focus may also be localized in the aorta (atheromatous ulcer) or in the pulmonary veins in which case it will be beyond recognition. But if it be in the heart itself we are dealing with a recent endocarditis or a thrombosis in the left side of the heart, i.e., either in the left ventricle or in the left auricle. In the latter instance stenosis of the mitral ostium is the cause of the thrombotic condition. A *ball thrombus* is distinguished by mitral stenosis and supervening gangrene in both lower extremities. This gangrene may originate either from thrombosis of the arteries in the legs, or from an embolism generated by the ball thrombus in the auricle. When the embolism affects both popliteal arteries and runs parallel with an ascending thrombus in both,

an occlusion—though not necessarily total—of both femoral arteries is the natural result.

There is still another form of occlusion of the arteries either in both legs or in one only which is attended with most maddening pains in the occluded area. Here mitral stenosis generates thrombosis in the left auricle with a subsequent *embolism of the abdominal aorta.* The end result is an embolism of the common iliac artery, with severe pains in the abdomen and legs. Paresthesia may have preceded or may accompany the attack, but there is no definite change in the arterial pulse. On the other hand motoric weakness and decline, if not total absence of the patellar reflexes is always noticeable. The pains gradually subside only to come on again suddenly with renewed vigor, at first in one leg only from the knee down to the toes. This is followed by missing pulsation in the arteries of the foot, pallor and coldness in the foot and in a part of the leg and anesthesia. The process repeats soon after in the other leg also. What I have said is taken from my own practical experience.

In some cases thrombosis or an embolism of the abdominal aorta localized in the bifurcation of the common iliac artery *alone* is sufficient to produce a high grade ischemia in it and in its branches. We find pains in the abdomen and in the lower extremities (sometimes in the lower

part only), pale appearance of the skin which feels cold to the touch, hypesthesia and anesthesia, missing or very small peripheral arterial pulse, painful paraplegia and gangrene. I have seen cases, however, in which all these symptoms were wanting, but instead I found a very pronounced stenosis of the mitral ostium with thrombosis of the left auricle, and in all toes and their adjacent parts symmetrical signs of a very severe venous stasis and arterial ischimia, strongly resembling initial gangrene. And yet there was no evidence of thrombosis or embolism of the arteries, but anatomico-histological signs of severe venous stasis with secondary terminal thrombosis of the venous trunks and strong constriction of the arteries could be noticed.

When an embolism in the branches or in the trunk of an artery in the lower extremity or in the region of the abdominal aorta produces sudden very intensive pains, a mistaken diagnosis of neuralgia is frequently made. The same may be said with equal force of *thrombosis,* especially in the initial stages of the disease and before a secondary constricting and occluding thrombosis has supervened. The symptoms of local asphyxia and gangrene are, of course, the same in both cases, for which reason the differential diagnosis is sometimes hard to make unless synchronous abdominal pains indicate the localization of the occlusion.

Arterial constriction is always a possibility when the patient complains of continuous pains in one of the extremities or in a distal part thereof no matter whether paresthesia is in evidence or not.

All that has been said in the foregoing pages about pains in the lower extremities applies in like manner to the upper extremities. It should not be difficult to recognize analogous affections of the anonyma or the subclavian artery.

In the chapter on "Muscular Pain" I have already said that severe pains in the extremities may arise from an attack of *periarteritis nodosa,* for which reason I only mention the subject here.

I will now speak of *morbid conditions in the veins* as a possible source of pains in the extremities.

Acute *thrombophlebitis* occurs more frequently in the lower extremities and is attended by very severe pains, a climbing pulse and premonitory rise in the temperature. Fever, edematous conditions along the entire course of the affected vein (marantic thrombosis in the radicular zone), local painfulness and the unyielding consistence of the vein when palpated are adequate symptoms for a correct diagnosis which may be further strengthened by the consecutive formation of collateral cutaneous veins or the accession of a pulmonary embolism or, if the foramen

ovale be open, of paradox embolism. These symptoms are not always very distinct, but the painfulness of the affected vein and the difference in the local temperature will help us to avoid an error.

When once satisfied that we are dealing with a case of thrombophlebitis we shall have to hunt for the originating factor. It behooves us here to consider that the cause of the affection may not only be an infection localized in the radicular zone of the vein, but may just as well consist of an inflammation in any part of the venous wall. I call attention to the frequency of phlebitis in the lower extremities when the patient is suffering from an infectious disease of the uterus (puerperal thrombosis), to other morbid processes in the small pelvis or in the retroperitoneal space, e.g., of the male genitals or the bladder, appendicitis, perityphlitis, pericolitis, perisigmoiditis, acute ulcerous colitis, perinephritis, pyelitis, renal neoplasm or a disease of the pelvic bones. Phlebitis may also have its genesis in a general septic infection, e.g., typhoid, influenza, smallpox, syphilis, etc.

Marantic or cachectic thrombosis is the sequel of insufficient cardiac action, of generalized cachexia, severe anemia, or venous constriction due to encroaching tumors or else to dilatation of the veins, etc. The pains arrive suddenly generally in one of the lower extremities with

local coldness and blue pigmentation of the skin. A thrombus can be palpated in the vein which feels like a quill, the distal collateral veins are enlarged (frequently the first and solitary sign of the thrombotic condition) to which may be added an edema in the radicular zone of the morbid vein. But I warn the observer not to take such an edema as a conditio sine qua non. Many errors have sprung from this assumption, because in many cases it does not exist, especially when the affection is in the deeper muscular veins. The presence of pain—mild though it be in many instances—of local sensitiveness, enlargement of the collateral cutaneous veins, no doubt, will guide our judgment in such cases, particularly when we are able to palpate the hard, waxy consistence of certain neighboring muscles.

The next thing to do is to ascertain the primary cause of the disease. With regard to that I repeat again that not only local defects in the circulation such as varicose veins, or contraction of the venous trunks, but also generalized infections may breed such thromboses. We call them cachectic thromboses because they may follow after any form of cachexia.

Every case of thrombosis in the extremities is a warning signal of a latent carcinoma, and in the same measure, thrombosis in a vein of the arm points to an expanding morbid process

in the mediastinum with constriction of the superior vena cava. Be it remembered that thrombosis of the superior as well as the inferior and also of the iliac vein is hardly ever associated with pain, because it seems to me, the affected extremity derives a competent supply of blood from collateral veins, which, however, is not the case when the principal vein is choked up. Perhaps, this explains the occurrence of thrombosis in the inferior vena cava with edema in only *one* of the lower extremities.

Thrombophlebitis or phlebothrombosis of this kind in one of the lower extremities is indicated when the patient complains of sudden pains in the affected part, when we find livid coloration, coldness, wanting pulse, anesthesia and initial gangrene. As a rule we are inclined to ascribe these symptoms to a defect in or an arrest of the arterial circulation, e.g., arteriosclerosis, endarteritis, embolism or thrombosis. And yet an error may creep in as I know from personal experience. In one case the post mortem showed a recent endocarditis; while the arteries in the legs were quite intact there was thrombophebitis of both the crural veins.

Another case presented an ulcerating carcinoma of the stomach in a man over 60 years old, who three days before his death suffered keen tearing pains in the left foot with coldness, cyanosis, wanting pulse and anesthesia. The

post mortem showed a marantic thrombosis of the left popliteal vein, very rigid arteries and atrophy of the heart.

A third case was that of a female patient. fifty-one years of age, who was afflicted with stenosis of the mitral ostium, insufficiency of the mitral valve and of the heart. The post mortem revealed defective arterial circulation causing venous stasis, secondary venous thrombosis and the clinical symptoms of an initial gangrene which could not be anatomically recognized.

Simple *phlebosclerosis* and also *phlebectasia* lead to paresthesias, an individual form of intermittent dysbasia, and rather moderate, dragging pains, especially in the calves. We find trembling, tension, a feeling of heat and itching in the lower extremities, symptoms which are sharpened by standing or otherwise overtaxing the muscles of the extremities. The pains come on also when the patient has been walking a long distance or is carrying a heavy burden, but disappear when he quickens the pace or is climbing steps. The diagnosis has already been discussed but I will add that here, too, mistakes may be made when the pains announce themselves in an unwonted fashion. Flat foot and varicose veins are possible sources which are frequently overlooked.

Phlebosclerosis bears the same stigmata as phlebectasia. The most prominent among them

is thickening of the venous walls, easily demonstrated by clinical means and the X-ray. Edema of the knuckle is common to both. It becomes troublesome in the evenings and if the disease is protracted it may assume proportions which resemble elephantiasis. The overlying skin is rigid and immovable but pigmented with a diffuse brownish tint owing to frequent local hemorrhages.

The same symptoms, i.e., pain, edema and perceptible enlargement of the collateral veins are attached to *chronic phlebitis.* It can be distinguished from simple phlebectasia by the intercurrent acute exacerbation of the inflammation and by the proof that it arose from an acute attack.

It is self-evident that pains in the lower extremities, especially in the calves, belong to the initial signs of a beginning congestion in the inferior vena cava and in consequence of muscular *insufficiency of the heart.* The other symptoms of the latter such as fatigue, headache, dyspepsia and dyspnea will assist the diagnosis materially.

Inflammatory wandering diseases of the lymphatic vessels are also attended with pains in the extremities. Acute *lymphangitis* as well as acute, subacute and chronic lymphadenitis are frequent causes of such pains, especially in the arms. The former stands out by the manifesta-

tion of chills and fever, general indisposition, long streaks of reddening, slight infiltration but very decided painfulness of the affected parts. Lymphadenitis, on the other hand, is distinguished by painful swelling of one or more lymphatic glands, especially in the axilla and in the groin.

Pains, swollen lymphatic glands, edema and infiltration of the periglandular skin (hemorrhages and vesiculation) and absence of lymphangitis are the most prominent signs of bubonic *plague*. The patient complains from the very beginning of pain in the glandular region (groin, armpit, neck) and of local tenderness.

The seat of pain in the extremities, of course, may also be in the muscles, in the nerves, joints, tendons, fascia, ligaments or synovial sacs, in the bones or in several or all of these organs. For fuller details I refer the reader to the respective chapters on these various subjects confining myself in this place to the discussion of pain arising clearly and definitely from nervous disorders only.

III. Pain in the Nerves

I will not go into details here about pains in the extremities which arise from *peripheral neuritis*, but only mention that a lesion of the peripheral nervous system does not always attack

the entire system in equal measure, but may only affect a certain portion of it; for instance, chronic alcoholism is often manifested by pains or paresthesia in one extremity only. Furthermore, I wish to point out that in neuritis of one nerve-trunk the pains are localized solely in the distal parts of the affected extremity that is to say in the peripheral branches of the nerve, e.g., in neuritis of the ischiadic nerve (ischias) the pain exists in the knee or in the heel, or in neuritis of the brachial plexus it is in the tips of the fingers. In such cases the behavior of the tendon and skin reflexes and the presence of nerve-pressure points are of decided diagnostic merit. And finally, let me add, that injuries of a nerve or a nerve-plexus may be associated with paresthesias and pains not only in the affected extremity, but also in the corresponding extremity, no doubt via the shortest route of sympathetic irritation.

Pains in one or in both extremities may arise from any kind of morbid conditions which irritate the trunk of the nerves proximal to the extremity or the radicular zone of it either by compression or by way of a perineuritis. We can recognize these truncal pains by their irradiation into the appurtenant branches, by synchronous paresthesia, by their neuralgiform character, their permanence with periodic, often excessive exacerbations, tenderness in the nerve-trunks,

sensitiveness in the pertinent muscles. We must also look for sensible and motoric disorders in the affected nervous area, changes in the tendon reflexes, muscular atrophy and fibrillary twitchings as well as electric, trophic and vasomotoric reactions. Nevertheless, there are cases in which some if not all of the aforementioned symptoms are wanting, but then the nerve itself will very likely make itself known as the irritating cause of the pain.

If the *pain is unilateral* its originating cause is to be found in a nerve-trunk of the extremities. It then either occupies the whole bronchial territory or is confined only to a certain area of the plexus, most frequently in the distal parts of the extremity.

Apart from the primary diseases of the nerve-trunks, insofar as the lower extremities are concerned, we must also consider as irritating causes of pain all morbid processes of the true pelvis, of the retroperitoneal space and of all organs that for morbid reasons may in any way encroach on this cavity which is so to speak a common meeting place of all sorts of pain. It is rather of interest to notice that even in affections of azygous median organs in this region unilateral pains in the extremities may exist. For instance, pain in the right leg makes us think of prostatic carcinoma. Other accompanying symptoms will help us to find the right diagnosis,

for instance: parallel irradiation of the pains into the abdomen and genitals, simultaneous psoas position as a sign of renal or pararenal (paranephritic) inflammation or suppuration.

Of equal importance is the fact that similar, rather light pains in the anterior side of the thigh, more distinct, however, in the right foot, combined with abdominal and sacral pains with fever or without, with vomiting or otherwise, are an indication of a possible acute periappendicitis, in which the vermiform appendix presses on the psoas muscle and produces an acute initiation of the crural nerves (perineuritis of the crural nerves). In these cases we often miss every symptom of peritonitis, chiefly vomiting, and the stormy onslaught of the disease altogether. The diagnosis is materially advanced by the radiating nature of the pain in the foot and by the fact that in active contraction of the psoas muscle (raising of the foot) the local pressure pain in the ileocecal region is much severer than when the leg is at rest. Palpation per rectum is painful and there is considerable tension of the abdominal muscles. The patient feels the pains in the thigh, but the abdominal pains do not seem to incommode him to any extent. That the abdominal pains are either provoked or sharpened by movements of the hip joint appears to me to be an important sign of acute or chronic periappendicitis.

These pains in the anterior side of the thigh are likewise a valuable symptom of renal diseases (tuberculosis, neoplasm, hydronephrosis) and in other *retroperitoneal* affections. Still, we must not lay too much stress upon this sign, because there are cases of *intraperitoneal* tumors which extend so far rearwards that they, too, may give rise to retroperitoneal pains.

In *paroximal hemoglobinuria* pains are experienced in the lower extremities during the attack, but they are superseded by those in the loins and in the region of the spleen and liver.

Pains—and casually paresthesia—on the inner side of the thigh down to the knee together with signs of sudden incarceration are the strongest hints of an existing *hernia obturatoria.* Distinct painfulness on deep pressure against the foramen obturatorium and a slight arching in the region of the oval perforation confirm the diagnosis.

A unilateral pain in the *upper* extremity introduced or accompanied by paresthesia warns of an expanding morbid process in the *mediastinum.* In my own opinion, which, however, is not shared by all authorities, pains in the right —very seldom in the left—arm (and shoulder) point to an aneurysm of the ascending aorta or its arc, likewise to solid or cystic, less frequently chronic inflammatory tumors of the mediastinum, especially of the glands therein (lymphogranu-

lomatosis). According to their anatomical localization these tumors produce pains in one of the arms, but also in both. These pains and possible parenthesias are at times the sole symptom of the prevailing disease and as such are of signal merit for the diagnosis.

Only a few cases are on record in which acute processes in the mediastinum provoked pains in an arm, but not also at the same time in the chest or back. I do not speak here of pericarditis when the pains radiate into the left arm as is their wont in angina pectoris.

These pains in the extremities have a special, characteristic habit of setting in when the patient is lying on his back but to vanish when he bends over forwards. The same happens also in aneurysms of the aorta and solid tumors of the mediastinum. In the prone position the aneurysm or tumor tears and presses on the adjacent nerves and thus exacerbates the pains, which, however, may also be sharpened by physical overexertion.

Tumors of the *superior pulmonary lobes* and acute as well as chronic inflammatory diseases thereof, e.g., of the pleural apex give rise to analogous irritation of the cervico-brachial plexus in the supraclavicular region and hence to neurogenous pains in the arm. They must always remind us of lobar pneumonia (with pleurisy of the apices and perineuritis of the cervical

plexus) or of acute apical pleurisy, or chronic, retracting lobar indurations of tuberculous genesis or otherwise, also of tumors in the same locality, i.e., the pleura. Mark this also: remittent-interremittent pains in one arm are the first signs of carcinoma in the upper pulmonary lobe or its pleural apex, or in the main bronchus.

It is, perhaps, easier to understand that an analogous irritation of the aforesaid plexus with subsequent pains in the arm, paresthesia, likewise hyperesthesia, weakness, paresis, apoplexy, may also emanate from any expanding or inflammatory morbid condition in the supraclavicular fossa (diseases of the lymphatic glands, of the clavicle, aneurysm of the subclavian artery or of the anonyma, etc.). The diagnosis comes easy because the disease is open to our senses, we can see it, we can feel it. Of course we cannot look upon this—if we except metastatic tumors (glands)—as a definite proof that the place of origin of the tumor and of the subsequent pains in the arm was from the very start localized in the supraclavicular fossa. There are, indeed, cases in which the patient complains of pains in the arm but not a trace of tumor in the supraclavicular fossa can be found in the beginning, although it puts in appearance later on: in other words the original mediastinal malign tumor has forced its way into the supraclavicular fossa.

If we overlook this point, if the anamnesis is incomplete and we have neglected to make a scrupulous examination of the whole chest—not to forget the X-ray—we may readily meet with bitter disappointments.

Palpation and perception of the supraclavicular fossa at times yield absolutely negative results. A cervical rib, for instance, may escape our observation for a considerable time, although it be the sole cause of unilateral or dual pains in the upper extremities. They are produced by motation, or under the influence of cold, ushered in by paresthesia, associated with motoric, trophic and secretory troubles, sensibility disturbances (i.e., typical neuritic disorders), high pressure in the subclavian artery, with whirring, hissing, blowing systolic noises above it. Only the most thorough palpation and the X-ray are able to discover the true source of the pains. What has been said may also be applied to *malignant struma.*

The aforesaid intrathoracic diseases require equally our attention where the pains in an arm are the reflex action of pains in the chest, but if they are reflected from pains in the abdomen (epigastrium, hypochondrium) they indicate subdiaphragmatic irregularities and go in company with pains in the shoulder. Witness cholelithiasis, perihepatitis and perisplenitis, gastric diseases (ulcer, carcinoma, perigastritis) and

diseases of the pancreas. In some cases the pains may constitute, when of a neuralgiform character, the first symptom, e.g., of ulcus ventriculi in the left brachial plexus.

Unilateral neurogenous pain localized centrally from the process of the extremity is not only due to an affection of the nerve trunk beyond the extremity but may just as well originate from a morbid condition in the *intraspinal nerve roots.* Of course, analogous painfulness in the contralateral extremity matures during the course of the disease. The bilateral and symmetrical nature of the pains constitutes the characteristic sign of these lesions of the nerve roots because their fibres lie so close together. Nevertheless, a regional disease can just as well have a unilateral influence on the nerve roots.

The question arises here how to differentiate between affections of the roots and those of the nerve-trunks. In the first place it is important to note that in fascicular (trunk) affections objective pressure sensibility is present in the entire nerve trunk (I mean isolated pressure points) which in affections of the roots are either totally wanting or of a negligible quantity, while in their place pressure points in the spinous processes are to be found. Secondly, the pains have a radicular character, that is to say they are as in tabes very keen lancinating pains provoked by motation or overexertion, hyperactivity

of the abdominal press, e.g., when sneezing. In the third place in radicular neuritis not the whole plexus, but only one or several branches are involved. Fourthly in radicular neuritis objective sensibility disturbances are of frequent occurrence while in truncal neuritis they are inconstant. And lastly in radicular neuritis the objective sensibility disturbances run in the spinal-segmental type, i.e., generally at the extremities in horizontal but in the thorax in circular patches, whilst in truncal neuritis they follow the peripheral type, i.e., the direction of the peripheral nerves.

Unilateral pain in the upper or lower extremities or in both or only in a section of one points to a possible cerebral genesis. I do not refer here to the sensible aura of epilepsy or to hemiplegia, but rather to those very molesting and severe attacks of exacerbating neuralgiform pains which manifest themselves in hemiplegic extremities, especially the arms. Sometimes they accompany hemiplegia, at other times they follow in the wake of it or arrive before the attack sets in in the form of premonitory pains owing to irritation of the intracerebral sensible course through the central focus (focus of hemorrhages or softening, cysts, tumors) or arising from the meninges. Paresthesias associated with these eccentric pains originate from certain centers in the cortex, or in the vicinity of the thalamus

opticus, or in the pons or the cerebellum. We can localize the cerebral seat of the pain by the fact that other cerebral manifestations accompany it, i.e., hemiparesis, hemiplegia, hemispasms, hemiclonus, hemiathetosis or hemianesthesia in the opposite side (hemianesthesia dolorosa—thalamus affection).

In some rare cases we have to deal with sensible manifestations of this kind *only*. Then the diagnosis will be guided either by changes in the tendon or skin reflexes or of the deep sensibility (stereognostic sense), or by other cerebral local or generalized symptoms, or perhaps solely by the anamnesis in the sense of a preceding apoplectiform insult (in a thalamus focus not recognizable!). These pains of cerebral origin in hemiplegic extremities are unfortunately too often taken for rheumatic pains because they are like those lancinating pains in diseases of the spinal cord so much influenced by weather conditions. I mention here that the same characteristics attach also to pains which are based on anatominal lesions (stenocardia) or on functional disorders (neuroses).

Of interest are also the attacks of pain in an arm or leg attended with high fever and hyperalgesia which come on sporadically in *progressive paralysis* and disappear again within a few hours. They are undoubtedly of central origin and will be properly understood if we take into

consideration the other psychic and somatic signs of the disease as well as the anamnesis, and apply the *Wassermann* reaction and lumbar puncture (three tests).

It is easy to understand that in *neuritis* the pains are present in the affected nerve-trunks in both sides and frequently assume the rôle of symptomatic manifestations of the causating disease, e.g., chromic alcoholism or leprosy, morbid conditions in the true pelvis or in the retroperitoneal cavity. So, too, a bilateral ischias is a valuable indication of prostatic carcinoma or multiple tumors in the pelvis, if not of diabetes, malaria or gouty diathesis or chronic constipation. Pains in both the lower extremities and in the hip joints, particularly in women, are the first sign of tuberculous peritonitis.

Radicular neuritis and *spinal disorders* are further irritating factors of bilateral pains either in the upper or in the lower extremities. I refer to tumors, tuberculosis, gummata of the vertebræ, compression of the intraspinal ganglia, morbid conditions in the meninges and intramedullary diseases. Likewise to multiple sclerosis, syringomyelia, myelitis, cerebro-spinal syphilis, tumors of the spinal cord, acute poliomyelitis, chronic meningitis, epidemic cerebrospinal meningitis, tabes superior, pachymeningitis (chronic hemorrhagic, syphilitic or tuberculous), hemorrhages in the spinal cord (trau-

matic), in hemorrhagic diathesis, scarlet fever, scurvy, pernicious anemia, and tumors of the spinal cord (extramedullary).

Also leucemic or lymphoid infiltrations in the epidural tissue of the lower vertebral canal deserve mention here. In vertebral insufficiency it will be noted that pains in the spinal column or in the back are of rare occurrence, as they predominate in the chest and abdomen, legs and arms. If we have proof of sensitiveness on pressure and percussion in a definite zone of the spinous processes, if we find spasms in the dorsal muscles, collapse of the spinal column or deforming curvature of the body with relaxation of these symptoms when the patient is resting, we should have no difficulty in forming a satisfactory diagnosis.

I include those extremity crises in tabes dorsalis in which the patient complains of violent, dragging and tearing pains with subsequent cramps in the calves and local hyperesthesia. Likewise those pains in the lower extremities which follow the interspinal injection of a cold fluid not properly warmed up, and also those very keen pulling pains in the arms and legs of which we see so much in epidemic encephalitis—the latter, however, may be also due to purely cerebral influences.

Pellagra generally sets in with pains and paresthesia in the extremities, no matter whether it

affects the spinal cord or the peripheral nervous system.

In *paraplegia dolorosa* we witness intensive pains in the back which radiate thence into the paralyzed, debilitated extremities. *Carcinoma of the spinal column* is the most pronounced characteristic of this disease, a fact which accounts for the violent pains mentioned just now.

In the diseases heretofore discussed the pains are wont to sneak in gradually and increase in intensity more or less rapidly. But there are other chronic affections of the spinal cord and its membranous envelope as well as of the osseous parts which introduce themselves with a very sudden, apoplectiform initial pain in one if not in both corresponding extremities. I am not referring, however, to tuberculous spondilitis, but rather to extramedullary *tumors of the spinal cord* which after a slow latent growth suddenly spring into evidence with a stroke-like terrible pain in the extremities. Their invasion which is accompanied by a feeling as if the spinal cord were severed in two must arouse in us at once the suspicion of *hematomyelia.* Although this disease is in itself pregnant with pains, still it is wise even here to remember that the greater the intensity of pain, the stronger is the probability of a membranous involvement of the spinal cord.

A hemorrhage as well as a sudden vascular

occlusion of the spinal cord leads to very severe pains in the lower extremities with a quick local relaxation in the power of motion and with sensibility disturbances reaching even into the hypochondrial region, witness certain cases of *dissecting aneurysm of the thoracic aorta* with hemorrhage into the aortic tube cutting the circulation in a portion of the intercostal arteries.

An ordinary aneurysm of the descending thoracic aorta (also of the abdominal aorta) is in the same manner liable to produce paresthesia and pain in the lower extremities through spinal influence, i.e., when a vertebra is worn down by friction and an opening into its canal is formed which naturally results in pressure on the spinal cord. The superceding symptoms of the transsection of the spinal cord (backache, paraparesis or paraplegia of the legs, gastric and bladder troubles, etc.) should put the diagnosis on a sound foundation. To find the cause for it in an aneurysm of the aorta may be difficult at times, but the difference in the arterial pulses of the upper and lower extremities and the X-ray are reliable guides in that direction.

Unilateral and why not bilateral pain in the nerves of the extremities, however, are not only dependent on neuralgia or apparently neuralgic conditions, but may just as well originate from any common cause.

Certain *infectious diseases,* such as malaria,

syphilis, gonorrhea, tuberculosis, typhoid, influenza, etc., are creators of neuralgia in its divers forms. Exogenous and endogenous *intoxications* are frequently at the bottom, such as diabetes mellitus, hyperglycemia, gout, uremia, chronic obstipation, alcohol, lead, arsenic, carbon dioxid poisoning, in fact all disorders of metabolism.

For the sake of substantiating this claim I will pick out as an example ischias. When the patient complains of such a pain in the ischiadic plexus we must be conscious of the fact that it is due to a lesion either of the corresponding nerve-roots or of the nerve-trunks. But we should also remember that there is such a thing as ascending neuritis (though very seldom applicable in ischias); in other words that an infectious disease in the radicular zone of a nerve may lead to an ascending inflammation of the trunk and its branches. From this we are safe to draw the conclusion that in cases of ischias we have before us a long string of possible complications.

There are expanding morbid processes of all kinds in the spinal canal whether they originate from the meninges (pachymeningitis, leptomeningitis, tumors), or from the spinal cord proper or from the cauda equina (tumor); diseases of the nerve-roots in the lumbo-sacral vertebræ and their interarticular ligaments (neoplasms, spondi-

litis, rhizomyelia) or in the intervertebral foranina (inflammatory, neoplastic, leucemic infiltrations); inflammatory or compressing processes in the region of the loins and sacrum, no matter whether they arise from affections of the bones, of the connective tissue, or of retroperitoneal lymphatic glands or organs (kidneys, adrenals); analogous morbid conditions in the pelvic area (pregnant uterus, tumors, inflammations, diseases of the ovaries, of the prostate, chronic constipation); inflammatory and constricting processes in the lower extremities; diabetes, gouty diathesis, malaria, etc.

Local traumata, diseases or infiltrations, also sclerosis of the arteries (arteriosclerotic neuritis), varices of the venous nerves, all these conditions may lead to ischias. We must also differentiate between true ischias and neurotic (hysterical) pseudoischias. So far as the latter is concerned we must fall back on the typical characteristics of hysteria, the fluctuating localization of the pains, the fact that a gentle pinching of the skin causes severer pain than hard, deep pressure and that the pains subside under the influence of mental distraction. The diagnosis finds further subsidy from the behavior of the tendon reflexes (decrease of the achilles tendon reflexes in true neuritic ischias), from the special characteristics of the pains, the pressure points, *Lasegue's* symptom, increase through the ab-

dominal press, coldness of the knees and last but not least, from the Roentgen ray.

IV. Other Pains in the Extremities

All these symptoms must be carefully considered whenever we hear complaints of pain in the small of the back and in the breast and the diagnosis balances between ischias and lumbago. Incidentally, these two diseases may run a parallel or alternating course, unless lumbago is in itself a neuritis of the ischiadic branches.

Slight, vague pains which are hard to localize in the muscles and which radiate into the bones and joints consisting more of a painful dragging, but acute under the influence of general indisposition and mostly attended with fever are a clear indication of an *acute infection.* Every form of infection, every kind of vaccination may begin with these sensations. But in some they are more pronounced than in others, the commonest among them being the ordinary nasal catarrh (coryza, snuffles) and influenza (grippe). (Cf. chapter on "Muscular Pains.") In smallpox, recurrent fever (pains in the bones, joints and muscles, especially in the legs), erysipelas (most intensive muscular and nervous pains), but also in influenza these pains are very severe and of a boring, tearing character not only in the incipient stages, but throughout the run of the infection and sometimes far beyond that.

In typhoid and paratyphoid this is not so often observed. Pappataci fever has articular pains, and glanders pains in the shinbones as companions.

In *spotted typhus* paresthesia and pains in the extremities are not only the expression of the generalized infection, but may also be the precursory sign of gangrene characteristic of this disease. I have already said previously that gangrene of the extremities follows also other acute infections such as typhoid, influenza, etc. In all these diseases, especially in exanthemic fever the pains endure at times for weeks without subsequent gangrene. They are evidently due to specific attacks of arteritis.

Of *Wolhynian* fever another causative factor of these pains I have spoken already in several places.

Pains in the extremities which come to the fore in septic diseases must remind us of metastatic conditions in the bones or in the muscles such as abscess or necrotic foci.

Periarteritis nodosa is often surrounded by symptoms which strongly resemble a septic affection. There are pains in the extremities which are evidently due to morbid changes in the arterial walls. We also find irregular attacks of fever, anemia, physical debility, tachycardia, leucocytosis, polynucleosis, edema and nephritis, articular swellings, cutaneous hemor-

rhages, cyanosis, dyspnea and abdominal pains. The diagnosis (cf. "Abdominal Pains") can only be secured when we can palpate the enlarged local arterial walls. Syphilis seems to be the originating cause of this disease in some, but by no means in all cases.

Pains in the extremities are not only initial and concomitant symptoms, but may also be the sequel of infectious diseases, i.e., signs of convalescence, as it were. The patient is run down and feels as if he had been beaten up after a severe attack of influenza, for instance, or smallpox. The same may happen even after slight infections, such as *dysentery* which was taken for simple diarrhea, a short acute attack of *gastroenteritis.* The toxic after-effects retard recovery and bring fatigue and pain with them.

In mild forms of *trichinosis* pains in the limbs are not uncommon although they do not bear the stamp of characteristic significance. Yet when they are associated with pains in the muscles of the neck and chest and with gastrointestinal troubles, they should be of assistance in finding the right solution, especially when we detect an edematous lesion in the eyelid and eosinophilia in the blood.

This applies also to mitigated forms of *acute articular* and *muscular rheumatism.* Here we get a hold on the diagnosis when local pressure sensitiveness and heavy sweating are present.

The effect of salicylic treatment is another adjuvant.

Complaint of mild pains—sometimes very perturbing, boring, tearing—especially in the tibia is an introduction to *scurvy.*

In metabolic infections the patient is apt to localize the pains in the joints and muscles of the extremities. Upon closer scrutiny we find that they rather affect the tendons and fascia, also the nerves and synovial sacs. I am referring to *gout,* but not to the typical acute, nor the irregular, but to that form which we know as gouty diathesis or a typical irregular gout not dependent on previous gouty articular affections. The pains are of a transient nature and wander about from one place to another. Clinically speaking there are no typical signs of gout and the diagnosis must be in most cases merely a good guess, justifiable, indeed, in stout persons with a familial gouty record, persons who indulge in sumptuous living, meat eaters, persons who eschew green vegetables and fruit, "bon vivants." They generally suffer from abdominal plethora, hemorrhoids, enlarged liver, skin diseases, such as chronic eczema, also from urticaria and chronic dyspeptic troubles. We find neurasthenic symptoms such as headache, dizziness, migraine, abnormal irritability, chronic lassitude, etc., without a specific causal element. Renal calculus is common in these patients. The

diagnosis derives much benefit from a proper urinary analysis and tentative drug treatment. According to my own very wide experience this disease is very common among city-dwellers who eat too much and do not take sufficient physical exercise and are hereditarily predisposed to such attacks. It is very difficult to properly localize the pains as they vary so much in different individuals and even in the same person as to time of appearance. Yet in the majority of cases we shall be able to spot a typical neuralgia (e.g., ischias), pseudorheumatic muscular and articular pains.

Tarsalgia must be mentioned on account of its close connection with gout and diabetes mellitus. The patient complains of dull, though sometimes very severe pains in the heel of the foot, especially in the lower plane thereof, corresponding with the process of the achilles tendon. Sometimes the outer margin of the heel is very sore to the touch. The pathologic anatomical causes for these pains (achillodynia) are quite a few. I mention affections of the achilles tendon (peritendinitis achillea, recognized by a peculiar friction fremitus in the movement of the tendon), enlarged synovial sacs which can be felt with the finger, morbid condition in the calcaneum or in the peripheral nerves (e.g., diabetic neuralgia or ischias), inflammation of the local adipose tissue. Some authors claim

that peripheral arteriosclerosis or a similar affection of the arteries of the bones is a causative factor as can be demonstrated by the aid of the Roentgen-ray. Tarsalgia as a rule is bilateral with pains in the median knuckle or corresponding with the cuboid bone. If dealing with a traumatic affection of the synovial sac, or an exostosis of the calcaneum (calcaneous spur) (Roentgen picture!) it is of importance for the internist to ascertain whether the cause is not to be found in flat foot, or gout or gonorrhea, or else in rheumatic conditions. The gonorrhoic form is distinguished by the fact that the pains are not only centered in the heel but involve also the sole of the foot (1. to 5. metatarsophalangeal joint).

But a *calcaneous spur* may also be congenital, or due to syphilis, a trauma, flat foot or osteoarthritis. Bear also in mind that pes planus and a calcaneous spur may co-exist.

Pains in the foot are also due to affections of the plantar aponeurosis, viz., fasciitis plantaria aponeurotica, due to gout, gouty diathesis, hence due to the presence of urates in the plantar fascia. The etiologic diagnosis is based on the characteristics of gouty conditions. It is also claimed that rheumatic indurations in the plantar fascia give rise to local pains.

Acute fasciitis is also among the sequels of acute infections, especially of influenza, and may

be recognized by radiating pains and tenderness in and around the aponeurosis. From my own esperience I cannot state with certainty but it seems possible that the pains in the feet observed in typhoid fever are due to a similar cause, if not more correctly to neuralgic influences.

Dysbasia angiosclerotica with symptoms similar to those here discussed may be observed in inveterate cigarette smokers.

In *metatarsalgia,* i.e., *Morton's* disease, a peculiar kind of pain in the feet occurs. It is centered in the metatarso-phalangeal joint, mostly of the 4. toe, radiates upwards and becomes so distressing in walking (narrow shoes the likely cause!) that the patient must stand still and rest. When the footwear has been removed and the painful parts are massaged, the pains generally disappear. Local reddening and swelling are seldom observed and I am not prepared to state in how far anatomical changes in the joint, or anomalies in the fascia or pressure on the ramus communicans by the head of the 4. metatarsus are the responsible factors. But I think that X-ray examinations should be made freely. If gout be the irritating cause it will be shown by local reddening and swelling. The differentiation from pes planus lies in the narrow limitation of the pains; and the presence of pulsation in the pedal arteries separates it easily from dysbasia angiosclerotica.

When the patient complains of pains in the soles of the feet whilst walking or standing, with local tenderness now in the heel, then in some other part of the sole, we must think of *neurasthenic* and *hysteric podalgia.* The diagnosis offers some difficulties even if we observe signs of neurasthenia and hysteria and no possible anatomic causes can be found. At times it is made positive only when the pains suddenly vanish.

But if the pains are vague in character and cannot be localized with ease, *neurasthenia* is the most likely causal factor. However, in neurasthenia the pains in the extremities are very rarely of *great intensity.* As a rule, the patient complains chiefly of headache, pains in the small of the back and in the chest, but rather of a painful feeling of fatigue in the limbs, burning neurasthenic dysbasia and podalgia. But what I consider the distinguishing features of this painful weariness, consists in the fact that the patient feels more jaded in the morning than at eventide, broken up, so to speak, all over the body, that the pains are not sharpened by local pressure, that they eventually are mitigated by moderate physical and mental activity (a nice walk in the open air), that we cannot find an essential reduction of the rude motoric power, though the patient tires out much quicker, and finally that we can always discover the typical

neurasthenic stigmata, foremost among them abnormal psychic and mental conditions.

In *nicotinism* and a good many diseases of the internal secretory glands (*Addison's* disease, *myxœdema*) pains in the extremities are linked with initial symptoms.

A similar painful sensation in the muscles of the extremities is often experienced by *epileptics* in the stadium between the fits, but then these unfortunates suffer from all kinds of neurasthenic symptoms during that period.

In *hysteria* (traumatic hysteria as well) the incessant complaint of pain is the ground pillar of the symptomatic structure. So far as the extremities are concerned, we must distinguish between two subdivisions, viz., tropalgia to which arthralgia and pains in the periosteum belong (cf. chapters on "Articular Pain" and "Pains in the Bones"), and secondly neuralgiform pains, rather rare, however. The diagnosis takes root in the fact that these pains are attended by distinct cutaneous hyperesthesia of a circulary character and nearly always by pains in other parts of the anatomy, e.g., in the mammæ, pit of the stomach or in the back and also by the other manifestations of hysteria.

In *pulmonary emphysema* the patient always suffers from "rheumatic" pains in the leg near the ankle-joint. In some instances there is no apparent connection between these pains and the

existing emphysema, for instance, in a case of concurrent alcoholism, when a closer examination divulges alcoholic neuritis. In other patients the pain rests in the bones, especially in the tibia above the knuckle with strongly localized tenderness. It is a sort of miniature edition of osteoarthropathie pneumique caused by chronic bronchitis and dilatation of the bronchi, so common in emphysema.

If we find dragging pains in the extremities and the patient cannot tell whether they are in the bones, muscles or joints we must think of "growing pains" in youthful individuals. If there are no traces of organic defects the diagnosis is patent, even if dyspeptic troubles and slightly febrile conditions might tempt us to think of another disease.

I append here a diagnostic observation of some value. We come across patients sometimes who cannot localize a pain in the leg which comes on after standing or walking for some time, possibly due to *overtaxing the limb*. It is advisable in such cases to make a careful search for a morbid process in the contralateral extremity. We may find muscular atrophy or an abnormal shortening of this extremity. Likewise examine the spine for deformities or other defects. The cause may also lie in occupational overexertion.

Extraordinary *forced marches* reveal many

such cases among troops. The tibia, the periostium and the processes of the muscles are here the seat of the pains. We find similar conditions in porters who carry heavy loads, and in very fat people.

Among the various pains in the extremities those in the bones receive, as a rule, the least attention, especially when they are of an unsteady nature, are changeable as to time and localization, and morbid changes in the bones which are not palpable. I refer the reader to the different chapters on "Pains in the Bones, Muscles, Joints."

Of course, it goes without saying that pains in the extremities may be due to several parallel causes. I have seen cases in which arthritis deformans coxæ dextræ, ischias, pes planus and varicose veins were all bunched together in one and the same carrier.

In some rare cases pains in bilateral extremities may derive their origin from a *bilateral cerebral lesion.* Just the same they may also proceed from a single morbid focus, e.g., a tumor in the pons may provoke paresthesia (also anesthesia) in the extremities on both sides of the body, and corresponding pains, too; or else in the arms only or in the legs or in three extremities with analogous disturbances in both sides of the face.

A special subspecies of pains in the extremi-

ties, mostly bilateral, are the so-called *lancinating pains*. They are intermittent in character, come on very suddenly, and penetrate the deeper muscles like a shot. The patient generally speaks of them as very severe rheumatic pains. At times they arrive in the form of a shock, moving the affected limb with an abrupt convulsive jerk. When this happens we should look for some lesion of the spinal nerve-roots, but if the pains recur with unusual frequency they are a possible warning of *tabes dorsalis*. Look for divers missing or diminished patellar with increased abdominal reflexes, absence of achilles tendon and triceps reflexes. We shall very likely find abnormally small pupils which are painful under the influence of light, constant accommodation and convergence reaction, *Bamberg's* phenomenon and disturbances of superficial and deep sensibility. From the observance of ataxia, of bladder and rectal disorders, changes in the innervation of the exterior ophthalmic muscles, and in the fundus of the eye, together with the aforesaid symptoms, the proper diagnosis will easily crystalize. When the lancinating pains are the first apparent sign of the disease the situation may be more intricate. A thorough probing for ataxia in the lower extremities is then indicated (especially in athletes, tourists, and persons who are given to violent bodily exercise). The tendon reflexes should

receive careful consideration and also the question of previous syphilitic infection. Globulin reaction, lymphocytosis of the cerebrospinal fluid and the *Wassermann* reaction are further adjuvants in the diagnosis.

Errors are, perhaps, more frequent when these lancinating pains enter the arms or only one arm at a time. Still, the symptoms described just now should suffice to promptly establish the existence of a *superior* or *cervical tabes.*

However, not only tabes or taboparalysis, but all encroaching diseases of the vertebræ and their marrow, are apt to irritate the intraspinal nerve roots to such an extent that lancinating pains are the result. In *gout,* a deposit of urates in the dura mater and in the nerve roots indubitably has this effect.

All morbid intraspinal processes, such as syringomyelia, myelitis, multiple sclerosis, tumors of the spinal cord as well as peripheral forms of neuritis, are possible causes of lancinating pains. Apparently few mistakes occur in the diagnosis when the lower extremities are concerned. But I must admit that when the arms are attacked by these lancinating pains, the diagnosis generally goes wrong, i.e., in the direction of rheumatism, gout, neuralgia, etc. The obvious reason is that this form of pain is affected in a similar fashion, as in rheumatism by weather conditions, thermic influences, and sudden

changes in the atmospheric temperature. The diagnosis should be made from the most careful consideration of the characteristic tabetic signs, especially in the eyes, and proper thought must be given to other diseases that are likely to affect the spinal cord. I have seen quite a number of cases of multiple insular sclerosis in which neuralgiform, lancinating pains in the upper extremities endured for years; in fact, were the sole perceptible morbid symptom. Intention tremor, irregularities in the vocal mechanism, *Babinski's* toe phenomenon, abdominal wall reflexes missing on one side and increased tendon and periosteal reflexes should be decided aids in diagnosing such cases generally observed in persons of youthful age.

In *caisson diseases,* lancinating pains in the extremities are prominent among the initial symptoms.

They are likewise the most significant concomitant manifestation of *peripheral neuritis.* In *diabetes mellitus* they appear in the lower as well as in the upper extremities, and prevail likewise in *nicotinism.*

It is these lancinating pains, combined with missing patellar reflexes, eventual ataxia and paralysis of the eye muscles which make diabetes mellitus resemble tabes dorsalis in so many cases when glycosuria attends the latter.

Lumbar puncture is an important factor here.

Of course, we must not forget that both diseases may also exist alongside of each other. But there is one point which does not seem to have received adequate attention in medical circles. I mean the fact that lancinating pains of great intensity in the extremities, combined with analogous shooting pains in the breech and in the chest, are not uncommonly a warning sign of an impending coma in diabetes, despite the fact that the patient has not previously experienced similar, not even neuritic (neuralgic) pains.

Peripheral pseudotabes, i.e., chronic alcoholism, ergotism and other intoxications, are likewise associated with lancinating pains in the extremities. To this list must be added *aneurysm of the abdominal aorta, abdominal* and *pelvic* and *intraspinal tumors,* especially when the pains occupy only one side of the body. Nevertheless, there are cases of tabes dorsalis on record in which continuous unilateral lancinating pains were observed.

In *acromegaly,* in *hypophysis tumors,* they are initial symptoms. Owing to their neuralgiform character, to the loss of vision, to diminished or wanting patellar reflexes, the erroneous diagnosis of tabes dorsalis or progressive paralysis is apt to be made. It is probable that a secondary degeneration of the posterior column of the spinal cord is the causative factor of these manifestations. The usual symptoms of acromegaly

should govern the diagnosis in that direction. Polyuria, signs of adipose genital degeneration, hemianopsia and hypophyseal tumors are possibilities to be reckoned with. The X-ray offers good opportunities.

Exceptionally, *tumors of the brain* exercise an irritating influence on the cerebro-sensible centers, e.g., in the thalamus opticus, especially in the pulvinar. I have in mind a tumor of the perineal body. Lancinating pains in these affections are common.

Tonic spastic conditions in the muscles are correlated with pains in the extremities, and may be part of the symptoms of intermittent *dyspragia,* as they are the essential element of *occupational cramps.*

Muscular cramps, especially in the calves during walking, belong to *polyneuritis,* unless they are caused by muscular sural rheumatism. The diagnosis of all these affections should easily result from the pertinent typical symptoms described in other pages of this book. I include here also all forms of *tetany* comprising *hysterical pseudotetany.*

When we encounter painful muscular cramps which resemble tetany we must look for some anatomical lesion, primarily *tetanus.* Here we may find at first only twitchings and muscular spasms (aura tetanica) in the extremities which are strictly localized and associated with drag-

ging or tearing, sudden, violent pains. The differentiation between convulsions in tetanus and tetany is based upon the fact that in tetanus the cramps affect principally the masseter (slight degrees of trismus!), also the nuchal muscles opisthotonus), which is not the case in tetany; further, that tetanus is caused by a wound which in many cases has been overlooked by the patient, not to speak of tetanus infections in the uterine cavity, in the air passages or in the intestines. Moreover, in tetanus the characteristic generalized muscular spasms and other typical signs (fever, although not uncommon also in tetany, heavy perspiration, eosinophilia of the blood) soon manifest themselves.

There are, however, certain rudimentary forms of tetanus in which a painful spastic stiffening of the muscles is the only perceptible symptom.

If we fail to find the cause for the tetanus infection (men working in the soil or stable, or wound in the body), if the animal test with the blood, fecal matter or bronchial secretion of the patient yields negative results, the diagnosis may be hard to make, and we may have to fall back upon other means for assistance.

There is a third form of painful muscular spasms in tetanus which must be mentioned here, i.e., they are definitely and permanently confined to an individual area of the body, mainly to one

of the extremities, and thus constitute the cardinal symptom of "local tetanus."

Painful muscular spasms resembling tetany are likewise found in a large number of exogenous, acute intoxications, and are always combined with other cerebral symptoms (arsenic, phosphorus, saltpeter, filix mas, etc., poisonings). The differential diagnosis is self-evident, and need not be discussed here.

Nocturnal cramps in the calves, in the *soles of the feet* and in the *abductor hallucis* concern the physician only from the etiologic standpoint.

A sudden attack of unilateral cramps in the calf may be the result of a muscular rupture; if recurrent, due to an induration in the muscles as a sequel of the primary rupture.

The causes for these spasms are either local or general. Nocturnal bilateral cramps call for urinary analysis. They may be symptomatic of diabetes mellitus or of uremia. In gout, they are accompanying symptoms sounding a warning note of an impending severe painful attack, or they act as an expression of chronic diathesis. Cramps in other muscles, e.g., of the thigh, arm, back or abdomen, often attend the sural spasms in gout. I have spoken of the presence of uratic crystals, hyperuricemia and uricemia, etc., in connection with this disease in another place to which I refer the reader.

Ectogenous poisons (alcohol), abuse of to-

bacco and physical overexertion (late hours) are other exciting factors, to which may be added gastralgia, cold, wet feet and spastic constipation, affections of the peripheral nerves, of the pyramidal tracts (multiple sclerosis, spastic spinal paralysis).

Anhydremia is often attended with muscular pains in the upper extremities—a valuable symptom in asiatic cholera and cholera nostras. These cramps in the calves are also observed in other diseases which run a course similar to cholera, such as paratyphoid, dysentery, malaria or fungus poisoning.

In *gastrosuccorhea* they come in with the periodical gastric attacks, but whether they are due here to toxic influences or to abnormal thickening of the blood is questionable. There are a good many other toxic conditions which assume the character of cholera and in which tonic spasms radiate from the sural region over the whole muscular system, such as arsenic, chrome, saltpeter, ptomain poisoning, and botulism.

Painful cramps in the calves are also due to flat foot and osteomalacia, to defective circulation in the veins, e.g., phlebectasia, phlebitis and phlebothrombosis. They also originate from ischias or polyneuritis, or constitute the solitary symptom of an initial compression of the ischiadic nerve. Perhaps this, as well as congested

circulation, are the provocative element of cramps in the calves of pregnant women.

Sometimes they are caused by stretching the feet, and may then be a sign of latent tetany or an accompanying symptom of tabetic crises in the extremities. But they may also be directly due to a disease of the sural muscles, for instance to a cysticercus or to trichinæ. If the former is calcified, the Roentgen picture will show its presence. Subacute and subchronic periarteritis nodosa must be mentioned also.

If in perfectly normal and healthy persons these cramps are sometimes observed, we shall very likely find that they are due to overexertion or overfatigue, e.g., after a long march or walk, climbing high stairs or mountains, riding on horseback, athletic exercises etc. The diagnosis should offer no difficulty.

The so-called cramp neurosis gives rise to such pains in the calves. I have had no opportunity to observe cases of this kind but it seems to me that the only causative element is a peculiar predisposition to this ailment.

A retrospect of the foregoing pages will tell us that pains and paresthesias in the extremities are to a great extent manifestations of the same diseases; the paresthesias, in fact, often forerunners, if not substitutes of the pains. We also have learned that these pains may have a totally different meaning in one and the same

disease. For instance, when the patient complains of such pains during or after an attack an acute infection, especially during the puerperal stage, the differential diagnosis revolves around *phlebitis* and acute *neuritis*. Muscular tenderness is common to both, but tenderness in the nerve trunks, subjective paresthesias, subjective and objective sensibility disorders, primarily increased, later diminished, tendon reflexes, temporary muscular paresis, palpable nodes in the nerve trunks, assure us of neuritis, whilst infectious or post-infectious phlebitis is clearly indicated when the veins feel like cords and cyanosis and edema of the skin are found in the region along the whole venous trunkline.

I wish to say something here about a mistake which I made once, at any rate for several hours. In a case that was brought to me, I diagnosed acute hysteria, whilst in reality it turned out to be acute *thrombophlebitis* of one of the lower extremities, with resultant hysterical conditions. My diagnosis would have been more reserved, if not correct, had I given more mature thought to the fact that every form of anatomical disease in an hysterical individual is apt to awaken a slumbering neuritis.

Before I conclude this chapter, I wish to state that flat foot may also be the cause of local pain in the foot itself. I mention it be-

cause it has been my experience that it is very frequently overlooked in the diagnosis. I have in a previous place stated that pains caused by pes planus generally disappear when the feet are resting, but this is only the case when there are no local inflammatory conditions which so frequently affect the ankle joint.

It is not uncommon that persons with flat feet complain of painful muscular cramps in the soles of the feet after standing for a longer time, which also appear in the plantar flexions of the toes, attended with local paresthesias. Perhaps a secondary neuritis of the plantar nerve is at fault here.

A long list of morbid processes in the feet and hands which leads to local pains belongs in the field of surgery. I refer to luxations, subluxations, caries, etc., etc. But I think it proper to mention *Morton's* disease and also the fact that there is an individual form of neuritis of the hands and feet. A careful examination of the fascia and ligaments should always be made when a patient complains of pains in the soles of the feet, particularly so after the attack of an infectious disease such as influenza, typhoid, etc., because in such cases the irritating cause is frequently to be found in neuritis, plantar phlebitis, fasciitis or ostitis. Pains in the heel or in the knee are often due to neuritic affections of the ischiadic nerve (ischias).

Inflammations or *new growths* in the *tendon sheaths* or in the *synovial sacs* are likewise possibilities not to be neglected. They are often enough, especially when symmetical in form, the result of articular rheumatism, syphilis, tuberculosis, pyemia, scarlatina or smallpox. I remember a case of multiple almost perfectly symmetrical hygroma combined with chronic articular rheumatism which was the effect of diphtheria. Tendovaginitis and hygroma are not only congenital diseases, but just as often the manifestation of some gouty affection or of hydrops hypostrophos.

In conclusion, a word about pains in the *armpit* or in the *groin*. They arise from some primary disease, unless they are merely irradiations of a morbid condition in the nerve trunk. Pains in the groin point to the appendix or some disease of the cecum (if on the left side they direct our attention to the sigmoid flexure); in fact, to any organ in the pelvic cavity. I name retroperitoneal muscular hematoma due to hemophilia. Affections of the male genital organs, particularly of the spermadic cord, of the testicles or of the epididymis, are possibilities here which we should ever bear in mind. Although they belong mainly in the province of surgery, they concern the internist equally as well. I have a case of pendulous abdomen in mind which came under my observation. It

caused the patient (female, otherwise not abnormally stout) very severe pains in the groins. Dietetic and balneotherapeutic measures and a properly adjusted corset removed the cause of the pains in a comparatively short time.

Muscular Pains

Muscular affections are generally indicated when the patient complains of pains in the "fleshy" parts of the body. By rolling and pressing the muscles in the affected part between the fingers we should be able to find local tenderness, and thus localize the pain, unless functional conditions (cramps) or morbid changes in the tissue itself (indurations, nodes, infiltrations, cords) have already furnished the necessary information. But it is well to bear in mind that sensitiveness on pressure in a certain muscle or in a complex of muscles is by no means always a definite proof of a muscular affection, because it may just as well be due to a morbid process in the intramuscular nerves or veins, or to a complication such as neuromyositis.

Where muscular pain exists the differential diagnosis revolves around the question whether it is due to a primary affection of the muscles themselves or to intramuscular neuritis or to a disease of the muscular veins (neuritis, phlebitis, phlebothrombosis, phlebosclerosis).

In *intramuscular neuritis* the muscles are decidedly sensitive on pressure, and the diagnosis

must be based on other symptoms. In some of these cases the nerve trunks are also very sensitive and show localized swellings (nodes). We also find paresthesia or sensibility disturbances, abnormal tendon reflexes or trophic disorders of the skin, epithelial formations, if not muscular convulsions. All these symptoms are calculated to harden the diagnosis of neuritis. The anamnesis is also likely to reveal certain etiologic factors (intoxications, e.g., arsenic or a preceding infectious disease) or some concomitant manifestation of the disease (a weak heart, an edema in beri-beri).

On the other hand, there are cases of neuritis in which only the muscular branches of the nerves are apparently affected. Whilst all other neuritic symptoms are missing, we only find local pain and tenderness. Here a differentiation between true muscular and intramuscular nervous diseases is simply impossible, at any rate in the initial stages. This explains the reason why some very eminent authorities—the leading figure among them is *A. Schmitt*—look upon so-called muscular rheumatism primarily as a disease of the branches of the muscular nerves; in other words, they consider rheumatic myalgia to be neuralgia.

Where complaint is made of pains which have persisted for some time in the lower extremities, particularly where the muscles of the calves are

sensitive on pressure, we might attribute these conditions to *phlebosclerosis* resp. to *phlebectasia* of the muscular branches of the veins. The existence of ectasy of the superficial cutaneous veins (trunks as well as branches), the presence in the evening of edema in the knuckles and increased pain when the patient stands on his feet for some time will help greatly to make the diagnosis concrete.

But when such pains suddenly arrive chiefly in the muscles of the calves, we should think of *intramuscular phlebitis* resp. *thrombophlebitis.* If, then, manifestations of phlebitis of the cutaneous veins or of the trunks of the veins accede, the diagnosis becomes self-evident. If, however, such an exclusively intramuscular phlebitis continues to exist by itself, the diagnosis is restricted solely to subjective pains and objective sensitiveness. It will gain, however, by the anamnesis, for we know that intramuscular phlebitis is the commonest sequel of a long-continued infection, more rarely of intoxications.

When, however, the two aforesaid etiologic factors are wanting and pain in the muscles is coupled with local sensitiveness, we are justified in looking upon the muscular system as the place of origin of such pains. We then have two alternatives before us, viz.: the affection is either purely local so far as place and time are concerned, or we are dealing with a morbid

condition which involves multiple muscular groups or an extensive area of the muscles of the body. In both cases the pains are either acute and of a short duration, or they have existed already for some time.

I. Locally Limited Unilocular Muscular Pain

Most of the details concerning this subject are contained in other chapters of this volume, as well as in Volume I, "Abdominal Pain." I will, however, repeat collectively this much. When a patient complains of pains in a restricted or even rather extended muscular area and we are satisfied that it is a case of myalgia, we must not look for sensitiveness in the whole area. It may be present only in a limited part of one muscle, especially in its processes, and, again, it may only react under the influence of the electric current. Moreover, there are two phases of acute *localized myalgia;* one is the rheumatic form—muscular rheumatism in the erector trunci or in the muscles of the shoulder—the other is traumatic myalgia. In the latter case the painful condition is either due to an overexertion of primarily normal muscles (sport, athletic exercises, etc.), or to constitutionally weak muscles which cannot stand any kind of normal exercise, e.g., in anemic or physically run-down individuals.

Pains that are localized in or restricted to one extremity or part thereof, also painful muscular convulsions, are often the initial signs of *tetanus,* both local and generalized.

Chronic pains in a definite single muscle or in a certain group of muscles point to the various forms of chronic *myositis,* primarily the *fibrous* chronic type, which attacks not only the lower extremities, but also independently the muscles of the nape of the neck, of the back and of the arms. The patient complains of dragging, rheumatic pains; soon the muscles become rigid, a tumor-like, hard swelling is formed which grows together with the skin and the bones. Of course, so long as this union is not formed, the diagnosis is easy to make from the palpable muscular swelling. Radioscopy and histological examination of an excised piece of muscle will help greatly, and also the fact that in chronic fibrous myositis local sensitiveness is not very pronounced.

Still easier is the diagnosis of localized *myositis ossificans* due to muscular overstrain (horseback riding, military exercises). The patient suffers from severe localized pains (in trauma due to the tendency of the original hematoma to ossify) provoked by the activation of the affected muscles. After a week or two a hard tumor may be felt similar to that found in intramuscular hematoma, syphiloma and osteosar-

coma. The Roentgen picture is sufficient for a proper diagnosis.

I implicitly affirm that localized muscular pains must ever remind us of all possible *neoplastic* (also echinococcus), acute as well as chronic *inflammatory* (gummatous, tuberculous and gonorrhoic) *changes in the muscular system.* The differential diagnosis, however, affects the surgeon more than the internist.

Not only new growths in the muscles, but also diseases of the bones, especially *myeloma,* are attended with localized, often severe muscular pains, for which reason the wrong diagnosis of lumbago or muscular rheumatism is an everyday occurrence.

An attack of sudden severe pains in a definite group of muscles, if not in multiple areas, especially when they bound from one group to another—without fever or, perhaps, with fever for a day or two—indicates acute *muscular rheumatism* or rheumatic polymyalgia. It may also be the manifestation of an infectious disease the character of which has not as yet come to the surface, but very likely due to catarrh. Toxic conditions with a possible secondary inflammation of the endocardium or of the pleura, of the pericardium or myocardium, are also to be considered. The affected muscle appears swollen, hypertonic when palpated in relaxation. The same may be said of subacute or chronic

muscular rheumatism, in which, however, certain muscular fascia feel to the touch more like very hard cords. We also find at the deeper insertion of the muscles nodules hard as bone and about the size of a pea, which are very painful at times. In muscular rheumatism the processes of the affected muscles are particularly painful on pressure. Lifting the muscles and pressing them between the fingers elicit exquisite pain. The patient feels the pains very intensely when he rises in the morning, but they relax as he gets about. They come on all of a sudden when he sneezes, stumbles or rides over a hard road, etc., also with a change in the weather. Antirheumatic drugs give generally desired relief.

Homogeneous muscular pains localized in a definite zone (lumbago, myalgia of the head or of the shoulder blades or neck and arms), also when of a shifting character, are often the sign of *uratic diathesis*. They have no basic value for the differential diagnosis, and we must look for other symptoms. So far as *gout* is concerned, we may be guided by: inclination to cramps in the calves, cutaneous affections, itching, chronic eczema, hemorrhoids, indigestion, neuralgic pains, inflammations of the synovial sacs, tophi (cf. chapter on "Pains in the Chest"), hereditary conditions, personal habits (meat-eaters, luxurious living), examination of urine, retarda-

tion of the nuclear metabolism, persistent uricemia by purin-free diet and slow secretion of uric acid after the administration of nucleinic natrium.

In *diabetes mellitus* and in *chronic alcoholism* we find analogous shifting pains. The etiologic diagnosis should offer no embarrassment.

Neurasthenia is likewise the source of vague, sometimes very intensive muscular pains. We find them particularly in the chest, back, loins and extremities. The patient anticipates some trouble in the lungs, kidneys, or in the spinal cord. Local tenderness in the muscles and sensitive reaction to the electric current reveal the seat of the pain.

Chronic pains in the muscles are nearly always there in *Addison's disease.* They chiefly affect those in the back, arms and legs, in the shape of a painful feeling of fatigue, but when the disease becomes more acute they gain in intensity and therapeutic measures seem to afford but little relief. Morbid changes in the muscles are not in evidence, and electric reaction remains the same. Mechanical hyperreaction in the muscles, painful or otherwise, is a definite expression of *Addison's* cachexia. The diagnosis can only be made with the aid of the other characteristic symptoms of this classical disease (melanodermia, adynemia, low blood pressure).

Myxedema, resp. *hypothyreoidism,* is another disease of the internal secretory glands which provokes sleep-disturbing muscular pain. Because pains in the back, a feeling of cold and abnormal psychic and physical fatigue, especially in the morning, accompany this disease, it is often mistaken for neurasthenia or anemia. But if we note the bloated face, the swollen eyelids, the puffed lips, the tendency to subnormal temperature, epilation, trophic changes in the nails, menstrual disorders (amenorrhea, menorrhagia), slight swellings in the joints, constipation and changes in the psychic and physical disposition of the patient (apathy, sleeplessness, sluggishness of the mental faculties), we have all the diagnostic facts before us.

In sporadic cases of *Basedow's disease,* muscular pains associated with signs of severe myasthenia have been observed. Whether an affection of the thymus gland is responsible for this condition is questionable.

There is also a case of tumor of the hypophysis with intense muscular pains on record. The presence of the typical signs of this disease may, perhaps, remove a possible doubt, but there is still the likelihood that these pains have their genesis in another morbid endocrine gland.

If slight pains have persisted in various parts of the muscular system, chronic *lead poisoning* may be the irritating cause. But the clinician

must look here for further adjuvants, such as lead colic, anemia, the blue line, articular pain, paralysis of the radialis and punctated red blood corpuscles. The pains are of a vicious, boring and tearing nature, especially during the night-time, and affect the joints and bones as well.

Subacute *mercury* (sublimate) *poisoning* is characterized by pains of short duration in the extremities, especially the legs. But I have seen a case in which this intoxication provoked exceedingly severe spontaneous and pressure pains.

If a patient complains of acute, at first short-lived pains which at the beginning are confined to a limited group of muscles but soon spread to other groups, or if from the very start various zones of the muscular system are involved, we will have to decide between acute *polymyositis, neuromyositis* and *dermatomyositis.*

Dermatomyositis may be acute, subacute or chronic, and is typified by: acute beginning, high fever, facial edema, especially in the eyelids, spontaneous and exquisite pain on pressure and on motation at first in a certain group of muscles, generally in one side of the calf, thence reaching over into the contra-lateral calf, and extending from there into the femoral muscles and even those of the trunk. With every fresh localization the general condition of the patient grows worse as new symptoms manifest themselves, such as eruptions of the skin which

strongly resemble erythematous conditions, or urticaria, or roseola, or even erysipelas. Later on we notice a hard edema in the skin over the affected muscles which does not yield to pressure, so that the extremities look like shapeless columns, and the face and trunk appear deformed. When the edema subsides the muscles remain strongly indurated. Profuse sweating is common and frequent.

The muscular symptoms of neuromyositis are a compound of those belonging to dermatomyositis and multiple neuritis.

Acute hemorrhagic polymyositis and acute polymyositis combined with erythema nodosum are rather of rare occurrence. Both have a certain symptom complex in common, i.e., febrile conditions, the attack is confined to the extremities and manifests itself in local pains, local, at times soft edema of the skin, local swelling of the muscles and painful muscular tumor. The muscular system of the trunk is not affected. The acute hemorrhagic form is specially characterized by hemorrhages and sugillations of the skin over the affected muscles, hemorrhages in the mucous membrane (intestinal hemorrhages). The other form is typified by complicating erythema nodosum and frequently enough by additional acute articular inflammations.

These three forms of polymyositis are so strongly marked that a differential diagnosis be-

tween them and other diseases is rigidly excluded.

Of course, the matter assumes a different aspect when acute or subacute dermatomyositis is in question. If the disease sets in with fever, edema of the eyelids, forehead or temples, accompanied by muscular pains in the extremities and thorax, which are at times so severe that the patient lies motionless, if the muscles are swollen and hard, and we find profuse perspiration and cutaneous exanthemata similar to those of dermatomyositis, the diagnosis must be *trichinosis.* A differentiation between these two diseases is always necessary and can easily be made, especially when trichinæ are found in the blood, in the muscular tissue or in the stools, and the biceps muscle is particularly affected. Involvement of the diaphragm and of the muscles of the eyes, jawbones, and throat is not ordinarily connected with acute dermatomyositis, but perhaps more common than in trichinosis.

In milder cases of trichinosis, when the patient complains merely of general indisposition, unusual fatigue and slight muscular pain, and the rise in the temperature is insignificant, the danger lies near to mistake the disease for *acute muscular rheumatism.* The same error may be made when the symptoms of trichinosis taper down into a softer character towards the end of the illness.

In recent years eosinophilia has been observed in muscular rheumatism, especially in cases of acute relapse. A muscular induration of old standing may be the possible remainder of a pristine invasion of trichinæ and invite a false diagnosis of muscular rheumatism. The excision of a small bit of muscular tissue should with the aid of the microscope promptly correct the error.

Cysticercus is another parasite which produces, though not of necessity, spastic pains in the muscles and through pressure in the nerves. The presence of the parasite in the muscular tissue or else in the fundus of the eye or in the brain is sufficient evidence not to speak of eosinophylia of the blood. Pains are rarely felt in this disease, which may be erroneously taken for a fibroma.

The differentiation between polymyositis and *purulent myositis,* which sets in with localized painfulness, swelling and induration of a certain muscle, should offer no difficulties. The collateral edematous condition of the skin, with early local fluctuation, the general symptoms, state of the blood and evidence of suppuration are typical enough of the nature of the disease.

It is different, however, when the initial stages of acute dermatomyositis are localized in one extremity only, e.g., in the muscles of the calf or some other lower portion of the leg. A sud-

den onset of fever and pain in the extremities, local sensitiveness and motoric inhibition, local edema of the skin have misled many a clinician into making a diagnosis of acute dermatomyositis, or acute myositis, when the patient was in reality suffering from an attack of *thrombophlebitis* of the crural vein. The error would not have happened if more careful attention had been paid to the following facts: In thrombophlebitis the edema shows an indentation; it does not do so in dermatomyositis; in the former the patient perspires profusely and the edematous condition of the skin does not permit us to palpate the thickened, painful collateral ectatic veins. I will also mention that thrombophlebitis may also occur in young people when the suspicion of acute *syphilitic myositis* lies near. But, let us remember that the latter disease carries with it pain, swelling and induration in the affected muscles, but is hardly ever attended with edema of the skin; but if it is, the edema is hard the same as in ordinary dermatomyositis. Besides, tentative specific treatment and the complement fixation test should soon clear the situation.

Acute infectious diseases are a prolific source of muscular pains. We may here be dealing with a true infectious myositis indicated by swelling and hardness of the painful muscles, e.g., the *rheumatic* or *gonorrhoic* form of *myositis*. The

former is either a preceding or an accompanying manifestation of acute articular rheumatism, or takes on the form of a subsequent relapse. The second form is easily recognised, being, as it is, merely a coincidence of a gonorrhoic articular inflammation.

The commonest form is acute *pyemic metastatic myositis,* associated with many different kinds of blood infection (*myositis acuta malleosa*).

Purulent myositis may also supervene by continuity in primary inflammations of the skin or bones.

Myalgia is of frequent occurrence in the majority of acute infections, and we are strongly reminded here of *articular rheumatism* (*rheumatic hyalgia*).

Typhoid fever belongs here, too; and we may be justified to use the term "myotyphoid" in this connection.

Muscular pains are not so unusual in *paratyphoid* either. In typhoid fever, these pains are often caused, in the 2. to 4. week of the disease, by the waxy degeneration of the abdominal as well as other muscles—of the femoral adductors. The muscles feel soft like wax even when they are contracted. Error of peritonitis is possible here. (Cf. "Abdominal Pain.")

But there is another type of similar muscular pains which often set in at the height of a fever.

They attack by preference the oblique and straight muscles of the abdomen and the adductors, likewise the muscles in the back. We should be careful not to attribute these pains to a waxy, but rather to a toxic parenchymatous degeneration of the abdominal muscles. From a clinical standpoint it will be, perhaps, difficult to make a differentiation.

Similar muscular changes occur in other acute infections such as *pneumonia, sepsis, pyemia, influenza* and *cholera.* But it may be observed that these morbid conditions are, as a rule, not associated with subjective pain, but rather with a feeling of muscular fatigue and local tenderness.

Whenever the patient complains of vague, dragging pains in a certain group of muscles, pains which have a tendency to jump from one group to another, we are safe to diagnose an attack of chronic mitigated *sepsis* in the oral, pharyngeal cavities (teeth, tonsils). Particulars will be found in "Abdominal Pain" and in the chapter on "Nuchal Pains." Furthermore, there is also a form of mixed infection of sepsis and tetanus. During the World War many such cases were observed and the opportunity was utilized to study the genesis of this peculiar complication.

There are cases of *pneumonia*—diplococcus infection—and also sepsis (pyemia) in which the muscular painfulness may be due to a bacterial

invasion of the muscular arteries, i.e., to a true acute *embolic myositis.* Here, too, the sensation hardly ever passes beyond the stage of muscular fatigue or tenderness.

In *recurrent* and in *exanthematous fever* muscular pains are predominant. They are combined with initial chills, and localized in the calves and in the margin of the cucullaris, in the axillary folds and in the processes of the sternocleidomastoid of the sternum. We may look upon them as a characteristic symptom caused by a waxy degeneration of the muscles.

In typical cases of *malaria,* especially tropical, intensive muscular pains accompany every attack of fever. We find them in dorsal and lumbar muscles as well as in those of the extremities.

During the prodromal stadium of the *plague* severe muscular pains are experienced in the nape of the neck and shoulders. And in *Wolhynian fever* they attack the calves, thighs, deltoid, back and chest.

The *influenza* patient complains of very severe, tearing pains with exquisite tenderness in the swollen muscles of the thigh, the calf, nape of the neck and back, particularly during the night time. They are not so much due to toxic influences but rather to localized influenzal myositis. The diagnosis can be easily made from the characteristic symptoms of the disease. But there is always a possibility of post-influenzal

myalgia and myositis accompanied by muscular pains. Complications with other diseases such as typhoid, etc., are not excluded.

Pappataci fever belongs to this same category. I have already spoken about the diagnosis of this disease in another place.

Poliomyelitis is apt to be confused with articular rheumatism or influenza. I refer the reader to the chapter on "Articular Pains."

Muscular and articular pains accompany *yellow fever* in its second stage. In epidemics the diagnosis is self-evident nearly throughout. But in solitary cases it can be made from the initial symptoms of generalized infection, high fever, headache, lumbar, muscular and articular pains, vomiting, feeling of pressure in the epigastrium, hemorrhages and icterus.

In *Weil's disease* we find among the earliest symptoms fever, chills, vomiting, diarrhea, followed by muscular pains, especially in the calves, the nape of the neck, chest, abdomen, sacrum and thighs, acute splenic tumor, and within three to five days icterus, enlargement of the liver, acholia in the stools, nephritis, delirium, nosebleed, hemorrhages of the skin and mucous membranes, herpes leucocytosis, polynucleosis and lymphopenia. The disease might be taken for infectious cholangitis or typhoid with icterus, but the detection of spirochetœ in the blood should soon correct any such error.

Paroxysmal hemoglobinuria manifests itself by chills and fever, high temperature, urobilinuria, hemoglobinuria and cylindruria together with muscular pains.

In chronic as well as in acute cases of *overfatigue* we find pains in the muscles, sleeplessness, muscular twitching, paresthesia, abnormal irritability, neuralgia, palpitation of the heart, trembling, burning sensation in the chest and in the extremities.

In *Weil's disease* hemorrhages in the muscular tissue cause local pains, and in *scurvy* pains in the calves are provoked by intramuscular hematomata.

Muscular pains in the loins, sacrum and thigh are initial symptoms of *hemoglobinuria* after forced marches and other strenuous military or athletic exercises through which the hemoglobin content of the muscles is used up.

Periarteritis nodosa is a subacute or chronic infection in which multiple muscular pains are frequently very prominent in various areas. It is sometimes attended with fever and paresis or paralysis of a degenerative character of the affected muscles. Acute nephritis, diarrhea with bloody stools, profuse intestinal hemorrhages and severe colic are other complications, likewise abdominal pain, peritonitis and strongly marked cachexia coupled with anemic conditions. The diagnosis, however, can be made positive only

when we find noduliform swellings in the small cutaneous arteries. If syphilis is suspected the *Wassermann* reaction may enlighten us. It is advisable to think of periarteritis in all cases which suggest the existence of polymyositis or trichinosis.

Muscular pains of a minor degree or rather an abnormal feeling of fatigue in the muscles frequently accompany the period of recovery from an *infectious disease* of the muscles in the sacrum, loins or extremities, such as smallpox, influenza, erysipelas and certain rudimentary forms of dysentery.

Local or generalized muscular pains indicate also chronic infections, e.g., tuberculosis or syphilis, which are apt to present pseudoneurasthenic conditions of a continuous, remittent or intermittent character. They are also forerunners of lactic exanthema. The usual local and generalized pertinent symptoms are the necessary requisites for a correct diagnosis.

Pains in the muscles of the calves are a common early symptom of *edematous diseases.* The diagnosis results from abnormal fatigue, hypotonous bradycardia and polyuria in addition to the edematous conditions.

In the chapters on "Pains in the Shoulders" and "in the Nape of the Neck" I have pointed out that pains which radiate particularly into the muscles of the shoulders should remind us

of some morbid process in the *lungs,* primarily of tuberculosis, but also of other *subdiaphragmatic* or *intraperitoneal affections,* although as a rule they are not spontaneous in character but rather elicited by local pressure. Similar tenderness in the trapezius, rhomboideus and pectoralis muscle is also possible in *dry pleurisy.*

In *trismus* painfulness and rigidity of the muscles are also perceptible. An acute inflammation of the maxillary joint or of the jawbone, or an articular stricture following an acute painful affection of the oral cavity (wisdom tooth, tongue, tonsils) or of the upper salivary glands at times bear a strong resemblance to lockjaw and render the differential diagnosis difficult, particularly so in cases of periosteal abscess in the upper maxilla due to an injury. If the maxillary stricture sets in *immediately* after the injury lockjaw is contraindicated, but if the stricture persists while the abscess recedes true trismus is affirmed. In some rare cases of acute rheumatism of the temporalis muscle similar conditions have been observed (*Oppolzer*).

I recall two interesting cases of *pseudotrismus* in two soldiers. One was that of a military surgeon who attended a wounded man with tetanus infection, the other was an infantryman who lay in the bed next to that of a patient afflicted with trismus and subsequent tetanus. Within twenty-four hours both men were at-

tacked by trismus. They were unable to open their mouths, the private soldier even showing signs of hysteria. A clear proof of pseudo-trismus caused by imitation neurosis.

In hysteria analogous phenomena have been observed. A local trauma, e.g., a gunshot wound in the face may produce an hysterical maxillary stricture with synchronous analgesia of the face. Under suggestion the trouble generally disappears. The diagnosis should not be difficult if we pay attention to the other typical signs of hysteria.

Trismus is frequently an early symptom of *tetanus.* Here, too, the diagnosis is obvious on the strength of the usual tetanic characteristics.

Epidemic and *tuberculous meningitis* as well as epidemic encephalitis frequently present in their incipient stages the clinical picture of trismus, and in many cases of poisoning lockjaw is an accompanying manifestation. If a patient shows signs of trismus coupled with intensive headache, pressure sensitiveness in the head or cerebral vomiting we should more correctly diagnose meningitis as the originating cause and not trismus or tetanus. Lumbar puncture will furnish additional proof. From a similar standpoint we can also explain the occurrence of trismus besides other cerebral symptoms in pernicious anemia of the cerebral type.

In other *acute infections* initial trismus may

be the effect of the bacterial poisons on the brain, e.g., in *recurrent fever,* in which disease the muscles at times are so stiffened that a suspicion of tetanus is pardonable. *Diphtheria, erysipelas, influenza, Weil's disease* and *tonsillar angina* are other pertinent instances. In *typhoid fever* trismus is more common in the final stages. When it occurs in any of these infectious diseases it must be taken as a definite proof of very severe bacterial intoxication and of great significance for the prognosis, more so even than clonic cramps in the masseters or grating of the teeth.

But it is not only general toxic conditions but also strongly localized *diseases of the brain* from which trismus may result that lasts for weeks, months and even years, e.g., a tumor (tubercle) in the posterior pons or a softening process from sclerosis or a syphilitic endarteritis of the basilary artery caused by an irritation of the motoric trigeminus. We can in this manner explain the occurrence of trismus and grating of the teeth in meningitis and in pseudomeningitis for which a basal posterior meningitis is responsible.

In some cases of *tetany* trismus is also developed. The differentiation between tetany and tetanus is discussed elsewhere.

If trismus occurs in trichinosis it is of peripheral and not central origin and a proof of the presence of trichinæ in the masseter muscles.

In *endogenous poisoning* trismus is likewise a

possibility. It may be a manifestation in part of *icterus,* of *cholemia* or *pellagra.* Perhaps more frequently and mostly coupled with other cerebral symptoms it is an associate of certain acute exogenous intoxications, chiefly blood poisoning (hydrocyanic acid, aniline, coal gas, coal dust, hemlock, helvella, fly agaric, scorpion poison). In acute atropin poisoning trismus may occur in combination with loss of consciousness, *Argyll-Robertson* sign, absolute dryness of the mucous membrane of the mouth, erythema, etc. Acute nicotinism belongs here, too. Yet, in all these conditions trismus does not occupy a very prominent position, if we except acute strychnine, opium and morphine poisoning, especially in children. In these cases trismus may be an isolated condition or, perhaps, a partial manifestation of generalized tetanic convulsions.

True *infectious tetanus* is always primarily suggested when the patient manifests sudden attacks of painful convulsions in the masseter or nuchal muscles. These paroxysms (local or universal) come on spontaneously or may be reactions of external causes, and are felt by the patient like the thrust of a dagger generally in the injured part of the body. They are, so to speak, a kind of tetanic aura preceding the general spastic attack. Nevertheless, they may just as well constitute the solitary symptom of a tetanus infection which persists as "local te-

tanus" without development of a generalized state of tetanus. I have given full details about this subject in the chapter on "Pains in the Extremities."

If the bacteriological examination of the wound fails to show the presence of the tetanus bacillus we must look for other morbid processes which are known to produce analogous tetanic convulsions.

Among these *strychnine* poisoning is the first to be considered. Here we find spasms in the extensor muscles of a tetanic nature with risus sardonicus and yet unclouded mind, reflectoric hyperexcitability of the patient, retention of urine, dysuria, rising temperature and profuse perspiration. If anamnetic data are wanting, but we discover a wound or a scar on the body of the patient, a differential diagnosis between strychnine poisoning and tetanus will be imperative. We can arrive at a proper recognition of tetanus by its gradual development. We first witness trismus, then opisthotonos and after that generalized extensor convulsions. In acute strychnine poisoning, on the other hand, the universal cramps set in all of a sudden, sometimes, it is true, after short precursory symptoms. It stands to reason that in most instances we are not able to observe these various stages. In fact, it is most difficult to ascertain in the vast majority of cases what has happened previous

to the real attack. Then we must lean on the fact that in strychnine poisoning the muscles relax during the intervals between the spastic attacks. The patient is able to move freely, can even stand on his feet without assistance; whilst in tetanus the muscles remain rigid until recovery ensues. And, again, tetanus is a long continued disease, while in strychnine poisoning the cramps (chiefly) in the extremities—especially in the hands—soon pass away. I do not know whether in strychnine poisoning leucocytosis and eosinophilia, so common in tetanus, are also found.

Tonic, tetaniform convulsions, opisthotonos, increased reflex action and also, it is claimed, leucocytosis are also observed in *hydrophrobia.* It is distinguished from tetanus and strychnine poisoning by absence of trismus, by the fear of, or inability to swallow water, fear of water, psychic agitation and a feeling of anguish. Furthermore, the anamnesis and the fact that in rabies the muscles in general, but especially those in the nape of the neck and of the masticatory apparatus, relax in between the spasms, are an additional help. Under the heading of dysphagia I speak of the differential diagnosis between hydrophobic tetanus and hydrophobia. I will, however, mention that in the former the muscles of deglutition and those of the diaphragm are deeply involved.

Hysteria also breeds sometimes painful, tetaniform extensor cramps. I have, indeed, seen myself a case of acute strychnine poisoning which was erroneously labeled "hysteria." The differential diagnosis must be built upon the anamnesis, the presence of other symptoms characteristic of hysteria and the course run by the disease. Of course, it is always possible that an individual with an hysterical taint and afflicted with exaggeration mania may at any time acquire a genuine attack of tetanus.

Similar spastic conditions especially in the muscles of the nape of the neck, the back and extremities follow sometimes in the wake of cerebral affections, but they are free from pain and do not affect consciousness. Here we find manifestations of paresis or paralysis of the cerebral nerves which might suggest tetanus of the hand, unless traces of a local wound are wanting. We also meet with bilateral *Babinski's* toe phenomenon and unilateral absence of abdominal wall and cremaster reflexes. Moreover, lumbar puncture yields a bloody-red or sanguinolent cerebrospinal fluid containing diluted red blood corpuscles without sendimentation—a sure proof of cerebral genesis. But I emphasize the fact that an anemic condition of the blood is also found when the puncture is made only sometime after the cerebral hemorrhage has taken place. If we are enabled to make the puncture soon—

even within an hour—after the injury, we are likely to find perfect red corpuscles. After sedimentation the cerebrospinal fluid may appear tinged with blood should the needle have passed through a meningeal vein. It is wise to watch the fluid whilst it is being drawn and see if it is evenly tinged with blood all throughout the act of puncturing. As a rule the first portions alone are tinged with blood or at any rate more so than those that follow. If the patient has been lying motionless for hours when we arrive, we may well exclude tetanus and acute strychnine poisoning.

When extensor convulsions in the dorsal muscles or in those of the extremities are found in a patient who is not in a comatose state the foregoing is of particular moment especially when other positive or negative symptoms affecting the central nervous system are present.

The differential diagnosis must be made from the same angle in those rare cases of *cerebrospinal* meningitis (purulent epidemic or non-epidemic, or tuberculous) which give rise to tetanic extensor convulsions. The same applies also to those rare cases of *acute encephalitis* which may be observed after acute influenza and likewise of *saturnine encephalopathia* which is fully dealt with in the volume on "Abdominal Pain" under the title of "chronic lead poisoning."

The severe form of *arteriosclerosis of the cere-*

bral arteries is another harbinger of painful tonic muscle clonus in the trunk and in the extremities associated with mono- and hemiplegia, disturbances of speech and slight apraxia.

Fatty embolism of the brain belongs to this section also. It may happen in babies before the foramen ovale has closed up or in persons of old age and in heavy drinkers. Although this disease is of minor interest to the internist I deem it advisable to mention that we find here cerebral irritations in the sense of trismus, opisthotonos and spastic attacks chiefly of an epileptiform character, followed a few hours later by dullness in the sensorium and a small, irregular pulse. But the involvement of consciousness is the master key to the diagnosis which might be led into an error by the fact that in tetanus also there is serious destruction in the tissues and vessels.

Similar tonic spasms are often observed in *cerebellar tumors* in which opisthotonos is not an infrequent accompanying symptom. It would be superfluous to discuss here the other typical signs of these tumors. This refers with equal force to tumors of the pons and of the medulla oblongata in which every form of tonic spasms, light convulsive tremor and slightly clouded consciousness is of diagnostic import.

Meningeal hemorrhages give rise to muscular contractions which strongly resemble convulsions

so typical of strychnine poisoning. The behavior of the sensorium and of the cerebral nerves and lumbar puncture are generally sufficient to throw the proper light on the situation.

Spasms of this tetanic character are, however, quite common in a number of exogenous poisonings such as carbon dioxid, sewer gas, arsenic, mercury, alcohol, dicyanogen, nitrobenzol iodoform. The coincidence of the cerebral manifestations, i.e., loss of consciousness and simultaneous spasms, are the strongest differential-diagnostic points in these cases.

Acute cases of *opium or morphine intoxication* are to be included here, especially in children. Trismus, opisthotonos and tonic spasms are always present. The diagnosis results from the observation of the comatose state, the want of reaction to light in the closed pupils, of bradycardia, bradypnea, fall of body temperature and the presence of the drug in the saliva, the urine and the gastric contents.

In *ergot* poisoning the tonic spasms do not only affect the extremities but also the muscles in the back, in the throat and in the diaphragm, lasting continuously sometimes for days. The diagnosis is materially assisted when gangrenous spots in the skin are observed and when a painful creeping sensation has made itself felt all over the body for some time previous to the spastic onset.

There are some cases of diphtherin poisoning on record which are called *"spasmogenous diphtheria"* in which tonic spasms are observed, but I cannot speak from personal experience on this matter. The symptoms are described as follows: presence of bacillus diphtheriæ in nose and throat, diphtheritic membranes, inefficiency of antitetanus serum, reaction to diphtheritic antitoxin.

Escherich and *v. Jacksch* speak of *pseudotetanus,* a condition of generalized tonic cramps in the articular muscles of the jaws, the nape of the neck and the back which continue for months at a time and are caused by some *infectious disease.* When it exists as an independent disease the tonic spasms affect principally the lower extremities; trismus sets in later on. It yields to treatment.

Kollert describes tetanus-like conditions in some severe cases of *spotted typhus.* In *tropical malaria* cerebral symptoms such as delirium, eclampsia and coma are not uncommon manifestations.

In *uremia* tonic as well as clonic spasms are observed. The diagnosis can be made without difficulty.

Painful muscular cramps in *eclampsia* and *epilepsy* are sometimes erroneously taken for a symptom of acute strychnine poisoning. But the mistake can be easily prevented if proper

attention is given to the presence of edematous conditions in the skin and of albuminuria so typical of the former diseases, and the solitary attack of convulsions and their clonic character, the total loss of consciousness during the attack, the involuntary discharge of urine and feces, bites in the tongue, the want of pupillary reaction and the anamensis in the case of strychnine poisoning.

There are likewise cases of *tetany* in which the spasms involve the whole body with the presence of opisthotonos and trismus which may put a mistaken diagnosis of tetanus within the range of possibilities. But the want of reflex action characteristic of tetanus, the presence of the cardinal symptoms of tetany, the mechanical overexcitation of the motoric nerves (the facial phenomenon), the hyper-reaction of the motoric nerves to the electric current (*Erb's* phenomenon) and *Trousseau's* sign (the arrest of the typical spastic attack by compression of the sulcus bicipitalis internus) should clearly establish the differentiation.

A long continued *exposure to heat,* e.g., working in an overheated boiler room, i.e., collapse from heat, provokes tetanic muscular spasms with trismus, no doubt due to the enormous loss of water in sweating. The same may be said of *sunstroke* or *heatstroke* which, clinically speaking, strongly resemble uremia: total loss of con-

sciousness, pulse hardly perceptible, respiration suspended, pale, cyanotic appearance of the face, very high body temperature, tetanic muscular contractions and trismus are the outstanding features of this condition which is due to abnormal over-exertion of the cardiac organs and excessive action of the respiratory muscles.

Pain in the Bones

When a patient complains of pain in a bone and we find local changes and tenderness, the diagnosis is simple enough. However, when the complaint is of a rather vague nature, for instance, of rheumatic, steady or variable or intermittent pain now in the arm, then in the leg, but with varying intensity, the thought of a muscular affection will be uppermost in our mind, and yet the originating cause may lie in a morbid condition of the bones. A careful search for local tenderness or swelling in the osseal parts will soon correct our first impression. Many diseases of the bones can be readily diagnosed in this manner.

I. Unilocular Pain in the Bones

If we find local sensitiveness on pressure or percussion with or without anomalous configurations we are confronted either by a purely regional, local disease or with the manifestation of a multilocular affection which is as yet, or may be permanently localized in that spot.

In the first case we are dealing with the various forms of osteoperiostitis as well as with the local primary and metastatic neoplasms of the

bones. These belong, in part at least, in the field of internal medicine, but chiefly affect the surgeon. Those of interest to the internist will engage our attention in the succeeding pages.

The first to speak of is *syphilitic osteoperiostitis* together with that of tuberculous, actinomycotic and malleose genesis, and also acute as well as chronic osteomyelitis. They will all be discussed in separate paragraphs. But I must add here various other divisions of secondary, infectious osteoperiostitis which follow in the wake of divers infectious diseases. Among these I mention acute articular rheumatism (osteoperiostitis rheumatica and postrheumatica) with demonstrable swellings sometimes of the character of albuminous osteoperiostitis or with limited sensitiveness in the bones, for instance in the calcaneum; likewise purulent infections and infections from the paratyphoid bacillus, bacterium coli or dipplococcus (pyemia); further, typhoid fever, smallpox, measles, scarlet fever, influenza, bacilary dysentery and gonorrhea.

In many of the aforementioned infections the morbid affection of the bones and the accompanying pains are merely a distinct expression of the gradual development of the disease itself; even though superficial reddening of the skin, and swelling or enlargement of the bones are not yet in evidence. Of course, we must not

forget that, when the generalized infection has been fully developed, the accompanying pain in the bones may just as well be the expression of the generalized infection itself although inflammatory neurotic changes in the bones are not present at all. In that case we are not dealing with one solitary local focus but with multiple foci for the pain, especially so when these pains are not spontaneous but only ensue from provocation.

This painful focus in the bone, on the other hand, may just as well be the first and only spot where the disease is localized. This is the case, e.g., in acute osteomyelitis, although this disease is often enough localized in several places either at the same time or in successive periods. It happens also in actinomycotic, tuberculous or syphilitic osteoperiostitis. In this the primary port of entry of the infection is not always apparent, a careful search may reveal it, but often enough it remains an unsolved problem. A similar dilemma faces us sometimes in other forms of post-infectious osteoperiostitis in which the generalized infection and the manifestation of the local focus of the pains are often years apart, for instance typhoid periostitis which may set in on the orbital margin or on the skull even during the first week of the disease.

These cases are very difficult to diagnose. Only the widest search for every detail that

might possibly be of use can clear the situation. We may find, for instance, a sign of ostitis (acute, chronic, purulent or plastic) on a rib, on the sternum, on the tibia or on a vertebra or even on the skull. There we have a suggestion of lues or tuberculosis. We may hear of an attack of typhoid fever, ten, fifteen or even twenty years ago, when typhoid periostitis would not be an improper guess, or we may be dealing with a case of autointoxication of a chronic typhoid bacillus carrier, due to some trouble of the gallbladder. A bacteriological examination of the contents of the lesion is very seldom attainable, but the *Gruber-Widal* reaction, repeatedly made during the course of the disease, will very likely give us the right clue, especially when antisyphilitic treatment has proved ineffective. But even so, a positive *Gruber-Widal* reaction or the anamnesis are not always absolutely reliable indications.

I may mention also that the *nocturnal habit of the pains* is not always a definite proof of their syphilitic genesis. We find it also in typhoid and posttyphoid osteoperiostitis, not to speak of *carcinomatous metastasis of the bones* and above all *gouty* pains in the bones. The latter would, however, rather represent articular pains owing to the presence of uric acid in the articular ends of the bones.

At any rate in these complicated cases we

shall derive much help from the local condition, the X-ray, the serological examination and tentative antisyphilitic treatment.

But there are very stubborn cases of syphilitic ostitis which resist every form of specific therapeutic measure or at the utmost give only a fleeting reaction.

The diagnosis of the other forms of osteoperiostitis generally results from the anamnesis of the causating acute infection. With regard to *gonorrhea* I must add that this disease may, in rare cases, give rise to an attack of osteoperiostitis (e.g., in the clavicle or femur) of such magnitude that an erroneous diagnosis of a sarcoma may easily creep in.

There is a special lower form of periostitis which is mostly of a localized, although at times also of a multiple character, which deserves special mention here, viz., *angio-neurotic pseudoperiostitis.* Angio-neurotic processes provoke very transient but ever recurring exudates in the skin and mucous membranes and also in the joints. We call them hydrops hypostrophos. But similar conditions may also be observed on the peristeum. We find swellings in certain parts of the bones which come on suddenly, are of a doughy consistence, spontaneously painful and very sensitive. They generally localize in one of the ribs or in the sternum. In the fleeting character of these swellings, in their variability

of localization, in the similarity of the symptoms to those of hydrops hypostrophos we find the key to the proper recognition of this disease. Among the concomitant symptoms we count, asthmatic attacks, mucous colitis, vasomotoric rhinitis, urticaria and vasomotoric disorders of the skin. All these manifestations are comprised in the term "exudative diathesis" recently introduced in medical literature.

In *Sudek's atrophy of the bones* active as well as passive movements provoke serious pains in the bones and are an important symptom of this affection. As it is a surgical disease I cannot claim personal knowledge of it, but the symptoms are described as follows. It sets in after inflammatory processes in the cellular tissue which are of simple, tuberculous, probably also syphilitic and gonorrhoic origin, specifically also after injuries to the bones. The distinguishing symptoms are: a characteristically hard, doughy edema of the subcutaneous cellular tissue, coupled with malnutrition of the softer tissues, a shiny, bluish-red skin, copious sweating and a feeling of cold in the morbid section. The X-ray is an important factor in the diagnosis.

In local processes caused by neoplasms the diagnosis will not be difficult, if we find demonstrable changes, such as thickening or enlargement of the bones, especially if the parchment crackling sound is present. The X-ray should

prove of great assistance, but in cases in which it cannot be employed, mistakes are not always avoidable. A sarcoma may be taken for osteo-periostitis resp. osteomyelitis, for frequently the fact is overlooked that in sarcoma also the skin over the swelling feels hot and is very sensitive. Let us remember that in primary neoplasms the skin is distinctly interwoven with enlarged blood vessels.

Metastatic neoplasms require the attention of the internist as well as that of the surgeon, for in many cases abnormal conditions, or even sensitiveness in the bones cannot be detected. This makes it very difficult to form an opinion whether the painful sensation arises from an affected bone or is due to some other cause. The situation is improved if we can find the evidence of a previous primary neoplasm, no matter how large or little, for that might put us on the right track in our search for the genesis of the pain. We might be told, for instance, that five or ten years ago a primary neoplasm was removed from the mamma. It is exactly this kind of case in which late metastases of the bones develop. Another point worth mentioning is that primary neoplasms generally are formed in just such places where metastases of the bones prefer to develop. I mean the mamma, the thyreoid gland, the prostate, the adrenals (hypernephroma), the ovaries, the male genitals (testicles), bronchi,

stomach and sigmoid. Sometimes these primary tumors escape our clinical observations because they are so small and hardly palpable, when the metastatic conditions in the bone marrow are already provoking most painful sensations. This is particularly the case in malignant primary new growths of the thyreoid and prostate. The spinal localization of the pains in any of these parts is always a suspicious signal. When the pains are localized near the head of the upper arm or of the thigh, or in the pelvic region or in the zone of a vertebra belonging to the lower costal or to the lumbar spinal column, or in one of the ribs or in the sternum, especially when these thoracic pains are provoked by motation or impair—completely arrest—the motoric action of the gait, and we cannot, on the other hand, discover a demonstrable cause for these manifestations, then we have sufficient ground for suspecting the development of a metastatic carcinoma of the bones.

Similar metastatic conditions are found in simple, but not malignant *colloid struma.* They are generally combined with other metastases in the skin, mucous membranes, etc.

Pains localized in one bone only may be the reflex action of a morbid process which is in the initial or rudimentary stage of development in other sections of the skeletal frame. I refer particularly to *Paget's ostitis deformans* which

may exist in only one leg for a number of years. The diagnosis should offer no difficulty, especially when the characteristic thickening, softening and curvature of the tibia is in evidence. The more so, when the patient is beyond the forties and the pains are rather severe, neuralgiform and constant. Besides, the Roentgen-ray should support the diagnosis. Syphilis, of course, is the only other alternative, but the *Wassermann* reaction and specific treatment are in this case the distinguishing features, unless both diseases co-exist.

When the cranial bones are the home of the pains we are very likely confronted by diffuse or tumorous *hyperostosis.* I shall speak of the differential diagnosis later on. I will mention, however, that, when the jaw bones are affected we must differentiate between maxillary hyperostosis and syphilis as well as neoplasms of the bones, chiefly sarcoma.

Fibrous ostitis is in most cases a generalized disease of the bones, but in the circumscribed form it prefers the long bones for the place of attack. Spontaneous fractures are frequently a telling factor. The X-ray reveals their nature (cyst formations).

There is a localized form of *rachitis tarda* of which I shall speak later on. I will only mention here so far as the diagnosis is concerned, that the Roentgen-ray is the most reliable item.

We should look for localized changes in the bones of the leg and the spinal column (genu valgum, pes valgus, kyphosis and scoliosis of the spinal column, coxa vara in the adolescent). Other symptoms are paleness of the face, abnormal fatigue and arrest of physical growth.

Other unilocular affections of the bones such as syphilitic, actinomycotic or tuberculous ostitis, and scleroma, will be discussed when we come to the multilocular conditions.

Greater difficulties surround the diagnosis when the pains are localized not only in one place, but are spread over various portions of the body. The patient locates them deep down in the bones, or he may only complain of "rheumatic" sensations. We should resort to the usual pressure and percussion methods. If we find local sensitiveness we have several problems to solve. The alternatives are: acromegaly with pains of minor importance; multiple osteomyelitis; various forms of multilocular ostitis (syphilis and tuberculosis); osteomalacia; osteoporosis; rachitis tarda; metastatic and primary neoplasms; *Paget's* ostitis; diffuse hyperostosis.

It may help the student of this book very much if he ever keeps in mind that he is dealing with a disease of the bones when the patient complains of periodic, or constant pains, now of a decidedly boring, tearing or shooting character, or of a vague, indefinite, rheumatic nature.

First in line is *acromegaly*. The differential diagnosis will be found at the end of this chapter, and its initial symptoms have already been discussed in the chapter on "Pain in the Extremities." The diagnosis is not hard to make when the disease is fully developed. The outstanding signs are: the progressive enlargement of the skull, the nose, the lower jaw, the lips and the tongue, the hands and feet and of the overlying parts. We also find marked spacing between the teeth, sexual impotence, atrophy of the genitals. Further, the enlargement of the hypophysis causes local symptoms such as, exophthalmos, paralysis of the optic muscles, visual impairment, bitemporal more frequently than homonymous, morbid changes in the optic nerve (reactions of the pupils, atrophy), cerebral pressure symptoms (headache, dizziness, weak memory, apathy), sleeplessness, moodiness, depression, enlargement of the larynx, abnormally deep, raucous voice, rigidity of the chest, kyphosis and cuplike dullness over the upper sternum. The X-ray will reveal the conditions in the sella turcica (abnormal dimension in the sagittal direction). Diffuse hyperostosis and *Paget's* ostitis are closest in resemblance.

The differential diagnosis of osteomalacia will be more fully discussed at the end of this chapter. The pregnancy (puerperal) form is easy to diagnose. When a pregnant or parturient

patient complains of rheumatic pains be on the watch for a case of osteomalacia.

The characteristic symptoms are: the pains are localized in the pelvic region near the last vertebra and gradually extend into the trunk and thighs, rarely into other extremities, scarcely ever in the head, later on they increase in violence during the menstrual flow and in subsequent pregnancies; wattling gait, clumsy movement of the body, spasms of the adductors, increased patellar reflexes, tenderness in the bones, especially the ribs, abnormal flexibility of the bones (feathering pelvis, feathering ribs, etc.), unusual softness and deformities (osteomalacia pelvis, kyphosis), indentation, especially of the lumbar vertebræ and subsequent shrinkage of stature, the dimensions of the body are out of proportion to the length of the legs, the costal arches almost touch the upper pelvis, causing a deep furrow in the waist line, lateral bending of the ribs, arching of the sternum (chicken breast) or depression of it, later on bow-leggedness, infractions or fractures on impacts ever so light, slightly brisk muscular action and surprising fluctuations in the intensity of the morbid symptoms.

Tardive or senile osteomalacia occurs also in men. It differs from the former in many ways, viz., it chiefly begins in the bones of the trunk. There are pains in the ribs and pains and de-

formities in the vertebræ (arcuary kyphosis), depression of the chest, sensitiveness of the spinal column on pressure, a strong compression of the thorax is painful, girdle sense, pains and changes in the legs and thighs, the long bones are flexible but do not break easily, muscular contractions especially of the adductors, knees and thighs are closely pressed together when standing or walking (in decubitus contracture of the adductors is not demonstrable) and shrinkage of the body. Excruciating pains accompany this tormenting disease through many weary years.

Hunger-osteopathy presents pretty well the same picture.

Senile osteomalacia is easily mistaken for *osteoporosis.*

In the latter the bones are very brittle and do not bend. Spontaneous fractures of the femur, the ribs, clavicles, sternum and spinal column without conspicuous accompanying symptoms are frequently found in Roentgengraphs. Moreover, osteoporosis as a rule involves the entire skeletal system including the skull and maxillæ, but shrinkage of the body, waiste-furrow and wattling gait are not found.

The differentiation, however, of senile osteomalacia from multiple tumors of the bones is much more difficult. Here we have a true picture of "carcinomosteomalacia."

In juvenile patients the question lies between osteomalacia and rickets, if a differentiation between the two diseases is at all possible because the pains in both diseases are almost identical.

Tardive rachitis runs its course from the eighth year after birth till the time of puberty. The pains are chiefly in the back and the legs and there are rosary-like tuberosities on the antero-external ridges of the bones and on the epiphyses, and we observe scoliosis and genua valga and vara very much the same as in osteomalacia. Differential points may be found in several respects such as the juvenile age of the patient, likelihood of infractions, absence of shortening in the spinal column and in the thorax, want of pressure sensitiveness in the chest and pelvis excepting the extremities, pelvis normal (not beaked, rostrated), involvement of the teeth, retarded growth. The remaining requisites for a positive diagnosis are the Roentgen ray and urine and blood tests.

Pains in the bones, physical shrinkage and spinal curvature have in recent years also been observed in osteoporosis attended with chronic *bilious fistulæ.*

The differential diagnosis between *senile* and *carcinomatous osteomalacia,* or more correctly speaking *multiple metastases of the bones,* can only be in doubt when there is no evidence of an existing primary neoplasm. In that case

pains, especially in the thoracic bones excerbated by motation, are common factors. This refers also to conditions which prevail in cases of osteoplastic neoplasms (osteoplastic new growths are recognized by the increase of body weight despite progressive cachexia). We find brittleness of the bones, deformities in the trunk due to columnar kyphosis and also in the sternum, the ribs and thighs, kyphosis in the lumbar portions, changes in the formation of the pelvis and of the proximal parts of the femur and shrinkage of the body the same as in osteomalacia, infractions and fractures, remissions and intermissions and a long protracted illness. But if we can feel a protuberance even in one part of the bone only and not of the nature of a callus—the crista ilei, the ribs and the skull (hypernephroma and carcinoma) are favorite spots—the diagnosis points to an existing tumor. When this sign is wanting, the Roentgen-ray and blood tests will probably furnish the required proofs.

In osteomalacia the blood test as a rule shows only normal conditions. But in multiple metastases there are signs of abnormal activity in the bone marrow, i.e., leucocytosis in the form of polynucleosis and myelocytosis, nuclear red blood corpuscles. Later on regeneration of the marrow ceases, the marrow elements are wanting in the blood, whole colonies of atypical marrow cells are found in it. A careful X-ray exam-

ination of the whole bony framework should be made in all cases. The differential diagnosis will not be concerned with these points when multiple osseous metastases are due to an ordinary, benign, adenomatous or insignificant struma. But note, that a malignant struma may manifest the clinicl symptoms of hyperthyreoidism.

The pains caused by these metastases are particularly distinguished by their fleeting, capricious character: today they are felt in one place, tomorrow in another; now they torment the patient only to vanish again later on. This often enough leads to the erroneous diagnosis of rheumatism, gout or hysteria, especially in cachectic or nervous persons. Leucocytosis demonstrable in the blood is of great help in the diagnosis, although in many cases the blood picture is quite normal. I again refer the reader to the necessity of a studious X-ray examination of the patient.

Sarcoma and principally hypernephroma are further instigators of multiple metastases of the bones with and without pain and pulsation. If they are present they may even arrive before the tumor becomes manifest. If a local swelling already exists on the bone, the diagnosis is made the easier, although mistakes are not always avoidable.

In hypernephroma the metastases may again

be of a truly fleeting character, rheumatic-like as it were, changing their locality constantly, before the clinician is enabled to recognize a change in the bones or a primary tumor in the kidney. Radiology is a conditio sine qua non here also.

The presence of a tuberculous hearth in the organism is also likely to mislead the attending physician in his diagnosis, but let us remember that it is very risky to believe that, because there is a tuberculous condition in one organ, morbid conditions in another organ of the same body must of necessity also be of a tuberculous character.

There are other proliferations attributed by some authorities to affections of the bones, by others to systemic diseases, which cause pains, similar to those described in the foregoing pages. In most of these cases the complaint is of very severe pains in the nape of the neck, in the chest, the back and shoulders, sometimes also in the limbs. They are sharpened by physical movements and come on in periodic attacks. There is local tenderness in the spinal column or in one rib or another. Later on softness of the bones sets in, deformities in the upper spine and in the thorax generally, together with kyphosis are found. The sternum becomes prominent owing to angular curving of the ribs through multiple spontaneous fractures. The chin is pushed down

on the chest as in senile osteomalacia with which *multiple myeloma of the bones* has so many other conditions in common. This latter disease chiefly affects the cranial bones but avoids those of the pelvis and of the extremities.

The differentiation is not difficult so long as protuberances on the affected bones, or multiple tumors on the cranial bones (erroneously also called atheroma) are in evidence. When these are missing, doubts may arise. But the X-ray and a test of the urine for *Bence-Jones* albumin which is hardly ever present in osteomalacia, will clear the situation. Moreover, in osteomalacia the extremities are nearly always curved when the trunk is markedly affected, whilst in myeloma the changes in the trunk and skull are much less pronounced. If fever is present it speaks for myeloma.

Whether we are confronted by a *primary myeloma* or a *secondary metastatic carcinoma* of the bones when we find a primary organic neoplasm in the place mentioned before, the diagnosis is self-evident. The presence of *Bence-Jones* albumin points to myeloma although this albumin is also found in multiple metastases of the bones. When the pains are felt only in the extremities or less markedly so than in the thoracic region we have little reason to think of myeloma. The condition of the blood should be of some help as in myeloma it generally

shows only anemic, rarely leucemic conditions, although myelocytes are sometimes found also. It is further of importance to remember that, clinically speaking, demonstrable metastases in the internal organs, including the lymphatic glands—unless they are highly regional—never originate from myelomata. The presence of such metastases, especially in the glands, consequently favors the assumption of secondary metastatic neoplasms. I do not wish to contend, however, that a post mortem may not reveal analogous growths in the bones in cases of myeloma.

On the other hand we come across, occasionally, *primary,* multiple, benign or relatively benign (osteoma, osteochondroma, echinococcus cysts, or cysts in the bones in fibrous ostitis) and malignant tumors of the bone marrow, e.g., in the vertebræ, ribs, thighs, upper arms, either in the shape of osteosarcomata, lymphosarcomata or giant cell sarcomata. They are all accompanied by pains in the bones and by anemia, cachexia, if not by recurrent fever. The *Bence-Jones* albumin may be found in this disease and its presence be taken as a sign of myeloma.

Painful swellings are also found in typical leucemic *lymphadenitis* and thus simulate a myeloma. The clinical aspect and the condition of the blood should suffice for a proper diagnosis.

Syphilitic or tuberculous multilocular osteo-

periostitis are here within the reach of possibilities.

In recent *syphilitic periostitis* and true *gummatous osteoperiostitis* we find new growths in the form of painful, if not painless, round tumors of various size. They are more or less hard, i.e., of a fluctuating consistency and mostly situated on those bones which are less protected by tissue, I mean the skull, the forehead, the clavicles, the sternum, the ribs and the inner side of the tibiæ. The diagnosis is not hard to make because the usual symptoms of secondary syphilis are always manifest, not to speak of their nocturnal character. And again, in recent cases of osteoperiostitis we find very little, if any, local reddening of the skin, unless it is due to some mechanical irritation; we also can notice that the swellings are flat in shape and gradually recede, while in gummatous ostitis necrosis, ulceration and subsequent scarification of the bones obtain. It is worth while to remember that gummatous periostitis manifests itself in the form of a diffuse and not circumscribed infiltration which breaks down in an ulcerous state. Minor flat and far-reaching enlargements of the bones are also demonstrable. Likewise that gummatous ostitis, e.g., in the clavicles, or in the fingers or forearm is apt to lead to spontaneous fractures. On the long bones and on the skull it may cause proliferation and subsequent thickening of the

bones, on the skull also hyperostosis or leontiasis ossea.

Secondary syphilis does not as a rule cause swellings in the bones, but rather transient, recurrent inflammations and moderate, not strictly localized and wandering pains. The patient is wont to call them "rheumatics." The fact that they are sharper in the night time, other characteristic symptoms and the *Wassermann* reaction should influence the opinion.

In recent syphilitic osteoperiostitis we find the same fluctuations in the body temperature that are characteristic of secondary luetic conditions, while in the gummatous form a normal degree prevails unless pus cells are present.

If children are affected in this manner hereditary syphilis suggests itself. In hereditaria tarda the first four to five years in the patient's life pass by without morbid symptoms. It is at a later date, sometimes even beyond the stage of puberty, that the characteristic manifestations on the bones make their appearance. The history of the case, *Hutchinson's* teeth, saddle nose, remnants of former peranchymatous keratitis, partial deafness, chronic swellings of the glands and knee joints, are all typical road signs, especially if the *Wassermann* is not neglected.

Tuberculous ostitis and also *osteomyelitis* may affect one or more bones at the same time. In both rising temperature is usual.

In the first named disease fever is moderate, if not absent, although there is local tenderness, the pains are not severe, but there are functional disturbances, for instance, if it attacks one of the lower extremities the patient is forced to limp. Its tuberculous genesis may be recognized from a tuberculous state seen in other organs, from the nature of existing tumors which are of a livid color, the thin, cheesy pus, the slack, yellowish granulations of the fistulæ, and last but not least, from the Roentgen picture. It is of interest to know that this disease has a preference for the small hollow bones such as the metacarpal, metatarsal and phalangeal bones (spina ventosa). In the larger bones it settles rather in the diaphyses, rarely in the epiphyses, which fact distinguishes it from *acute purulent osteomyelitis.*

With the assistance of laboratory blood tests and the Roentgen-ray the diagnosis of this disease can be made from the following symptoms: the initial stages show chills and high fever, a marked general indisposition, diarrhea, typhoid state, soon to be followed by severe pains around the heads of the affected bones (shin bone, thigh, upper arm, sometimes the lower arm, clavicles, ribs and the short hollow bones), local reddening of the skin, higher local temperature, collateral edema, later on septico-pyemic generalized infection. While tuberculous ostitis may be of

an acute character with high fever throughout its course, purulent osteomyelitis is generally subacute or chronic in its nature and the patient suffers from rheumatoid pains in the affected parts, and even from minor functional disturbances, for many a year. When a differential diagnosis from neoplasms, syphilis or tuberculosis tests will furnish the required information. Marked leucocytosis points to osteomyelitis; in tuberculous ostitis a mixed infection does not exist.

Albuminous periostitis is, so to speak, an intermediary disease between purulent osteomyelitis and tuberculous ostitis. It sets in gradually with little or no fever, is mostly chronic in character (sometimes acute in the beginning with fever) and localized in the same bones as in acute osteomyelitis and also at the ends of the epiphyses, sometimes associated with osteomyelitis in other bones, local, well defined, elastic, fluctuating swellings which secret a stringy, albuminous, synovia-like fluid between the periosteum and the coating of the long bones showing traces of tubercle bacilli or strepto- or staphylococci.

I add two relatively rare forms of unilocular or multiple acute or chronic osteoperiostitis, viz., acute or chronic *osteoperiostitis malleosa* and *actinomycotica.* The former is easy to recognize if it occurs in a case of positive glanders, because

it is merely a manifestation of pyemia of the bones. In chronic farcy the diagnosis meets with some difficulties. The salient features which require consideration are: does the patient come in contact with horses or other animals subject to glanders, the presence of typical sores in the nose and skin, of abscesses in the muscles or in the periarticular tissue (a subacute or chronic-exudative articular rheumatism might be simulated by the latter condition), sub-febrile or slightly febrile temperature, a positive diazo-reaction and bacteriologic findings.

Actinomycotic osteoperiostitis or *osteomyelitis* is mostly of a secondary character and arises from a continuity in a vertebra, rib or in the sternum, but it may also make its appearance in a long bone, e.g., in the thigh after a trauma, with local swelling and all the symptoms of an acute osteomyelitis. When it runs an acute course it is very likely of metastatic origin.

Typhoid osteoperiostitis generally sets in during the period of convalescence from typhoid, principally in the tibia, also in the ribs, the sternum, clavicles, etc. It may be purulent, but is as a rule reconstructive in character. The etiologic diagnosis may meet with difficulties if the symptoms have been overlooked or the primary infection is of very old standing. Blood tests are necessary.

Malta fever may also produce suppuration of

the bones and an abscess in a dependent part. The diagnosis depends on the possibility of an infection, the examination of the pus and the presence of the micrococcus militensis.

Acute *articular rheumatism* may cause the formation of various forms of periostitis, for instance, the initial acute rheumatic form, in which very severe pains, swelling, reddening of the skin and high fever are noticed.

Paget's ostitis must again be mentioned. In the beginning it is an individual disease of the skull, or tibia, femur or spinal column, but has also been observed on the clavicle and ribs. It is a painful, sometimes, however, indolent affection of the bones which gradually involves the whole thoracic frame if not also the pelvis.

The patient complains of rheumatoid pains in the affected bones strongly influenced by weather conditions, difficulty and clumsiness in walking. The progress of the disease is assymmetric throughout. The head grows larger and larger and leans forward until the chin touches the chest, the cervical and thoracic vertebræ become kyphotic, the thorax looks shortened with the lower part expanded and separated from the abdomen by a deep furrow, the extremities are curved, the knees approximate each other, and the affected bones are abnormally thick, although motation is seemingly little impaired. The X-ray materially assists the diagnosis.

We will now consider the differential diagnosis between *acromegaly, Paget's ostitis* and *hyperostitis,* since each of them involves the enlargement of the head.

Acromegaly is distinguished not only by the unusual expansion of the head, but also by the symmetrical, uniform enlargement of the arms and legs (bones and tissues as well) and abnormal bulkiness of the hypophyses. Mark the symmetrical progress of the disease as against the asymmetry prevailing in Paget's ostitis, and again that in hyperostitis the distension of the head is diffuse and not tumorlike. But when it is tumorous the various growths are concatinated, but each prominence has its own individual circumference; moreover, they attack in the first line the bones of the face, whilst in Paget's ostitis these remain unmolested or nearly so; furthermore, Paget's ostitis presents a smooth surface all over the growth. Besides, in hyperostitis we find exophthalmos and paralysis of the cerebral nerves, which is not the case in the other disease. But this latter nearly always involves the bones of the lower extremities, quite an exception in hyperostitis, which, moveover, occurs chiefly in youthful individuals when Paget's ostitis prefers old age.

Gummatous ostitis may be confused with Paget's ostitis because it also leads to scleroting conditions which would enlarge the head con-

siderably. If it is of syphilitic origin it can easily be separated from Paget's ostitis by the typical symptoms and the *Wassermann* reaction, unless both are of a luetic character.

There is a peculiar aspect to *Paget's ostitis,* viz., very often the *neoplasms* of different localization, e.g., endothelioma of the pleura, gastric or hepatic carcinoma, likewise neoplasms of the bones, such as sarcoma of the tibia, enchondroma of the pelvis, are associated with it. Thus we may be tempted, when we see the patient only in the last stadium of the disease, to ascribe the whole symptom complex direct to the neoplasm and its metastases. But the anamnesis and relatively long duration of the affection, the well-nourished physical appearance of the patient and his fit condition, also the smooth surface of the enlargement all over the head and the curves in the lower extremities, together with the Roentgen picture should prove sufficient evidence insofar as the diagnosis is concerned.

To differentiate Paget's ostitis from *osteomalacia senilis* is not always such an easy matter, because in both very intensive pains are endured and in both the body shrinks in height. But in osteomalacia the thorax is principally involved, while the head and the lower extremities, especially the long bones, remain intact, or if the bones are at all affected the changes are symmetrical in form, while in ostitis the progress is

irregular and asymmetrical in form and the head is very much deformed. Here again the X-ray shows the differentiation in a marked fashion.

Furthermore, a differential diagnosis may have to be made between *fibrous ostitis* (*Recklinghausen's disease*) and the two aforementioned affections on account of the intensive "rheumatic" local pains and the bending of the bones. In *Recklinghausen's* disease we can notice fibrous changes in the bone marrow, with spongy alterations in the bone tissue and the formation of giant cell sarcomata and spindlelike swellings around the metaphyses, along the long bones, with parchment crackling and fluctuation, also infractions, if not fractures, of the bones. We must resort to the Roentgen-ray once more.

I must refer here to still another disease which I have purposely avoided to mention thus far because it is in reality not a primary affection. I mean *"osteoarthropathie hypertrophiante pneumique"* with drumstick fingers and its intermediary form, i.e., *Bamberger's disease* with osteoperiosteal enlargements of the long bones, especially at their distal ends in the thighs and forearms.

I confine myself to the two principal forms.

Bamberger's combination type is characterized by drumstick fingers and parrot-beak nails, spontaneous and sensitive swellings in the distal

zones of the forearms and the lower thigh bones. Around the radius and malleolus we find distinct enlargement and the middle of the hand appears plumper. The X-ray and anatomical examination show still more changes in the shape of inflammatory periosteal growths.

In osteoarthropathie hypertrophiante the fingers are shaped like drumsticks whilst the hand itself is shaped more in the form of a paw, the feet are very much deformed, the whole distal surroundings of the lower thigh and forearm are swollen, and similar enlargement may be observed in the sternum, the ribs, clavicles and pelvis. Kyphoscoliosis also accedes in some cases.

From this short sketch it will be seen that osteoarthropathie hypertrophiante—I shall speak about its diagnostic importance later on—possesses a certain resemblance to *acromegaly*. Yet a mistake between these two diseases can be easily prevented. I will not speak here of the secondary nature of osteoarthropathie (see later on about drumstick fingers), but only of other phases which distinguish this disease from acromegaly. In the former the fingers are shaped like drumsticks and the nails like a parrot's beak; in acromegaly they are harmoniously enlarged in every dimension, though the finger nails are comparatively small. In osteoarthropathie the hands and feet are misshapen like

paws; in acromegaly they are enormously, but symmetrically, enlarged, not deformed. The bony deposits and protuberances on the distal ends of the long bones so typical of osteoarthropathie are missing in acromegaly. The swelling of the overlying tissues, the enlargement of the hypophyses and all the other characteristic symptoms of acromegaly are missing in osteoarthropathie. A combination of the two diseases has been reported in one case.

There is a case of *bone disease* on record which is *ascribed to intestinal troubles.* The patient suffered since early childhood, but with periodical interruptions, from diarrhea, apparently due to a toxic decay of albumin in the bowels. By avoiding meat and other albuminous foodstuffs the patient was cured. What is of interest from the clinical standpoint is the fact that the symptoms had much in common with acromegaly and *Paget's* ostitis. At times they simulated those of Marie's osteoarthropathie hypertrophiante, or showed rachitic characteristics. The patient complained of abnormal fatigue, and at times of pains in the bones and joints. Swellings, enlargements and deformities were also observed in the bones.

There are a few other affections of the bones in which local pain is a prominent symptom. As they are of minor significance for the differential diagnosis I will only mention them. *Noth-*

nagel's lymphadenia ossium is not as yet properly classified, although it seems to bear the character of a leucemic lymphomatosis. *Bruck's disease* is typified by enormous contortion in the bones and frequent fractures thereof, also by multiple ankylosis of the joints and by muscular atrophy. Then there is *Ziegler's osteotabes infantum,* and another childhood disease, i.e., *osteogenesis imperfecta tarda,*

Some of the morbid conditions mentioned in the foregoing pages, such as acromegaly, are in reality not primary affections of the bones; and there is still a number of others belonging to this category in which likewise pain in the bones plays a significant rôle, although a primary disease of the bones does not exist. Among them are the following:

In *degeneratio adiposo-genitalis* arising from hypopituitarism, periodic spontaneous pains in the ribs and extremities are felt, although from a diagnostic standpoint they are not of the same importance as in acromegaly.

Such local spontaneous pains are not uncommon in *scurvy* in the adult, when hemorrhages set in between the bones and the periosteum, and in *Barlow's* disease (a combination of scurvy and rickets). The children cry out loud upon being merely touched.

If the patient complains of so-called "rheumatic" pains which increase in violence when he

moves about or is dressing, it is a reminder of a primary disease of the blood or of the blood-producing organs, viz., acute or chronic myeloid *leucemia, aleucemia* and *myeloid chloroma,* but also (in a minor degree) *lymphatic leucemia,* in which, as a rule, only sensitiveness on pressure prevails in the bones. *Pernicious anemia* (anemia aplastica, thrombopenia) and *Jack's* disease (see "Articular Pains") deserve a place, too. The subjective sensation is here restricted rather to a dull sensitiveness in the bones similar to that in some cases of chlorosis, but local pressure or percussion of the bones elicit most intensive pain. A combination of myeloid leucemia and osteomalacia is on record.

Pains in the bones and accompanying swelling of the lymphatic glands which occur in *extirpation of the spleen* are, no doubt, due to over-activity forced upon the bone marrow by the missing splenic function.

I will add here an observation from my own practice which may interest the reader. I have seen girl patients, before and during the stage of puberty, who complained of abnormally quick fatigue and also of pains in the joints or in the tibiæ, with pronounced tenderness on percussion in the bones, and even in the thighs. I could find no other explanation than an *abnormally quick development of the body.* This physical growth seemed to come on in regular spells,

one after the other in quick succession, and with each the pains in the bones would arrive, only to disappear when nature had finished its task. Noteworthy changes in the condition of the blood I was never able to observe.

Pains in the bones, particularly in the frame of the chest, and very hard to distinguish from intercostal neuralgia, are often enough part of the symptoms in the *climacterium.* If we consider the age and sex of the patient, the waning function of the ovaries, the general gynecological conditions and other climacteric disorders, above all vasomotoric disturbances (flushing of the face, sweats, paresthesias of the extremities, swooning spells, palpitation of the heart, anginoid attacks), accumulation of adipose tissue, *Heberden's* nodes, etc., the diagnosis could not go amiss, and yet it will be wise to keep a sharp eye on the possible development of a tardy osteomalacia.

The *Wolhynian,* or *five days fever,* which became common during the World's War, requires some attention here. Both names are badly chosen, because it did not exactly originate in Wolhynia, neither is it recurrent in five-day periods; only in rare cases so. Still it has the character of recurring four or five times at irregular intervals, and is generally associated with a tumor of the spleen, and also with osteo-periosteal neuralgic pains.

Tibial pains are observed in *abdominal typhoid, influenza* and *spotted typhus.* They may even degenerate into generalized pains of the bones, as is also the case in paratyphoid and recurrent fever, and sometimes in typhoid vaccination. When they occur in *erythema nodosum,* a wrong diagnosis of osteomyelitis may result.

If spontaneous pains arise in *gout,* they are of a minor degree, but very severe on percussion, especially on the condyles of the humerus, the head of the radius, in the os calcaneum, and in the small tarsal bones, but not more acute in the night time.

Certain *exogenous poisons* may cause pains in the bones. I do not refer to the phosphorous necrosis of the lower jaw or of the nose caused by chromic acid poisoning (the latter resembling a syphilitic defect of the septum), but to acute intoxications from arsenic and phosphorus. Although the pains are rarely spontaneous, pressure and percussion evince them sharply in the bones, chiefly in the ribs, the vertebræ and the tibiæ, and according to my own experience, constitute a frequent and diagnostically most important symptom. They are, no doubt, due to overactivity of the bone marrow which, in acute poisoning from phosphorus, manifests itself in the form of severe polycythemia and leucocy-

tosis with an increase of the bone marrow elements.

In mercurial poisoning similar pains are observed. This is interesting from the standpoint of the differential diagnosis in syphilis and mercural intoxication. *Chronic* arsenic poisoning belongs here, also. Lancinating pains in the bones are not uncommon in *tabes.*

Pains which in their nature are *not spontaneous,* but *provoked by pressure or percussion,* are of diagnostic value in a series of affections such as: *diseases of the blood, leucemia, aleucemia, pernicious anemia, chlorosis,* acute hemolytic *anemia, v. Jacksch's* disease, various forms of severe *secondary anemia,* especially after neoplasms (red bone marrow), some cases of *chronic osteomyelitis,* and all the initial stages of the aforementioned disorders. In some cases of *Basedow's disease* I have been able to observe in the bones a very marked sensitiveness on pressure or percussion when a demonstrable anemia or overstimulated activity of the bone marrow were not present, not even when a complicating osteomalacia set in or signs of abnormal softening of the bones were in evidence.

In *pulmonary emphysema* the pains are of a rheumatoid nature, especially in the bones of the lower extremities. The tibiæ are most sensitive on pressure and percussion. The pains are

unmistakably due to periostitic growths at the distal ends of the tibiæ, and remind one of the osseous changes described by *E. Bamberger.*

In certain *infectious diseases,* sensitiveness on pressure and percussion (sometimes rather vague and quite indefinite), and also pain in the bones, will be found a very valuable asset in the diagnosis. This is particularly so in *generalized septic conditions* (septic endocarditis) and in *scarlet fever.*

Pains in the bones belong also to the prominent prodromal symptoms of *secondary syphilis,* even when the patient is not affected with luetic periostitis. There are no local changes in the bones and the pains disappear with the cure of the exanthema.

If in *abdominal typhoid* pain and tenderness in the bones are experienced, it may be the expression of an existing acute osteomyelitis, the more so when we find a local edema or vivid coloring of the skin and lymphangoid striæ.

In *malaria* pain and tenderness in the bones are often very molesting symptoms. They prevail in the long bones (tibiæ in particular), likewise in the cervical and upper thoracic vertebræ. But they carry also another very important message. We come across cases in which the patient may have suffered from some kind of fever, but to the best of his knowledge has never had an attack of malaria before or has not dwelt

in a malarial district, a fact, which would remove the suspicion of a possible tumor in the spleen. Moreover, we find no traces of other morbid conditions (unless it be a minor, irregularly recurring rise in the temperature) and no plasmodia or pigment (but mononucleosis) in the blood. But pressure and percussion elicit intense pains in the bones, especially in the tibiæ, and a week or two later we are confronted by a typical attack of fever, perhaps, superinduced by the electric current, or a supervening cold or by radiation of the spleen. In such a case we are justified to assume that the plasmodia lay hidden in the spleen and bone marrow and have only now entered the blood. It is my firm conviction that a definite cure of malaria can never be claimed until every vestige of pressure pain in the bones has disappeared.

A similar sensitiveness of the bones is also demonstrable in malarial *cachexia.* It is due to anemia and changes in the bone marrow. The same conditions prevail in carcinomatous cachexia and in tuberculosis.

It should not be difficult to learn from the foregoing dissertation that the differential diagnosis of "rheumatoid pains" leads us through a very wide field of internal medicine. We must not only take into consideration the rheumatic and rhematoid pains of the muscles and joints, the morbid conditions of the skin (adipositas

dolorosa, neurofibromatosis, sclerodermia), a multitude of diseases and their localization in the muscles, in the fascia and in the ligaments, in the joints and in the bones (gout), affections of the arteries and veins (sclerosis, arteritis, phlebectasis), neuralgic, chronic neuritic, neurotic and lancinating pains and painful rheumatic sensations which originate from diseases of internal organs, but also ailments of the skeletal organism.

Acromegaly, primary and secondary neoplasms of the bones, myeloma, osteomalacia, osteoporosis, fibrous ostitis, *Paget's* ostitis, deforming, recent syphilitic and gummatous ostitis, chronic purulent osteomyelitis as well as *Marie's* osteoarthropatie hypertrophiante pneumique including *Bamberger's* subdivision, all are irritating factors of rheumatoid pains.

Osteopsathyrosis, the symptom of brittleness of the bones, is observed in osteoporosis, osteomalacia and rickets, in primary and secondary metastatic tumors of the bones (we must add enchondroma, sarcoma, echinococcus cysts and those of the bones), myeloma, deforming, fibrous and gummatous ostitis and *Bruck's* disease. It occurs also in rare cases of chronic osteomyelitis and tuberculous ostitis. Diseases of the central nervous system such as tabes, syringomyelia, poliomyelitis, progressive paralysis and mental disturbances may through minor traumatic con-

ditions lead to spontaneous fractures. The intermediary cause here would be some trophic disturbance or a simple atrophy of the bone. *Basedow's* disease and old age are likewise causative factors of osteopsathyrosis. But there is also an independent form of this disease which is called *idiopathic osteopsathyrosis* which is either congenital in its nature or at any rate acquired in childhood. It may be hereditary (familial), but there are also solitary cases on record. The bones are of very soft consistence and frequent fractures ensue which cause abnormal curvatures and deformities. Hereditary cases are easy to diagnose. In acquired cases we must look for rickets or juvenile osteomalacia. Syphilitic affections of the ephiphyses must also be considered. The absence of syphilitic symptoms and the X-ray will determine the differential diagnosis, especially when the fractures are located in the shaft of the bone between the epiphyses and not where the diaphyses and epiphyses meet. The X-ray also shows in syphilis a swelling of the epiphyses, while in idiopathic osteopsathyrosis there is only an apparent thickening, a proof that the epiphyses are normal. This thickening is caused by an atrophy of the diaphyses through pericostal dysplasia.

Localized *isolated* or *multiple swellings of the bones* may be observed in various morbid conditions, such as an ordinary callus, the different

forms of acute or chronic infectious and non-infectious osteoperiostitis (purulent or otherwise), primary and metastatic plasmata, myeloma and tumorous hyperostosis.

Sporotrichosis is another malady which leads to pericostal and osseal abscesses. It is not hard to diagnose it. We find multiple papulous, or vesiculous and pustulous manifestations in the skin and mucous membrane, also muscular infiltrations thus presenting very strongly the features of tertiary syphilis or tuberculosis. The microscopic examination of the pus showing absence of bacteria and the agglutination test will give ample proofs of *Schenk-de Beurman*'s sporotrichosis. Prompt reaction to iodide treatment is another good test.

Chloroma is a disease which often causes the formation of tumors of the bones, especially on the cranium and on the sockets of the eyes. At the present time it is recognized as a subspecies of lymphatic myeloid leucemia and is distinguished by the green tint of the distended tissue. We encounter chiefly subperiosteal growths composed of lymphatic tissue especially on the skull or in the orbita, sometimes in the temporal region or around the cheek bones. Exophthalmos and constriction of the optical nerve are frequent complications when the tumor is localized near the visual organs. The diagnosis must be based upon the following symptoms: generally simul-

taneous swelling of several lymphatic glands and of the spleen; regular, perceptible aggressiveness of the tumor on the surrounding tissue (nerves and muscles), penetrating even into the spinal canal; the condition of the blood which strongly resembles that in lymphatic or myeloid leucemia. Myeloid leucemia may change into a myeloid chloroma when the proliferations suddenly assume an aggressive character and reach out from the clavicullar fossa not only into the spinal canal, but also into the face, the lower jaw and the oral cavities. The tonsils, in fact, the entire mucous lining of the mouth look as if they were dyed a vivid green, even the blood serum shows that color.

In rare cases of *lepra* swellings of the bones (lepromata) are observed preferably on the phalanges of the hands and feet. They are spindleformed and strongly resemble the spina ventosa in tuberculosis or syphilis. The diagnosis should result from the presence of the lepra bacillus in the nodules of the skin, or from the appearance of erythematous, hyperesthetic areas on the skin, from the existing leprous neuritis and the synchronous affection of the mucous membranes of the nose, the mouth, the palate and the porterior oral cavities.

When we see fingers formed like drum sticks, or nails on hands and feet shaped like the beak of a parrot, or like the crystal of a watch, we

naturally ask the question: "What is the originating cause of these deformities?" The answer to this question will also solve the problem of the occurrence and clinical significance of *Marie's osteoarthropathie hypertrophiante* and *Bamberger's disease* because the etiologic genesis is the same in these three affections. In the majority of cases the underlying cause is a prolonged purulent condition of the respiratory tract such as: cavernous tuberculosis, chronic induration of the lungs with dilatation of the bronchi, gangrenous pulmonary abscess, gangrene of the lung and empyema. Drum stick fingers occur also in cystopyelonephritis and in dysentery. For the differential diagnosis it is worthy of mark that the deformity of the fingers is of a specific type in tuberculous diseases. In non-tuberculous affections, e.g., in bronchiectasy the drum stick form is very conspicuous and the ungual phalanges are short and thickened and spherical, while in tuberculosis the latter are longer, narrower and bent more like a hook. But the difference is not always so pronounced.

Analogous misshapen fingers are also observed in chronic actinomycosis of the lungs. A proper diagnosis cannot fail to result from the bacteriologic examination of the sputum and the likely discovery of a purulent tooth.

After pneumonia or non-purulent pleuritis and also in influenza similar disfigurements of

the fingers and nails may be seen, mainly in the tissue and not so much in the bones. This is also the case in chronic icterus, biliary cirrhosis, chronic alcoholism, cholelithiasis, liver abscess, congenital or protracted syphilis, recurrent articular rheumatism, dilatation of the stomach, tuberculous peritonitis or severe chronic dysentery even without stenosis or intestinal stagnation.

Diseases of the heart (especially congital defects in children), acquired vascular affections of this organ, chiefly in sclerosis of the pulmonary artery, intrathoracic tumors (carcinoma or sarcoma of the lungs), mediastinal tumors (lymphosarcoma or granuloma), rachitie deformities in the thorax, tumors of the parotis or carcinomata in the posterior pharyngeal cavities belong to this category. In some cases of intrathoracic tumors, especially in aneurysm of the aorta or of the subclavian artery the drum stick formation is noticeable only on one side.

Perhaps we are entitled to conclude that disturbances of the venous circulation are the originating factors of these deforming processes which in other diseases may also be caused by neurogenous influences, for instance, in syringomyelia and neuritis.

Whether in myxedema the affection of the thyreoid gland itself or the concomitant tuberculous condition of the lungs is the causative

element of these changes in the fingers, is a matter of speculation. I know of one case which pointed to a glandular source. It was that of a woman with an infantile uterus who suffered from pains and swellings in the fingers at every menstrual flow but only during this period.

I may add here also sporadic cases of senile osteoporosis and primary polycythemia. In other cases concomitant disfigurations of other parts of the body have been observed, such as enlargements of the nose, of the shoulders or the lips, also diastasis of the teeth, abnormal growth of the hair of the head, glycosuria, polyuria and polydypsia, also loss of the sexual powers. These changes are possibly due to either an anatomical or toxic affection of the hypophysis. The subject is worth further detailed study.

The peculiarly shaped finger nails mentioned above may also be of hereditary origin, familial landmarks as it were.

It is meet that I speak here of certain morbid processes which do not take place primarily in the skeletal system and yet have a certain affinity to acromegaly because their symptoms are to be found in the soft parts of the tissue. But before doing so I will first mention a few anomalies which are also due to abnormal conditions in the bones but entirely devoid of pain. For the purpose of differential diagnosis this might appear to be decisive were it not for the

fact that certain cases of acromegaly develop also without painful sensations or paresthesias.

I refer especially to physiological *giantism.* The differentiation between this and acromegaly offers no difficulty, because giantism is not a disease but an abnormally large development of all sections of the body in physiological proportion. In acromegaly there are only certain parts of the anatomy which are enlarged in an unduly manner, i.e., out of proportion to the rest of the body, by a slow process reaching back sometimes into the very period of childhood. In many cases the X-ray does not show an enlargement of the pituitary body (small adenoma of the hypophysis?). Aside from acromegalic giants we find another kind, i.e., pathological giants, persons afflicted with tumorlike hyperostoses, abnormal curvatures of the bones, e.g., in rachitis, or with hemihypertrophy of the face, or who are disfigured by hereditary syphilis or early in life by a tumor of the testicles. Rare cases as they are, they can by no manner of means offer difficulties in diagnosing. The same may be said of partial giantism or hypertrophy of particular parts of the body. The asymmetrical and unilateral development of the morbid condition and the complete absence of all acromegalic symptoms at once lead us to a positive finding.

Pregnant women—especially in the second

half of the child-bearing period—present sometimes a thickening in the bones of the face and the arms which strongly resembles acromegaly (*pseudoacromegaly*). These swellings recede again in the course of a year or so. We may look upon them as an over-activity of the hypophyses superinduced by the altered function of the female genital glands.

In abnormal growth of the hands acromegaly is apt to be mistaken for *syringomyelia.* In the latter, however, the hand or as the case may be, both hands are really deformed, the skin is thickened, the bones of the phalanges are enlarged, differently though in each finger, the nails are unusually small and shaped like claws.

Moreover, the skin of the palm of the hand is abnormally thick, there are painless paronychiæ or scars or defects in the bones and muscular atrophies together with sensibility disturbances characteristic of syringomyelia are present, while other prominent parts which undergo changes in acromegaly remain intact. Of course, a combination of the two diseases is not among the impossibilities.

Abnormal *lengthening of the fingers* caused by *neurotic,* apparently *vasomotoric* conditions can easily be distinguished from acromegaly.

The condition called *cranium progeneum* in which the lower set of teeth protrudes beyond the upper row may be a partial symptom of

acromegaly of the lower jaw. But it may also be due to other morbid processes. It occurs in cretinism, in mongolism, in degenerates, but also in otherwise quite normal persons. The differentiation from acromegaly should offer no difficulties.

In the child *cretinism* may erroneously be taken for acromegaly as *both have many symptoms in common.* But in cretinism the physical anomalies are accompanied by mental defects and irregularities of speech. Moreover, the majority of these unfortunates have low foreheads, broad, flat noses, wrinkled faces, prominent shoulder bones, deformed knees and legs, pendulous arms, a wattling gait and very thick skins like pachyderms. With the observation of these symbols and the aid of the X-ray the proper diagnosis can run no risk.

There is a certain similarity between myxedema and acromegaly, because in myxedema the face is very much swollen and lumpy and the hands and feet and the tongue are considerably enlarged. The patient complains also of pain in the bones. But it should not be hard to separate the one disease from the other if we consider that in myxedema the edematous nature of the swellings is so palpable. Nevertheless, I will enumerate here some of the distinguishing features: the back of the hands looks like upholstery, the eyelids are swollen, the cheeks

are puffy and pendulous, the whole face has a dull, sleepy expression, mental activity is retarded, the voice is raucous, the skin dry and scaly, the physical movements are clumsy, the body temperature is low and the patient complains of feeling cold and chilly, the hair falls out, there is stubborn constipation, the thyreoid gland is very small, if not missing altogether. If anything else is required the Roentgen-ray will supply the information.

The absence of osseous lesions in *pachydermia* and *elephantiasis* makes a separation from acromegaly easy. So far as *habitus scrophulosus* is concerned it is readily distinguished by the swollen lips, nose and eyelids. In *rachitis* the resemblance to acromegaly is perhaps more pronounced, but there are so many characteristic signs in this disease that a mistake is well nigh impossible.

There is, however, a certain rudimentary form of acromegaly in which "rheumatic" pains in the bones manifest themselves and in consequence should be mentioned here. I may be permitted to call this condition *"acromegaloidism."* The hands and feet and sometimes also the nose, ears and the lower jaw appear very much enlarged, in fact there is a general impression of acromegalic symptoms, the cerebral and genital manifestations are missing. It is not yet definitely known whether the underlying

cause consists of small adenomata in the anterior lobes of the hypophyses or whether we are dealing simply with concomitant manifestations of chronic bronchial affections.

A small, benign, hypophyseal tumor of this kind may very well mask an attack of *neurasthenia* and be accepted as such. The patient merely complains of unusual fatigue and intermittent. headaches. Ocular symptoms are missing and the X-ray reveals no abnormal conditions. But a very careful and painstaking examination may yet disclose lesser enlargements in the extremities, spacing between the teeth, progressive impotence or cessation of the menstrual function.

Further details of the differential diagnosis of *osteomalacia* claim some space in the present discussion, because up till now I have separated this disease only from osteoporosis, myeloma, rachitis and tumors of the bones. It is not necessary to mention that osteomalacia may upon a superficial examination be mistaken for muscular or articular rheumatism, or for neuralgia and even gout. More difficult is its differentiation from *arthritis deformans* in both the hip joints, for difficulty in walking and pain in lateral abduction of the hip joints are common to both diseases. But here again the Roentgen-ray will promptly bring light. Nevertheless, I will say that a careful, slow movement of the hip joint

with the patient in a recumbent position will prove free motility in cases of osteomalacia while this is not so in arthritis deformans. Treatment with phosphoric drugs is also a promising test, although good results with these same remedies have likewise been obtained in deforming arthritis.

Senile osteomalacia settles with preference in the thoracic bony frame, chiefly in the spinal column. In this case the differential diagnosis is a question of *ankylopoietic spondylarthritis.* The former is proved by painful sensations on pressure in other bones apart from the articulations of the thoracic frame and by the X-ray.

We may also be called upon to differentiate between osteomalacia and multiple tertiary *syphilitic affections of the bones,* especially when the pains are both of a subjective and objective nature, with adductor spasms, increased tendon reflexes in the lower extremities, perhaps also ileospasms, and wattling gait. The presence of localized enlargements of the bones, nocturnal pains, unilateral onset or asymmetry of the manifestations, the *Wassermann* reaction, the Roentgen picture and the effect of specific therapeutic measures should prove determining factors.

Diseases of the nervous system must also be considered in this connection. Likewise affections of the *spinal cord* such as *sclerosis* of the

local *arteries.* Initial paresthesias are common property in these diseases as well as in osteomalacia, and so are local pain and increase of the tendon reflexes. But the remaining symptoms such as tendon clonus, positive *Babinski,* intermittent dysbasia, muscular atrophies and bladder and rectal troubles always point to spinal arteriosclerosis.

Spastic spinal paralysis or some other affection resembling it are not unlikely to be taken for osteomalacia. But a careful observer knows how to circumvent such a palpable error.

Hysterical pseudoosteomalacia on the other hand offers greater difficulties. It requires sometimes long continued and close observation of the patient before the contrast between the pains and the motoric properties of the two diseases, i.e., osteomalacia and hysterical pseudo-osteomalacia, is recognized. Suggestion and phosphoric treatment will solve the problem.

Pain in the Joints

In the majority of cases the patient is able to correctly localize an articular pain. But it happens also that he will attribute pain to a certain joint when the real seat of it is to be found elsewhere, or he will experience a painful sensation in a place somewhat removed from the affected joint.

From local tenderness, impairment of the articular motility and patent changes in the joint the truth can be easily learned.

For the purpose of a clearer understanding I divide the subject into two groups, viz.: *acute* and *chronic articular pain.*

I. Acute Articular Pain

When a patient complains of sudden acute pains in the joints our first suspicion is naturally that of *articular rheumatism.* It will be confirmed if we find the following symptoms: pains in the small and large joints, distal as well as proximal, as a rule symmetrical in those of the extremities, possibly in those of the spinal column, of the lower jaw, of the clavicle and the symphyses, etc., often synchronously in several joints bounding from one joint to another,

enlargements in the joints, the superficial skin has a reddish, shiny appearance and is feverish, rising body temperature, definite reaction to salicylic treatment, profuse, annoying and peculiarly acid perspiration even before salicylic drugs have been administered. Duration of the disease three or four weeks, but sometimes repeated recurrent attacks in previously affected joints. The heart, especially its serous coating, may be implicated and at times initial signs of a slight, transient angina are present.

In children we are able to discover sometimes multiple nodules the size of a pinhead or pea in the subcutaneous cellular tissue over the affected joints of the extremities. These nodules feel like soft cartilagenous matter and are more or less tender upon pressure, the skin above them appears quite normal, at the utmost only a slight tinge of reddening is perceptible. Similar nodes may be found in the tendons or in the tendon sheaths or in the periosteum, spinous processes, sacrum or in the galea aponeurotica. This condition is associated with endocarditis and chorea. Perhaps an appropriate name for it would be *acute nodose rheumatism.*

The necessity to differentiate between acute articular rheumatism and septic polyarthritis on the one hand and gonorrhoic arthritis on the other is of common occurrence in every day practice.

In *gonorrhoic arthritis* we are confronted by its monoarticular characteristics, i.e., that it settles principally in the knee joint—in women also in the wrist—and the contrast between the relatively low degree of fever and the high intensity of the pains.

However, there are cases in which these differential symptoms fail. I have seen, not infrequently, polyarticular forms of this disease in which several joints were progressively affected. Much information came to me from the discovery of a fresh gonorrheal infection in the patient, and also from the finding of gonococci in the urethral, i.e., genital secretions; and again, it is of importance to know that gonorrhoic articular rheumatism often develops only after the abnormal secretions from the genital organs have completely disappeared. It may also be due to latent gonorrhea or to chronic gonorrhoic prostatitis which has been overlooked. Hidden gonococci discovered by manual pressure furnish the evidence. If this prove a failure we must consider the peculiarities of the articular affection.

In the gonorrhoic form we miss the progressive element which is characteristic of acute articular rheumatism, but we find the distinctive, periarticular, pasty edema extending far beyond the articular limits. We also encounter endoarticular swellings and periarticular muscular

atrophy, intermittent fever and an early inclination to ankylosis. A trial puncture of the joint shows a cloudy, serous, cytologic fluid consisting chiefly of polynuclear leucocytes (later on also mononuclear cells) and gonococci. Furthermore, the relatively long duration of the disease is a speaking factor not to mention inefficacy of salicylic treatment, local reaction to gonococcus vaccine (arthigen). A concomitant disease of the joints, foreign to acute articular rheumatism, even an isolated affection, e.g., of the maxillary arthrosis or of a sterno-clavicular articulation must be taken as strong evidence of gonococcal arthritis. An affection of the endocardium or pericardium, of the pleura or peritoneum does not exclude gonorrhea and in that case we are dealing with a *gonococcal septicopyemia.* Presence of the gonococcus in the blood is positive proof.

Acute septic polyarthritis is often erroneously disguised as acute articular rheumatism. Of course, this cannot happen in a classical case of severe septicopyemia, but is not uncommon in cases of endocarditis bacteritica or lenta in which pains and swelling are of a lower quality and of short duration. Likewise in cases of a generalized septic infection of a mitigated character. In this condition the bacterial deposit in the joints causes multiple local pains and swellings. Mention must be made here of angina. In it

as well as in articular rheumatism we meet the same difficulty in deglutition and local pharyngeal affections either immediately before or with the initial stages of the attack. The matter may be further complicated by the fact that in both diseases a oncomittant infection of the cardiac valves and abnormal sweating are in evidence thus rendering an erroneous finding still more plausible.

If the patient has been under our observation early enough we must have noticed the lesions in the pharynx and the painful swellings in the lymphatic glands of the neck, a sure proof of bacterial infection. In the later stages of septic polyarthritis we must fall back upon the progressive nature of the disease, acute splenic tumor, marked hyperhemocytosis and positive diazo reaction. (I agree with *R. Schmidt* that the diazo reaction is a positive contraindication against primary as well as secondary infectious articular rheumatism no matter of what genesis.) Furthermore, I mention hemorrhages or white spots in the retina, laboratory examination of the articular serum, be it clear or cloudy, likewise of the blood and relative failure of salicylic drugs.

Akin to those connected with septic polyarthritis are the initial and later on intermittent articular pains caused by *endocarditis lenta* so frequently mistaken for articular rheumatism

with concomitant heart disease. The error is not pardonable, for the symptoms are plain enough. We have an intermittent, mostly long continued morbid condition, a leaning to infarcts, a relatively hard, often painful tumor of the spleen, pronounced leucocytosis, embolic nephritis, all the symbols of a recent or recrudescent endocarditis and the presence of the streptococcus viridans in the blood. We have, moreover, pus cells, gonococci, *influenza bacillus,* and *Weichselbaum's meningococcus,* every one of them a septic agent. The latter alone are able to parade before us under the false face of an acute febrile polyarthritis without displaying the characteristic symptoms of their own isolated, classic disease. To make here a correct diagnosis of a specific, septic polyarthritis is only possible with the aid of a competent laboratory examination of the blood and the articular fluid, rarely through the port of entry, i.e., the postnasal pharyngeal cavity.

A similar state exists in *paratyphoid bacillosis* with gastro-intestinal symptoms. Bacteriologic and serologic blood tests will make the diagnosis positive.

Sepsis caused by the *pneumonia diplococcus* requires mention here, too. The blood test will make us recognize it as a true originator of specific polyarthritis. Our attention will be quickly invited by the unusual adherence of

blood to the fibrin net and leucocytosis in the absence of eosinophile cells.

Acute tuberculous articular rheumatism presents pretty well the same features as acute articular rheumatism. We have to distinguish here between two forms. (1) The patient shows high fever and multiple articular pains and swellings, cough, cyanosis, dyspnea and cerebral complications, and dies within a week or two. The postmortem reveals miliary tuberculosis implicating the joints. The diagnosis in such a case is rather difficult. Acute articular rheumatism is, practically speaking, out of the question; but cerebral rheumatism or miliary tuberculosis involving the meninges might be considered, and above all septic infection. The differential diagnosis is confined to miliary tuberculosis and septic, infectious-chorioidal tubercles, dyspnea and cyanosis with negative pulmonary findings, rather soft tumor of the spleen, positive diazo reaction, leucopenia with polynucleosis and progressive lymphopenia and monopenia in miliary tuberculosis, hemorrhagie retinitis, cutaneous hemorrhages, pain in the bones, leucocytosis (only in severe cases of leucopenia), absence of lymphocytosis (nearly always with lymphopenia in sepsis). So far as special localization in the joints is concerned the bacteriological findings of the articular fluid and of the blood should prove useful. Of the cytologic conditions of

the articular contents we have no definite knowledge. It is not unlikely that in these cases multiple articular swellings and an accumulation of fluid in the abdominal cavity together with local tenderness are found.

(2) The second form approaches more closely the clinical picture of articular rheumatism, but similar conditions as under (1) prevail: pains in the joints, likewise enlargements, high temperature, generally constant above 39° centigrade. The differentiating points, however, are: we observe, as a rule, no local reddening of the skin, no characteristic, sour-smelling perspiration; witness the progress of the disease by bounding into new joints, the absence of complicating cardiac affections, the failure of salicylic treatment except that it mitigates the pain and somewhat reduces the swellings, lymphocytosis of the articular fluid and above all the local reaction of tuberculin. Pulmonary tuberculosis and other tuberculous phenomena may be concomitant symptoms and materially assist in the diagnosis which will find another adjuvant in the general course of the disease.

Multiple painful articular swellings and sometimes local reddening of the skin are not unusual in resorption of tuberculous serous exudates, especially in the pleura.

Tuberculosis may also be the generator of simple toxic multiple *arthralgia.* The same

applies to *syphilis.* On the ground that acute articular rheumatism is able to provoke multiple pains in the joints strongly resembling those due to such arthralgias, a confusion in the diagnosis is likely to arise. But salicylic drugs promptly clear up the situation.

This applies in the same measure to *secondary syphilis* when the patient suffers from pains in the joints that are accompanied by fever and general indisposition, but not local changes. It is different, however, when local reddening of the skin is noticeable. The salicylic test is here not adequate. We must watch closely the progressive nature of the pains, look out for tumors in the spleen and make sure of the character of the primary affection; search for the presence of spirochetes and the *Wassermann* reaction become a necessity.

A differential diagnosis between *syphilitic arthritis* and *gonorrhoic articular affections* is urged upon us when a chancre with purulent discharge is discovered in the urethra. The distinguishing features are easily recognized: in syphilis the articular swellings are less painful, or even painless altogether, whilst in articular rheumatism we have multiple localization, evidence of bacteriologic and serologic tests, and extreme local painfulness, to which in gonorrhoic arthritis is also added affection of the periarticular tissue.

Late syphilis sets in years after the initial attack has been overcome with or without fever and even without swelling in any of the joints. In such cases where doubt exists it is advisable to resort to antirheumatic therapeutic measures. With negative results we know that the disease is not rheumatism but of a tuberculous or syphilitic nature. The *Wassermann* reaction and antiluetic remedies will clear up the situation still further. Remember also that tuberculosis is indicated by the presence of a pronounced splenic tumor.

There are other cases of indubitable aortic insufficiency with slight dilatation of the heart in which the anamnesis lays bare a pristine attack of syphilis or acute articular rheumatism. And again, I have seen individuals with undeniable gonorrhea who were suffering from pains and swellings in several joints and yet they proved to be cases of genuine syphilitic articular rheumatism.

Multiple enlargements of the *lymphatic glands* are of importance in the diagnosis not only of syphilitic conditions but particularly so of *acute rheumatism in the joints.* As a rule they are strung together in chains or in groups around the affected joint and are sensitive on pressure. Both these qualities are helpful in differentiating between syphilis and acute articular rheumatism.

In the former the swellings are universal and indolent, in the latter sensitive and regional.

In the *Still-Chaufford disease* of which we have all seen so much during recent years, we find multiple swellings of the lymphatic glands with multiple affections of the joints, (also accompanied by swelling and articular pain) in every form of the ailment from the subacute to the subchronic stage; sometimes also periodic or else continuous subfebrile temperature. The joints are painful and disfigured—more in the sense of multiple chronic articular rheumatism without ankylosis—, the glands are fused and tumefied, those in the armpits may lie so deep that they escape attention unless the patient is examined in an upright posture (the histologic examination of excised glands does not show a distinctive pathological tissue form but only the picture of a simple fibroadenitis). We further find a moderately hard splenic tumor, vasomotoric disturbances (cold hands), exophthalmos, tachycardia and slightly raised temperature. In some cases we may also observe a slight tuberculous apex affection, or indurated mediastinitis or a questionable pericardiac concretion with the heart. In my experience I have found on several occasions that the general as well as the local reactions in the joints and glands to bovine tuberculin were very much stronger than to the human product, i.e., the

curative qualities of the former were more striking and satisfactory, for which reason I took, at any rate, several cases of this disease as a specific form of bovine tuberculous articular rheumatism.

In *acute leucemia* enlargements of the joints and glands are by no means a rarity.

There is another form of *typical chronic, articular rheumatism,* though not very common, with strong pains, exudation in the joints and ulnar position of both hands which we may witness in young women. We shall find chronic indolent lymphatic swellings in the armpit as well as in the forearm, not a trace of tuberculous affection in the whole system, but evident endocarditis of some standing and mitral stenosis and insufficiency. The genesis seems to be not subject to doubt.

Among the *infectious articular rheumatoids* or *pseudo-rheumatisms* I name here first *scarlatinous polyarthritis,* a polyarthritis which appears in the second or towards the end of the fourth week of the disease. It is a serous, sometimes a purulent affection of various joints, finger, hand, foot or knee, often accompanied by pericarditis, more rarely by endocarditis. When it follows on top of a purulent arthritis it assumes a septic pyemic character. It differs from the common form of articular rheumatism insofar that it does not, like the latter spread

by leaps, as it were, from one joint to others, but persists in the affected parts to disappear again after three or four days. The joints may be similarly affected in *smallpox, chickenpox,* erysipelas, possibly with endo-myocarditis, and also in influenza, but with more frequency in or after pneumonia. In other localized diplococcus infections, e.g., ulcerating teeth or otitis, generally only one joint, preferably the shoulder, is involved unless streptococcic infection supersedes. It follows also in the wake of dysentery (with stubborn persistence in a single joint), the same as in ulcerous colitis complicated with endocarditis. The attack survives the original sickness for a considerable span of time and sometimes only sets in as an aftereffect when the characteristic intestinal manifestations have already gone away.

Under the last named conditions pseudo-rheumatism in colitis may set in all of a sudden after a chill, with high fever, intensive pains, immobility and swelling of the joint with reddening of the skin, to last for several months. Considerable disfiguration and periarticular muscular atrophy are seen, and yet complete recovery takes place, sometimes after cecostomy.

In sporadic instances of multiple articular pain and swelling after dysentery we may have to deal with a case of septic polyarthritis instead of the aforementioned toxic pseudorheumatism

when the blood becomes irritated by the invasion of bacteria (e.g., streptococci) into the blood from an intestinal ulcer. The blood test will confirm this. Even in ordinary diarrhea *enteric polyarthritis* may be observed. Although here the local reddening of the skin, heat, sweating and cardiac complications are not in evidence and salicylic remedies prove ineffective, we often find conjunctivitis and urethritis in company with this disease.

Mono- and polyarthritic conditions are not so common in *epidemic cerebrospinal meningitis* (see "Sacralgia"), or in *epidemic encephalitis,* in *malaria,* (especially tropical malaria, and the chronic type with intermittent articular swellings) or in *diphtheria,* but are very rare in measles, German measles, whooping cough, varicella and spotted typhus, although in all these diseases they are more in the nature of arthralgias of an infectious toxic origin.

A similar state exists in the initial stages of *tetanus.* In *leprosy* very severe pains and swellings in the joints with fever prevail analogous to syphilitic polyarthritis and periarthritis. These arthralgias either accompany the incipient stages of the infection or develop gradually during its course. If the former is the case an erroneous diagnosis of acute articular rheumatism or a sepsis is not excluded, for instance, in smallpox in which articular pains are often

predominant. Here we must be guided by the other early signs, namely pains in the sacrum, prodromal exanthema, the eruption of papules on the mucous membranes of the eyes, mouth and throat, negative bacteriologic findings, cytologic condition of the blood (in sepsis leucocytosis, leucopenia with neutrophilia or lymphopenia, in variola leucocytosis and monocytosis). In influenza arthralgias are very common.

Serum injections frequently cause articular swellings and local pain, also in lymphatic glands, with high temperature and marked disturbances in the general condition of the body; even herpes and albuminuria may be concomitant factors. After two or three days these manifestations disappear again. The absorption of any *hydropic* fluid, e.g., of a serous, tuberculous pleural exudate, in the system has the same effect, an anaphylactic manifestation, no doubt, due to the influence of endogenous albumin.

In *paroxysmal hemoglobinuria* the pains in the joints are not accompanied by swellings or other articular changes and disappear with the attack.

Very tenaceous pains go with *recurrent fever,* especially in one or more of the larger joints, but without swelling. Sometimes they are of such a vicious character that they are easily taken for articular rheumatism, but the temperature, the enlargement of the spleen and the

presence of spirochetes in the blood will soon bare the error.

In *dengue fever* pains and enlargements in the joints, reddening of the skin, fever, affecting one joint after another, are typical symptoms of the disease. A false diagnosis of acute articular rheumatism or of acute influenza is not among the impossibilities. The geographical locality in which the disease occurs must be considered. Generally speaking a close observation of the course taken by the malady will secure a positive diagnosis, especially if one bears in mind that besides the symptoms already enumerated there exist also stiffness in the knee and other joints, likewise in the vertebræ, loss of appetite and a thickly coated tongue. The fever is on the wane within three to seven days, perspiration is copious, but a few days later high temperature returns, the articular pains come back and with them exanthema appears in the face, on the hands and the forearms, similar to purpura or measles. The skin peels off soon afterwards and a slow recovery ensues.

In *Malta fever* the patient suffers from multiple pain chiefly in the larger joints which set in about two weeks after the beginning of this pseudotyphoid disease. The principal symptoms are gradual rise of temperature, chills, continuous remittent or intermittent fever, frequent heavy sweating, thickly coated tongue, nausea,

vomiting, constipation, meteorism and acute splenic tumor. The pains are of a rheumatic nature affecting one or more of the joints. We notice swellings and local reddening of the skin, serous extravasations in the shoulder, hip or knee joints for a month or so. When the acute articular affections make their appearance at the very beginning of the disease, as seems to be the case sometimes, the differential diagnosis from acute articular rheumatism is not so easy. The agglutination test for micrococcus melitensis and certain clinical signs such as painful testicles should, however, positively distinguish this disease from articular rheumatism as well as from septic polyarthritis. This refers with like force to those cases in which a metastatic purulent arthritis results from Malta fever.

More difficult is the differentiation from *abdominal typhoid,* especially *arthrotyphoid.* This, of course, refers only to those cases in which pains in several joints with swelling and reddening accompany the symptoms of typhoid. The resemblance to acute articular rheumatism may here easily lead to some mistake. But when we observe that the affection does not bound, as it were, from one joint to various other joints, that the characteristic perspiration is wanting the diagnosis of abdominal typhoid should be made positive, especially in the presence of a tumor in the spleen, of bronchitis, of a diazo

reaction (when urobilinogenuria is absent), of leuco- and neutropenia with lymphocytosis and eosinophilia, not to forget the bacteriological blood test and the *Gruber-Widal* reaction. It is worth while also to remember that in abdominal typhoid fever secondary, mostly purulent articular affections make at times their appearance. They are of great importance for a correct etiologic diagnosis. Especially in children localized pains in the cervical spine with wryneck have been observed which makes it advisable to think of a possible typhoid infection when dealing with a case of feverish torticollis.

That *antityphoid serum* may cause articular pains has already been mentioned. But I will add here that acute polyarthritis with light fever and a septic aspect may originate in a typhoid carrier by way of auto-intoxication thus producing a *typhoid bacillary septicemia.*

In paratyphoid articular pains and swellings of an infectious toxic nature may also be observed. In fact the paratyphoid bacillus may in some instances be the causative element of an acute, purulent arthritis. In a like manner multiple affections of the joints may be conditioned by a coli infection, i.e., in severe cases of enterocolitis. Or in the absence of a coli infection the articular pains can just as well be merely the expression of a coli bacillosis.

I have already mentioned in another place

that in *trichinosis* a paratyphoid state combined with articular pains is not an unusual occurrence.

While speaking of the differential diagnosis between *arthrotyphoid* and *articular rheumatism* I wish to call attention to another point of importance. It is this: abdominal typhoid, like many other infectious diesases, is apt to revive a dormant articular rheumatism with all its pristine pains and changes in the very joints which were affected by the primary attack. In other words every acute infectious disease that is capable of producing morbid conditions in the joints may be the connecting link between the infection and genuine acute articular rheumatism. *Typhoid* and *gonorrhoic polyarthritis* are by no means an impossible combination.

Glanders (malleus) in the acute form bears a stronger resemblance to septic pyemia than to articular rheumatism. In the primary stages of this disease we observe a painful infiltration of the skin which rapidly develops boils typical of farcy; the surrounding lymphatic glands and vessels are inflamed; the nose is infected with a specific form of rhinitis and sores form along its margin and in the mucous membrane. But when these typical symptoms are not present we only gain the impression of a general severe septic pyemia, the more so as towards the end of the first week the eruption in the skin looks

very much like a multiple, at first papular, then pustular exanthema. Most conspicuous are the multiple affections of the joints and muscles by purulent exudates, the formation of pustules on the skin and the mucous membranes, of abscesses in the muscles and pus under the skin, genuine guiding symptoms for a correct diagnosis, which may be further assisted by the anamnesis (contact with animals), bacteriologic examination of the pus, etc., for the presence of the malleus bacillus.

The diagnosis, of course, will be more difficult when an acute attack after several days of continuous fever with very severe pains suddenly develops swelling of one or more joints, thus simulating a multiple septic arthritis. But we shall find safety from error if we consider the following points. Malleus shows a distinct, diffuse reddening of the skin of a deep hue as in erysipelas, almost brownish red, a simultaneous infiltration of the skin which subsequently reaches into the muscles, and cutaneous pustules typically characteristic of malleus.

Farcy sometimes travels under the guise of subacute disease. It is then somewhat difficult to arrive at a proper conclusion. The main points to keep in view are always: examination of the blood, livid red coloration of the skin over the affected joint, edematous infiltration of the surrounding parts, mild febrile conditions and

general indisposition. I may add that chronic malleus may persist for several months.

Sporotrichosis is likewise connected with articular pains and sometimes with hydrarthros. The presence of subacute, gummatous nodes, fistulæ, resemblance to tertiary syphilis, and *Beurmann's* sporotrichon should be sufficient evidence for a correct diagnosis.

Periarteritis nodosa presents sometimes a septic aspect with sporadic pains and swellings in several joints. The diagnosis is fully discussed in "Abdominal Pain" and in the section on "Muscular Pain," and will again be referred to under "Typhoid Diseases."

Acute leucemia is apt to be ushered in by multiple articular pains and changes thus leading to the false impression of articular rheumatism or septic polyarthritis. To differentiate look for signs of scurvy in the mouth, more generalized glandular swellings, harder consistence of the splenic tumor, progressive pallor and tendency towards hemorrhages, and do not neglect the blood test. Our present state of knowledge does not in any way clear up the relation of myeloid leucemia to streptococcic sepsis, for we must remember that in sepsis both myelocytes and lymphocytes may be found in the blood.

In *erysipeloid* pain and bulging are observable in the middle joints of the fingers. The color

of the skin is of a bluish, violet tint and reaches out in tongue-shaped extensions with pale spaces between them.

Rheumatic *peliosis* with multiple articular pains and prominences distinguishes itself from articular rheumatism by periarticular hemorrhages especially on the minor surfaces of the extremities. Still it is wise to bear in mind that arthralgias are not an uncommon occurrence in the beginning of purpura hemorrhagica and likewise in scurvy. In the latter disease particularly severe pains are caused by hemorrhages into the joints which become enormously enlarged almost reaching the stage of an ankylosis.

In *hemophilia* and *hemophilic hemarthrosis* the hemorrhages into the joints cause exquisite pain therein. If there is also high temperature and we are not aware of the fact that the patient is a bleeder, we may easily be led astray and make a diagnosis of articular rheumatism or even of acute monarthritis. A careful anamnesis is of paramount necessity in all such cases. Additional adjuvants are found in the presence of other hemorrhages, diminished coagulation of the blood during the intervals which gradually reaches the normal stage with the ensuing hemorrhage (extravascular coagulation), and concomitant lymphocytosis with a normal count of the blood platelets. Hemarthros ends either in a

slow recovery or degenerates into a chronic condition with resulting ankylosis.

I will now speak of those articular diseases which are not necessarily of a metastatic origin.

Foremost among these is gout, a constitutional disease which is more frequently the causative factor of monarthritis than of polyarthritis. If it affects only one particular joint, for instance, that of the big toe (podagra) or at any rate only the distal joints of the lower extremities we have a very fair proof of its monoarticular nature. But gout may also attack the upper extremities (*chiragra, omagra*) or some other joint, or even quite a number of joints. In any case it is always characterized by the fact that the pains generally set in after midnight, or if they do come on in the daytime they grow worse during the night. The patient is slightly feverish, the skin shows a velvety blush which tapers into the normal of the surrounding parts, is shiny, tense and hot, with tactile hyperesthesia and hyperalgesia, is marked with lymphangitic striæ, and the cutaneous veins are puffed up. The attack generally lasts from three to four days or a week. This differentiates it from articular rheumatism.

I have already said on a previous occasion that gout is one of the hereditary diseases, also that it is characterized by the peculiar behavior of the uric acid in the blood (hyperuricemia)

and that an attack may be provoked by the consumption of nucleins. But I will add here that a multiple acute articular affection which makes its appearance for the first time after the age of 50 should arouse the suspicion of a possible gouty involvement. But it does not always prove true. On the other hand persons who have suffered in bygone days from articular rheumatism are not immune from an attack of genuine gout in later years in the very joint or joints that were involved by the previous attack. I will refer to the differential diagnosis between gout and other articular affections later on when speaking of the rudimentary development of this ailment.

Although the *chronic* form of *multiple articular rheumatism* shows a strong resemblance in its incipient stages to the acute attack, yet there are certain definite signs by which the two can be separated. First of all there is the course of the disease itself, then there is the fact that in chronic rheumatism it is particularly the joints, especially the small ones of the upper extremities that are involved, there is no recovery in the joints once affected when the disease leaps into others, characteristic sweating is absent, but there is a prompt response to salicylic treatment. Consider also moderate rise of temperature, the stubbornness of the disease and the rare occurrence of complicating heart dis-

eases. I am inclined to believe that the originating force is to be found in some primary affections of the oral cavities, more likely than not in the tonsils.

There are other acute diseases which do not originate from the joints themselves but from adjacent parts such as the epiphyses of the bones, and in consequence are mistaken for acute *rheumatic* or *septic arthritis.* In such cases we should always think of the possibility of an existing *osteomyelitis* or *syphilitic ostitis.* If only *one* joint is involved a *monarthritis* may be simulated, but generally several joints are affected. In purulent osteomyelitis the joints are swollen and their integument is reddened, the patient complains of local pain (more acute in motoric action), chills and fever. If upon careful scrutiny we find that pain and tenderness are more acute in the adjacent osseous parts than in the joint itself and that the swelling extends beyond the capsule, the diagnosis of osteomyelitis is correct. The X-ray can easily confirm such a finding, doubly assured by the develcpment of an edema and the livid coloring of the skin over the affected part, the presence of lymphangitic striæ and the cordlike appearance of the superficial veins. I may add that in osteomyelitis fever and pain precede the swelling, while in arthritis they come at one and the same time. This is important insofar

as a purulent monarthritis of metastatic origin may easily obscure the primary infection.

Luetic ostitis is mentioned here because in it gummatous growths are formed in the bone under the periosteum nearest to the joint which may be taken for mon- or polyarthritic conditions. Analogously we find in arthritis gummosa intermittent or periodic fever as a concomitant of the gummatous affections of internal organs. The anamnesis, the typical symptoms of syphilis, the *Wassermann* reaction, specific treatment and the Roentgen-ray should not leave the diagnosis in doubt.

Jacksch's disease is another carrier of articular pains. The patient is subject to irregular attacks of fever and to pains in the joints or in the extremities. A careful study of the disease shows that the pain is really located in the bones and not in the joints, for the Roentgen-ray distinctly reveals a thickening of the periosteum. Salicylic treatment is ineffective which at once speaks against rheumatism. But if endocarditic complications accede, which is often the case, the diagnosis may meet with obstacles. Much help is derived from the clinical examination of the blood for an increased number of polynuclear or eosinophile leucocytes and, perhaps, also myelocytes. I cannot speak from personal experience as I have never seen a case of this ailment. I must admit this also of *tetanus*, for

which it is claimed that during the so-called latent period dragging pains in the extremities are in evidence in addition to swelling and reddening of one or more joints, even eruptions resembling purpura as an expression of vasomotortic disturbances. In *Basedow's disease* and *arthrogryposis* similar observations have been made. In *bronchiectasy* and *pulmonary emphysema,* both closely related to osteoarthropathy, the articular changes are subject to many fluctuations; they come and go to reappear again during many weary months.

The acute, painful, articular swellings at the beginning of a *multiple insular sclerosis* find their explanation in the presence of other initial symptoms of this disease, such as painful paresthesias of the extremities, intention-tremor, nystagmus, missing abdominal reflex by increased tendon reflex, visual disorders, headaches, vertigo, disturbances of speech and bladder.

In *acute polyneuritis* acute articular swellings coupled with sensibility and motility disturbances in other parts of the body are bound to interfere with proper diagnosing. I say purposely "in other parts of the body," because if these symptoms manifest themselves in the immediate surroundings of the swollen joints the originating cause may be found in a secondary periarticular neuritis due to the primary affection of the joint. The differential diagnosis,

i.e., whether we have before us a case of a primary neuritis with a secondary articular swelling, or vice versa, may be a difficult problem to solve, because motoric impairment is evident in both cases. It, that is the diagnosis, can only be based upon the fact that "neuritic changes" may exist in places where no articular swelling can be proved, and upon the X-ray. (See "Shoulder Pains.")

An attack of *acute polyneuritis* may be mistaken for that of acute articular rheumatism in the absence of swellings dependent on polyneuritis. But this error can be quickly mended by a careful search for tenderness in the muscles and nerves, enlargements in the latter, sensibility and motoric disorders and anomalous tendon reflexes, all of which defects are claimed by polyneuritis.

Ischias may be taken for *gonitis.* In both the patient complains only of pains in the knee-joint or close to it. Observations of tendon and skin reflexes, pressure points and the appurtenant signs of ischias should prevent the error.

During the incipient stages of *acute poliomyelitis* in children we may observe similar conditions as in acute articular rheumatism, i.e., the patient lies motionless in bed and complains of pains in the arms and legs and all the members of the body which are aggravated by any attempt to move. Only the most exacting ob-

servation of the tendon reflexes, lumbar puncture (?) and the epidemiologic moment will bring light in the situation.

Besides the diseases referred to in the foregoing pages there are certain other rudimentary forms of articular rheumatism to which we must advert in this place. In mitigated articular rheumatism the pains and anatomical changes are of a less virulent, milder type, the disease develops only in one joint with less pain and less noticeable swelling.

The diagnosis will find much assistance in the fact that the attacks come by leaps and bounds now in this, then suddenly in another joint, there is tendency towards perspiring, hardly any fever, if at all, localization in small as well as in larger joints and a prompt reaction to salicylic drugs. The anamnesis is most useful insofar as it generally reveals the fact of a previous attack of typical fullblown articular rheumatism.

These low grade polyarthritic forms are often symptomatic of some mild *septic infection.* If a catarrhal angina preceded the attack, the diagnosis may be somewhat troublesome, but the symptoms described above should suffice to remove the doubtful element.

During the *menstrual period* high temperature, pain and swelling in the joints, general indisposition are common affairs. In some in-

stances they might arouse the suspicion of some latent tuberculous condition, and it is advisable to make proper inquiries in this direction. In most cases they are, no doubt, due to bacterial septic or toxic influences. The cause may also be found in some lesion of the mucous membrane of the uterus caused during delivery or by some other interference.

But there is also a very mild form of purulent *osteomyelitis* in which the patient complains only slightly of pain and functional impairment of the joints. The clinical points already mentioned before and the Roentgen-ray should make the diagnosis positive.

If the affection is confined to one joint only the differential diagnosis must decide between *rheumatic* (also *monarthritis rheumatica nodosa*), *gonorrhoic, syphilitic* and *uratic monarthritis.* In acute monarthritis we must also bear in mind *pneumococcic infection.*

Acute tuberculous monarthritis is sometimes mistaken for the rheumatic form. The symptoms are: a sudden sharp (sometimes only slight) articular pain in the finger or in the hand, the joint is swollen and its movement gives pain, the skin over it is hot and tense, although at times there is no rise in the local temperature. Now if we find that the swelling is of a doughy consistence and assumes the form of a spina ventosa and that the skin over the joint has a

dull, glazy appearance we are warranted to decide in favor of tuberculosis, especially if in addition fistulous formations should come into evidence.

It is important to remember that a uratic arthritis not infrequently follows a local articular injury. There may have been a contusion or a distortion or some other traumatic condition, or pressure from a narrow shoe or a cold of some sort which primarily caused the swelling and impaired the motility of the affected joints and finally degenerated into arthritis urica.

Acute hemarthrosis in *hemophilia* is our next point of consideration. In this disease we find the development of a painful swelling (in the early stage the pains are frequently absent) in a joint, chiefly the knee or elbow, not always accompanied by fever. It generally comes on after a cold and sometimes is preceded by a slight traumatic condition. The joint becomes stiff and immovable, the hemorrhage in the joint finds extension into one or two more and we soon get the impression of an acute articular rheumatism. If, however, lateral hemorrhages appear in the skin and in the mucous membrane, the diagnosis is soon turned in the right direction. When these dermal signs are missing the only means to fall back on will be the anamnesis: is there inclination to bleeding in other parts

of the body; if so, is it a familial charasteristic; is the patient a male, his age—there are cases of hemophilia in females, but not many—presence of lymphocytosis, reduced vascular coagulability of the blood during the hemophilic hemorrhages while the extra-vascular coagulability in the intervals between the fluxes is diminished? Gradual return to normalcy during the course of the disease.

Multiple articular pain without hemorrhage in the joints is not a rarity in *hemophilia,* in fact in every form of hemorrhagic diathesis (*scarlet fever, scurvy*).

The pains in the joints or their swellings that accompany hemorrhages into one or successively into several joints may originate from other causes, such as a spontaneous fracture of the bones themselves, e.g., the neck of the humerus or femur through a local neoplasm, e.g., myeloma or neoplasmatic metastasis. If motoric impairment and crackling sounds accede, a false diagnosis of acute mon- or polyarthritis may result. A careful palpation, however, for protuberances either at the injured or any other part of the affected bones and the Roentgen-ray should forestall the mistake.

Acute arthropathy is only mentioned here as a possible source of the pains described in this department, but it will receive more detailed attention in the following chapter.

II. Chronic Articular Pains

Many of the acute diseases described in the preceding pages often become *chronic* in their character in which state they manifest the same morbid conditions and localized pains, for instance, gonorrhea, tuberculosis, articular syphilitic affections, hemarthrosis in hemophilia and scurvy. This refers principally to those lasting changes (leading eventually to ankylosis) in one joint only succeeding an attack of *gonorrhoic* or *tuberculous articular rheumatism* or else of *hemarthrosis,* a rather important symptom in these affections of the joints.

Gummatous arthritis is apt to show similar results, for here, too, we come across swellings of the smaller as well as the larger joints often involving the entire head of the bone. Deforming arthritis in tertiary syphilis of joints must also be considered here. In most cases chronic gummatous arthritis is monoarticular in its character. In both cases we shall find light in the very conspicuous colateral affection of the epiphysis and periosteum, in the anamnesis (premature births, abortions), in the nocturnity of the pains, serologic blood test and the results of specific treatment. In congenital syphilis, which may cause likewise various chronic articular conditions, the picture is often that of osteo-

chondritis, or a simple sinovitis or arthritis deformans.

Gout requires special attention in this place. After one, chiefly after repeated typical attacks we find a series of weaker attacks, but of longer duration, with strongly marked exudates in one joint; the painless intervals come to a stop, deposits of uric acid begin to show up and a crunching sound may be heard in the affected joint; there is increasing stiffness, motoric inhibition and subsequent deformity and ankylosis. Hereditary gout will be dealt with in another place. I will only mention here that the diagnosis in chronic gouty changes of one or more joints is much assisted by the evidence of previous gouty attacks.

Chronic hydrops of *one,* chiefly the knee joint —it matters not whether a multiple articular affection has preceded it or not—should at once remind us of a tuberculous if not a syphilitic (congenital or acquired) or gonorrhoic condition, or of sporotrichosis, or of a chronic osteomyelitic, para-articular disease of the bones. In hemophilia the appearance of a joint may be changed by hemorrhage into it until it resembles a chronic hydrops. This is particularly the case in an affected knee joint. The differential diagnosis in all these processes is surrounded by great difficulties. In case the anamnesis, the serologic test and an attempted diagnosis ex ju-

vantibus (anti-syphilitic treatment) lead to no results, the cytologic and bacteriologic examination of the articular fluid, or in some instances the X-ray examination of the joint itself may prove successful. But there are cases in which even the knife does not furnish unmistakable proof of the nature of this disease.

There are other forms of chronic articular infections with permanent pains of varying intensity, and connected with inhibitory motoric action, enlargements, changes in the outlines, and certain anomalous conditions revealed by the X-ray within the joints. Diplococci, staphylococci, after-effects of diphtheria, scarlet fever, influenza, etc., may be the provoking causes of this *acute infectious pseudorheumatism* which itself has borne from the very beginning the character of a chronic evil or else degenerated from the acute into the chronic state during the course of the disease. If the latter was the case, the diagnosis would be apparent, especially when supported by bacteriological evidence.

Of the commoner forms of chronic articular infections that are here concerned it is as yet not possible to give a satisfactory survey because the opinions of the present day clinicians vary so much as to their classification and even nomenclature. I prefer to adhere to the older system which looks upon chronic rheumatism of the joints as an atrophic process established by

the Roentgen-ray, and keeps it separate from chronic deforming arthritis which it considers in the light of a degenerative hyperplastic articular condition. By this system the first named affection is divided again into two subsections, viz., *primary* and *secondary chronic articular rheumatism.*

The primary form which today is called "primary chronic progressive polyarthritis," is characterized by a slow creeping, sometimes subfebrile, but mostly fevereless beginning. It progresses symmetrically from the small joints of the fingers, toes and wrists. It has a chronic course with many intervening remissions marked with periods of increased virulence and temperature. The affected joints, especially the metacarpophalangeal ones, are swollen and nodose, and thickened with spinous protuberances owing to the enlargement of the caps of the bones or in some rare cases through articular exudates. The pain is, as a rule, sharper at the beginning of the movement of the joints which themselves are impeded in their actions if not rendered helpless altogether. Quick motions are intensely painful because the synovial villi are constricted.

The most characteristic criterion, however, is the contorted shape of the finger-joints. The ungual phalanx is bent to one side, the metacarpophalangeal joint is abducted towards the ulna, the wrist is enlarged, the distal end of the

forearm is cylindrically thickened. The toes also undergo changes. The big toe is, as a rule, drawn away from or under the other toes. The skin over the affected parts has generally a paler tint and is of a doughy consistence, but grows gradually very thin as the illness progresses, seldom sclerodermic. Gradually the process finds its way into the elbow and knee joints. Ankylosis is one of the later symptoms. But I have seen cases in which this affection, eschewing slowness, settled with savage rapidity and sudden feverish attacks simultaneously in several of the small joints.

In the *secondary* I comprise every other form of *chronic articular rheumatism* which had for a precursor an attack of undoubted, typical acute rheumatic polyarthritis. This secondary form does often present the manifestations described in the previous paragraph. More frequently, however, the result is a chronic fibrous ankylosed rheumatism, i.e., in one or more joints enlargements of the caps appear, also of the lateral ligaments of the joints and their tendons until a complete fusion into one solitary fibrous mass is formed causing complete rigidity and ankylosis. This transformation may continue after the symptoms of acute rheumatism have already disappeared if it has not completed its course during the progress of the disease.

The differentiation between the secondary and

primary form rests principally on the history of the case and the nature of the articular changes which are distinctly characteristic for both affections. It is well to remember that after an attack of *acute articular rheumatism pains will reappear* in the affected joints from time to time—often for years—influenced generally by metoreological conditions, likewise painful minor swellings and stiffness which may put the joint temporarily out of commission until the normal state is gradually reached again.

The second form of classical chronic affections of the joints is hypertrophic arthritis, more accurately called *deforming osteoarthritis.* It runs a slow, sneaking course, is not very painful and does not interfere much with the motility of the joints until later on in life. It may be also polyarticular in its nature and attack the smaller joints (especially in the spine). As a rule, it affects at first only one of the larger joints, the hip, shoulder, knee or the big toe, but sprouts out from there into the other larger joints. Anatomical as well as radiological examination shows onion formations, which naturally affect the motoric faculties of the joints more and more until the latter become quite immovable and very much deformed. Cardiac complications are rare whilst in chronic rheumatism they are ever plentiful.

There is another distinguishing sign to which

I desire to call attention. In both the aforementioned principal forms of chronic articular disease the pains are generally aggravated by any movement of the affected joint, whilst in the initial stages of chronic articular rheumatism and of deforming arthritis the pains, as a rule, start with the transition from rest into activity of the joint, is most acute during the first few movements but declines in virulence or disappears altogether with continued exercise. Another important sign in deforming arthritis—in fact in every painful anatomical joint disease—is this: a knock or a stroke on the elbow produces pain in the corresponding shoulder; in deforming omarthritis a kick on the heel or a slap on the sole of the foot or on the bent knee elicits a similar pain in the hip joint. This same distinguishing sign is also found when in deforming arthritis the pain radiates into the entire extremity with demonstrable tenderness on pressure of the nerve roots thus simulating an *ischio-* or brachial neuralgia. The decision should not be hard to make for it is easy to see that this overlapping painful zone really emanates from the joint. Moreover in neuralgia the joint can still be used although with discretion.

Chronic articular rheumatism due to *ochronosus* deserves special mention. The presence of homogentisic acid is the cause of this trouble as well as of chronic endocarditis which so fre-

quently accompanies it. The evidence of existing alcaptonuria and dysuria facilitates the diagnosis.

Gout must be our next subject of consideration. The chronic condition of this disease evolves from one or more preceding acute attacks, a fact upon which the positive diagnosis must rest. In both forms we should look for crepitant râles in the affected joints, especially the knee-joint, for chronic inflammatory changes in the joints due to deposits of uric acid, and to subsequent deformities, structural anomalies and ankylosed conditions. Uratic deposits in the softer parts around the joints (tophi) are often the causative factor of these changes. The fingers appear swollen or thickened in certain parts and twisted out of shape, the wrists ulnad. The toes, especially the metatarsophalangeal joint of the big toe, are bent in a devious manner under the other toes, thus forming, as it were, a foundation for them. This abnormal position may, however, be due to chronic articular rheumatism or to ill-fitting shoes. The presence of gouty nodes would be clinical evidence.

These nodes are often found on the helix of the ear, but chiefly around the joints, with preference about the olecranon. They rise from the subcutaneous tissue, from the synovial sacs, rarely from the periosteum or the nerve processes and develop gradually but without causing

pain. If their origin is derived from any other source they may be recognized by sliding them about over the bone. Otherwise the diagnosis can be made from their soft, doughy feeling and by needling a young tophus when a white, chalky, mushy mass exudes which consists essentially of uric acid and its salts. This is also the case when this exudation breaks spontaneously through the skin leaving a scar in the place of the former fistula. Beneath the scar prickly points or horns may be felt.

If the nodes are stony hard the diagnosis may be rather difficult to make owing to the fact that such protuberances are also observed in chronic articular rheumatism. (The differentiation between these and *Heberden's* nodes will be discussed later on.) In such a case it is wise to fall back on the Roentgenogram, the blood test for uric acid after a purin-free diet.

Notice also that the morbid conditions of the joints in chronic gout are quite abnormal and so different from those prevailing in chronic rheumatism: in the latter, simultaneous and symmetrical attacks and an even disfiguration of several joints, in gout, asymmetry of the joints, affection of one joint only, perhaps nodes on only one joint thus making the asymmetrical appearance of the various joints more drastic.

In gout the crepitus (especially in the knee-joint) is more like the crackling sound over the

lungs, while in chronic articular rheumatism it is of a coarser character and sounds more like friction or large bubbles exploding. This is particularly the case in deforming arthritis. If the pains are felt more acutely during the night time and begin to tone down towards morning it speaks rather for gout, because in chronic rheumatism the painfulness awakens with the sleeper's first movements as he tries to rise from the couch. Nevertheless these conditions are sometimes reversed in different patients.

Of course these are clinical points which establish the genesis of a gouty affection: such as hereditary conditions, the life led by the patient, preceding or concomitant manifestations of chronic gout, chronic-uratic diathesis, the patient is subject to catarrh in the air passages or to asthmatic attacks, to iritic or sclerotic affections, dyspeptic or neuralgic troubles, localized pains, e.g., achillodynia or tarsalgia, cramps in the calves, eczema, hemorrhoids, glycosuria, contracted kidney and nephrolithiasis, likewise increased uric acid content in the venous blood even with a purin-free diet.

The assumption that severe pains in the big toe point to a gouty affection often enough leads to a mistaken diagnosis and wrong therapeutic measures. Frequently what is taken for gout is only a case of chronic arthritis caused by the wearing of shoes which are too narrow or pointed

at the toes—more often observed in women. The tightly fitting footwear forces the big toe into a valgus position, the metatarso-phalangeal joint is misshapen and the synovial villus is badly squeezed in. The resulting pains strongly resemble those caused by gout, and yet a narrow boot may arouse a gouty attack from its slumbers.

That *periarticular gouty tophi* are apt to be mistaken for *Heberden's* nodes is only mentioned here. The subject will be discussed more fully later on. To take a cutaneous tophus erroneously for a cystic growth or a small abscess or a milium, or else for a ganglion if seated in a synovial sac, seems to me well nigh impossible if a careful inspection is made. The error might be pardoned when concretions on the helix are observed which may be a congenital malformation on the ear, or a cartilagenous defect caused by frostbite resembling a pathological condition. The microscopic examination of the tissue and the anamnesis should remove any false impression.

Sebaceous cysts form sometimes on the cartilage of the ear and may be misinterpreted for gouty nodules, but it is easy to distinguish between them as the former are softer to the touch and are seated in the subcutaneous cellular tissue, while the gouty formations are harder and sprout from the cartilage with which they even coalesce

as they grow older. On palpation, it seems to me, the sebaceous cyst presents a hard but uniformly smooth surface whilst the gouty tophi have more of a sandy feel in them. If in doubt the chemical test will decide the presence of uratic contents.

There are cases in which we find a *combination* of both diseases, viz., *chronic gout* and *chronic rheumatism* of the joints. In these cases the gouty affections generally select those joints in which the rheumatic virus has already settled. An early uratic diathesis often lays the foundation for chronic articular rheumatism or gout in later years. When I use here the expression "chronic articular rheumatism" I mean only that affection which is entirely free from gouty symptoms and presents purely the typical features of the disease. In many cases of this kind I could find no therapeutic results whatever from the administration of antiuratic remedies, whilst they reacted promptly to the measures which it is customary to apply in chronic rheumatism.

It is by no means an uncommon occurrence in every day practice to come across cases of *chronic uratic diathesis* in which complaints of chronic pains in almost any possible joint are ripe. And yet a most careful scrutiny discloses no crepitus or deformity, not to speak of gouty tophi. Here it is wise to be guided by the hered-

itary and constitutional idiosyncrasies of these chronic sufferers. The diagnosis must depend rather upon a reliable blood test than the customary overestimated examination of the urine which from the standpoint of present day science is of a minor significance in these cases. Copious residue of the largest possible amount of uric acid and diminished *Zerner's* coefficient can at the very best only suggest the thought of gout.

Basedow's disease, hyperthyreoidism will in some rare instances be found associated with typical chronic progressive polyarthritis with exudations in the joints and must be looked upon as a thyreoprival affection which complicates the disease but not the diagnosis.

Gout may be, according to some authors, associated with a second metabolic anomaly, viz., *oxalemia* and *oxaluria.* They also combine with diabetes mellitus and obesity. The clinical signs are claimed to be abnormal fatigue, chronic dyspeptic and neurasthenic troubles, kidney colics, albuminuria, hematuria and chronic pains in the joints. The proof for its existence is to be found in the exaggerated amount of oxalic acid in the blood equal to from three up to ten times above the normal content which is at the utmost 1 cg. in 1000 ccm.

Chronic articular affections are often combined with psoriasis (*arthropathia psoriatica*). (This does not refer, however, to isolated pains

in the joints in the shape of mere acute after-effects.) I am not able to give a correct classification of this subdivision of articular affections.

Chronic swellings in the joints come under our observation sometimes during the course of *sclerodermia.* Patients afflicted with myxedema (thyreoprival articular rheumatism) complain of chronic pain in the joints of the hands and feet. We find the same condition also in adrenal affections (*Addison's disease,* tumors). Articular changes have also been reported in *myositis ossificans,* in *osteopsatyrosis* and in *Paget's ostitis.* Their genesis has never been properly cleared up and I myself cannot speak from personal experience regarding them.

On the other hand *chronic tuberculous articular diseases* are of greater importance. The diagnosis will find much help in the consideration of the regular tuberculous symptoms such as the usual manifestations, swelling in the glands, anal fistulæ, comptodactylia in the ring-finger, reaction to tuberculin and perhaps a complementary restringent reaction. There is a distinct difference between this and the common chronic articular rheumatism. The former begins, as a rule, in the larger joints, the knees and the hips, but when it reaches the finger joints the spindle formed swellings make their appearance rather in the joint between the middle and ungual phalanx, whilst in ordinary

chronic articular rheumatism the joint between the proximal and middle phalanx is affected. And again, in the tuberculous form the prominence comes into view in only one finger, when in the other several or all the fingers are simultaneously attacked. The Roentgen picture shows highly marked differences between the two. Chronic syphilitic articular rheumatism (pseudotumor albus lueticus) is another possibility that requires our attention in this connection.

Nervous Affections of the Joints

Patients troubled with *hemiplegia* are wont to complain of pains which come on either spontaneously or by passive movements mainly in the shoulder joints. They are, no doubt, due to a tension of the capsular ligaments caused by the dead weight of the paralyzed arm, for they disappear when the arm is properly supported.

Analogous pains are felt in the spinal cord in *poliomyelitis,* but in *tabes* and *syringomyelia* the pains are of a different kind. Here they settle with special viciousness principally in the joints of the larger extremities. The diagnosis is rendered more difficult by the fact that these pains, like all lancinating pains are largely influenced by weather conditions, damp, clammy days being the worst offenders. The conven-

tional symptoms of myelonal affections should, however, correct any possible error as to their true nature.

Cerebral diseases of a *hemiplegic character* are also guilty of producing articular pains which are felt along the lines of the nerve paths in the corresponding half of the body or its extremities. Sometimes they are continuous, sometimes intermittent, neuralgiform in character. Sometimes they are precursors or companions of locomotor paresis and at other times they follow in the wake of it. The diagnosis is fully given in the chapter on "Pains in the Extremities." In spastic hemiplegia the pains may also be due to a change in the static element of the joint caused by the increased muscular tonus.

In chronically diminishing muscle tonus (atrophies and tabes) articular changes and pains develop in a similar manner.

The trophic disturbances in the joints which manifest themselves in *arthropathies,* in *tabes* and syringomyelia are the natural progeny of other diseases in the central nervous system. Their resemblance to deforming osteo-arthritis and to chronic articular rheumatism, especially in syringomyelia, is so pronounced that even prominent neuropathalogists are at times misled into a false diagnosis. This is principally due to the fact that the arthropathy whose seat is

as a rule in the upper extremities, i.e., shoulder and elbow joints, does not come on suddenly but very slowly and is one of the first symptoms of the disease and at a time when the sensibility of the joints is still unimpaired. The patient complains of pain in the affected parts while arthropathy is still in the hypertrophic stage in which the swelling of the joint terminals and the osseous new growths in the articular capsules are only beginning and even exostosis on the diaphyses is forming.

On the other hand there is always the risk of mistaking the swellings in the finger joints due to syringomyelia for a symptom of chronic articular rheumatism. To avoid this error we must look closely for muscular atrophies, trophic conditions in the fingers, painless whitlows, scoliosis, spastic paresis in the lower legs, and bulbar symptoms the most important of which is dissociated insensibility to pain.

It is much easier to differentiate between tabetic arthropathy and deforming arthritis. The former comes on suddenly with large, painless swellings in the joints that may extend over the whole extremity. It may also follow on the heels of paresthesias in the articular region or in the joints themselves. The presence of free bodies in the joints unaccompanied by pain, grating on motion and crackling sounds are characteristics. The lower limbs are the favorite

site for attack in tabetic arthropathy, i.e., the knee, foot and hip joints, but when the shoulder is also involved tabes superior is rather indicated. The X-ray and the usual typical symptoms are the proper aids for a good diagnosis. Spontaneous fractures or infractions should not be overlooked.

It may be of interest to mention here that *loose joints* are often the consequence of acute articular rheumatism and mongolism, undoubtedly due to an anomalous development of the articular cartilages.

Diseases of the nervous system not only lead to anatomical changes in the joints but also to *arthralgias* and *multiple sclerosis.*

In *neurasthenia* the pain is, as a rule, less intensive but rather of a dragging, pressing, or burning kind. Often it consists simply of an aching tired feeling. Bodily motion softens or suppresses it altogether, whilst in chronic articular rheumatism the pain is sharpened thereby, although I must admit that here, too, the change from rest into activity in the latter case only awakens the pain sometimes which tones down again and gradually disappears under continued exercise. Of course, it goes without saying that radiological articular changes are not observed in neurasthenia.

The *epileptic* patient suffers also from pains in the joints during the interparoxysmal period.

Hysteria is another fertile ground for these pains. with preference in the lower extremities and more often due to some slight traumatic influence. If the attacks continue for some time a permanent fixation of the affected joint may be the result, atrophy of the surrounding muscles may also set in with secondary, trophic, anatomical changes in the articular portion. This makes it rather difficult to differentiate it from organic articular affections. But the following points will be of great assistance: the skin over the affected part has an hyperalgesic appearance, the sense of pain is reduced by distraction, the latter also allows of motions which seemed impossible before, the pains are sharpened when sympathy is shown the patient, if the head of the joint is pressed firmly against the acetabulum no pain is felt, but a slight touch of the skin over it is exceedingly painful. Naturally the X-ray is a great help in the diagnosis of these cases. Still we must never lose sight of the fact that even hysterical persons may be afflicted with anatomical lesions of the joints.

In some cases these articular pains without manifest hysterical or neurasthenic stamp appear as true *articular neuralgias,* that are sometimes accompanied by vasomotoric changes in the joints. One patient complains of pains in the joints when he is walking, another claims that

the pains disappear when he is moving about. Of importance for the diagnosis of such cases is the finding of pressure points and hyperesthesia of the skin over the articulation, especially when symptoms of an angioneuritic character, or fever—articular inflammation or gout—are present.

In *tabes dorsalis* arthralgias may also be observed, quite independently of arthropathy. They arrive in sudden attacks, persist for several days, sometimes for a whole week, are located in the joints of the big toe but radiate thence into other joints, and the affected parts feel hot, so to speak, a veritable "tabetic pseudogout." True gout generally comes on after midnight, but these tabetic arthropathies seem to prefer the time before or close to that period. Objective tabetic symptoms should clear up the situation.

Similar chronic arthralgias with local hyperesthesia of the skin may also follow in the wake of *chronic obstipation* (autointoxication). Remove the cause and the symptoms will disappear.

The case is different in articular pains, especially in the hip or knee, which arises from affections in the *small pelvis* or in the *abdomen* nearest the hip such as hemorrhoids, diseases of the rectum, the uterus, the bladder, the prostate or in chronic appendicitis, and are felt in walking or other bodily exercises, sometimes also during

the night or when resting. Objective changes in the joints are rarely found, but sometimes a slight congestion or a crunching sound in the joint may be noticed. However, muscular fixation, sensitiveness to pressure, an exquisite tenderness of the skin just over the affected joint, in fact in the whole field of the morbid segment and its corresponding nerve complex (n. ischiadicus, cruralis, saphenus) as well as paresthesias may be expected.

Certain arthralgias may also parade as masked *malaria,* while other recidivous forms spring from a chronic *osteomyelitic* focus near to the affected joint. In *rachitis tarda* we likewise hear complaints of articular pains coupled with unusual fatigue. Proof of hyperostosis (rosary formation) on the ribs, of genu valgum (both, however, of recent origin only), skiagrams of the bones, also the examination of lime balance should be winning features when forming the diagnosis.

Of great importance is the fact that arthralgias are a prominent symptom of *lead poisoning.* The blue line, clonus, the punctated erythrocytes, etc., are the residual signs.

Lead is a strong factor in many severe articular changes, e.g., *lead gout,* gouty tophi, gouty kidney.

A predisposition to gout is often the road that leads to arthralgias, or rather to articular

pain even though demonstrable and definable changes in the joints themselves are not apparent. Long before the real attack comes on, even before clear clinical signs of gout are observed, these cases will complain of transient, recurrent pains, lasting sometimes for days, in various joints or in one only, especially during changes in the weather. Purin tests, hereditary familial traits, observation of habitus in eating and living should be guiding points.

Arthralgias (toxic) as accompanying symptoms in *chronic uremia* are of rare occurrence. Objective sensitiveness upon pressure on the joints may prevail—it may also be absent. The seat of the pains (often intermittent) may be paraarticular. Whether there is an anatomical subtratum for these arthralgias I am not prepared to claim. But I can say this, that in none of my cases the articular pains were in the foreground, they were always surpassed by the other morbid sufferings and in consequence never an obstacle to the diagnosis.

Strictly distinct from these are the cases of true articular gout due to the retention of uric acid due to a preexisting primary or secondary granulation of the kidney. The differential diagnosis is selfevident.

Articular pains come also to the surface during the *climacterium* and in the *adolescent period* in girls.

In *adiposis dolorosa* they often make movement painful owing to the fatty deposits in the joints which are also very sensitive to pressure.

In *insufficiency of the thyreoid* the articular pains are outstripped by the other clinical signs.

Acute arthralgias are common in a whole series of *acute infectious diseases,* especially in their incipient stages, in all infectious pseudorheumatisms and in the *Wolhynian* fever.

Intermittent articular hydrops deserves mention here. It is partially due to nervous influences but also connected with acute swelling in the affected joint or joints. As a partial symptom of hydrops hypostrophos (*Quinke's* angioneurotic edema) (probably due to regional venous cramps), it appears at times in exchange with other manifestations of "exudative diathesis" .asthma, urticaria, mucous colic, vasomotoric rhinitis, etc.), sometimes also as an isolated condition, but principally in one of the kneejoints or in both (hydrops genus intermittens), or in some instances in one knee joint and one or more joints in different parts of the body. Fever or chills are rarely present, but often a sudden and considerable infiltration in the affected parts may be observed. This may not be inhibiting movement and quite painless, while in some cases the pains are very severe; and again, whilst in some cases there is a rise in temperature and the skin over the affected

joint is reddened, in other cases it does not lose its normal aspect. The course of the disease is about 8 days. In some women it sets in regularly before every menstrual flux; in other patients it appears with regular periodicity. The symptoms should be ample for the diagnosis.

But we must not forget that a hydrops chronicus articulorum and not any the less an intermittent hydrops articularis (the latter is also, as a rule, the manifestation of an idiopathic vasomotoric neurosis) may sometimes be only a deuteropathic disease. It is found, though rarely, in company with arthropathies in tabes and syringomyelia, with *Basedow's* disease and polycythemia, but may also be (but only when in an independent state) the expression of a tuberculous or heredo-syphilitic, more frequently of a gonorrhoic affection. Hydrops artic. intermittens of a joint, e.g., in the knee, may also be caused and sustained by a chronic osteomyelitis. The fact that in every instance only one joint is affected and the X-ray should be sufficient for proper recognition. In neurotic hydrops the affection settles now in this, but the next time in the contralateral joint.

Heberden's nodes, about the size of a small pea, are always connected with chronic arthritis or chronic gout. They develop on the proximal extremity of the ungual phalanx on the dorsal side of the finger, preferably the little finger,

either on one or both sides, are not always soft but of different degrees of hardness, rather painful and tending to become paresthetic. Their presence impairs the motility and gradually bends them in a solar or radial direction. As companions of gout or chronic arthritis they are common enough, but there is an intimate connection between them and an impairment of the genital functions such as the climacterium or the surgical removal of some internal genital organ.

The differentiation between *Heberden's* nodes and periarticular gouty nodes is not difficult, unless the latter grow out from the periosteum or the tendon roots in which case there is patent motility in the bones while in the *Heberden's* nodes there is none. In gouty nodes consistence and touch are telling signs; they have an elastic hardness of a granular, gravelly, warty, prickly quality, while the hardness in *Heberden's* nodes has a more even smoothness especially when they get older.

The seat of the affection differs strongly in both. *Heberden's* nodes choose the ungual phalanx particularly of the little finger; the gouty nodes and other articular affections prefer the pedal extremities, with predilection the big toe. In chronic gout or after acute gouty attacks these nodes are formed—but not exclusively—on the big toe, as they appear in other joints

of the lower extremities as well. The X-ray plainly shows a marked difference between the kinds of nodes. It is my experience also that *Heberden's* nodes which stand in causative connection with gout and arthritis are, forsooth, much more painful in themselves as well as sensitive to pressure, than those which follow upon impaired genital function.

Chronic arthritis of a very painful character is quite common in stout, fat women of the *climacteric age.*

Observe also that in this disease nodular formations resembling the gouty nodes will appear under the skin. The X-ray will plainly show the difference between them. (*Rheumatismus articulorum nodosus chronicus* as against rheumatismus artic. nodos. acut.) Spontaneous pain speaks for gouty and against rheumatic nodes.

A similar affection is *comptodactylia.* It consists of excrescences on the side of the bones of the medial phalanx, only at times painful. They assume sometimes spindle-shaped figures around the medial phalanx and seem to come into evidence with disturbances in the intestinal canal only to disappear again when the cause has been removed. I have never seen a case myself.

A disease of the central nervous system which is often erroneously taken for chronic arthritis, in fact, at the first glance strongly resembles it, is *paralysis agitans.* The fingers frequently

show the same position to the hand in both diseases, i.e., turned towards the ulna. Moreover, the patient not seldom complains in paralysis agitans of rheumatic pains in the extremities, and in both affections the tendon reflex is increased. These circumstances make allowance for the error. But in paralysis agitans we fail to find those changes in the configurations of the joints which are such salient features in chronic arthritis. We miss the swelling, the impaired motility of the joints between the ungual and middle, and also between the middle and metacarpal phalanges so characteristic in chronic arthritis.

As for the rest the special symptoms of paralysis agitans should be a sure basis for the diagnosis, viz., ague, muscular rigidity and the bodily posture of the patient. Put there are cases in which both diseases may simultaneously exist.

It is easier to avoid another error. The patient complains of stiffness in the joints and difficulty in moving them. *"Chronic arthritis"* is the first thought. But the real cause will be found in the skin which is drawn taut by *sclerodermia,* has lost its softness and pliability, in fact feels as if it were not long enough. Nevertheless sclerodermia may lead to arthritis.

To mistake chronic arthritis—*osteoarthritis deformans*—for *osteoarthropathie hypertrophi-*

ante pneumique Marie, seems to me impossible. I admit that the diagnosis may offer some difficulty when the latter affection is associated with articular troubles. But the clinical signs characteristic of the disease together with the Roentgen picture ought to be sufficient criteria. Still the error might be pardoned in the incipient stages of osteorarthropathy when the changes in the bones have just barely reached the cartilage in the joint caps with resultant pains and impaired motility in the joints.

Under "Pains in the Bones" I have already mentioned how an *arthritis deformans* may erroneously be diagnosed in *senile osteomalacia,* or *osteoporosis.*

A relatively rapid, subacute arthritis deformans with well-defined disfiguration and enlargement of the joint may be readily simulated by a primary, malignant neoplasm in the bone or by an epiphyseal or juxtaphyseal sarcoma of the bone-marrow, for we find here enormous swellings in the articular region, the articular outlines disappear almost entirely and the skin has a reddish, or rather bluish red appearance and is feverish, no doubt owing to venous stasis. But the fact that the motility of the joint remains unimpaired, even though pain (mostly slight) does exist, and the evidence of deformities in the bones, of ectatic veins in the skin of the affected part, together with the X-ray should

be the guiding points in the diagnosis. Only *a tuberculo-fungoid* or perhaps a *chronic syphilitic* (pseudo-tumor albus lueticus) *articular affection* or an *arthropathy* might become a disturbing element.

The differential diagnosis between an arthropathy and an arthritis deformans may always be based on the following lines: in arthritis deformans the beginning is slow, in arthropathy the attack is sudden and acute; in arthritis deformans the articular processes are impaired, impeded, in arthropathy they are enlarged (loose joints); in arthritis deformans the disfiguration is confined to the joint, in arthropathy it extends beyond; in the former there is pain in the joint, in the latter, as a rule, none.

In subacute or chronic, mostly exudative *monarthritis,* for instance, in the knee joint, we must always think of a subacute or chronic *osteomyelitis* as the provocative cause. This is generally located in the part of the bone nearest to the joint affected. Articular tuberculosis or articular neuralgia is very often the erroneous diagnosis in these cases. The seat of the trouble is centered in some purulent condition in the lungs or pleura. Roentgenographic evidence will assist the diagnosis.

When we hear of pains in the knee or hip joints we should not only think of local affections, among them genu valgum, varum, flexum,

recurvatum, but we must likewise give attention to reflex pains due to *flat foot.*

Coxitis shows pains in one kneejoint, a sign of importance not only for the surgeon, but also for the internist because the same conditions apply in osteoarthritis deformans of the hip as they do in coxitis. The examination should be based on the proof that when standing up the patient cannot properly abduct the femur in its joint. The X-ray will show the existence of an arthritis deformans in the hip joint when the knee joint is normal.

Sometimes it will be difficult to differentiate between *omarthritis* and *brachial neuritis.* (Cf. chapter "Pains in the Shoulders.") Here it is wise to make a thorough examination of the legs, especially the vascular regions. We may find then whether we have before us a case of arthritis of the hip joint or an acute osteomyelitis of the head of the femur or of the iliac section, or an arthritis of the sacro-iliac articulation or a rheumatism of the gluteal muscles. The latter will be recognized by the presence of pressure points (not always in evidence), accentuated pain in abdominal exertion, and the sciatic phenomenon. If we find that the sacro-iliac region is especially tender to pressure, that a quick and full lateral pressure of the pelvis sharpens the pain, we are dealing with arthritis of the articulation belonging to that region. In

arthritis of the hip joint the salient features are: deep seated pain, throbbing sensation in the major trochanter, the impossibility to hyperextend the hip joint to twenty-five or thirty degrees when the patient is lying on his stomach —always possible in normal individuals, but impossible also in spondylitis with abscess in a dependent part as well as in all forms of psoitis, likewise in retro-cecal appendicitis, impaired motility in the sense of abduction, especially in the standing posture, the disappearance of pain when lying prone (except in sciatica when pain is always present). Much may be learned from the Roentgen-ray picture. The constancy of the pains, leucocytosis and the fact that even cautious movement in the hip joint is not possible, speak for osteomyelitis and against articular affections. In rheumatism of the gluteal muscles—a rather rare disease—the spontaneous and pressure pains are diffuse, not only exacerbated by movements of the lower extremity, but also by simple, active contraction of the gluteal muscular plexus without locomotion of the extremity.

Headache (Cephalea, Cephalalgia)

The causes of headache are as numerous as the complaints thereof. To recognize headache properly and make a fitting etiologic diagnosis —without the latter there is no efficient therapy —we must always be alive to the fact that this affection is primarily located within or without the cranial sphere, but that the originating cause may be centered in a local, or distant part of the anatomy or may be quite general in its nature.

It is my experience that those headaches which are due to changes in the *cranial shell* are most frequently overlooked, because an important rule which I earnestly advise my readers to keep always before them, is so often overlooked, viz., "In complaints of headache make a thorough inspection and palpation of the cranium."

If you observe this rule carefully you will soon learn that headaches may have their origin in the *scalp*. Wounds, sores, inflammation, phlegmons concern the surgeon. To the internist of interest is erysipelas of the scalp. I do not mean the form which is so often an extension of facial erysipelas, but that affection which originates directly in the scalp itself from a

scratch or a fissure in the skin, with a dragging, drawing pain. Only a very painstaking search for such a scarcely noticeable lesion will lead to a happy diagnosis. Mark the localized reddening of the skin and the inflammatory areola—neither of these signs ever appear so distinctly marked in erysipelas of the scalp because the skin in this affection is drawn too tight—the localized pressure sensibility, glandular swellings and the symptoms of a general infection. The diagnosis is beset with difficulty in individuals of advanced age because in them the general manifestations, especially in the temperature are no longer so pronounced.

That form of headache which is called clavus hystericus, (*hysterical hairache*) is basic in a hyperesthesia of the scalp. The patient feels a sensation as if a nail were being driven into the head, or as if there were a wound, a sore on the aching spot. Sometimes the pain is quite diffuse, or centered in the very vertex, and the hair is sensitive to touch. When a woman pulls the hair upwards in combing there is pain in the scalp similar to toothache, especially on the top of the head.

Painful sensitiveness on pressure is a prominent symptom of *neuralgic headache.* But here the pain is confined to the zone in which the affected nerves are located. It generally follows two distinct lines, either along the trigeminus or

the nervus occipitalis, both major and minor. In consequence the local sensitiveness to pressure pain in supraorbital neuralgia corresponds with the supraorbital nerve process from the supraorbital foramen, in neuralgia of the nervus occipitalis to about the middle of a connecting line between the mastoid and spinous process.

In both a proper etiologic distinction between genuine and deuteropathic neuralgia must be made. If the latter is the case and supraorbital neuralgia is present, we must direct our mind to affections of the osseous surroundings of the *n.* supraorbitalis, to arteriosclerosis of the vasa vasorum of the nerves, diseases of the oral cavities, the eye, the nose and its cavities, the ear, the brain (tumors of any kind, also tuberculosis, gummata, multiple sclerosis, etc.), all of which are causative elements, especially preceding infectious diseases, principally malaria and syphilis, existing metabolic disturbances (gout, diabetes mellitus), colds and constipation.

In *cervico-occipital neuralgia* similar conditions prevail, but in addition we should look for affections of the cervical vertebræ, especially the first and second, also the spinal cord, but particularly for syphilitic cervical hypertrophic pachymeningitis in which pains also occur in the arms, and, in exceptional cases, for pains in the occiput. Neither must we forget localized affections in the posterior section of the head (cere-

bellum, medulla oblongata), radiations of pain along the neck into the shoulders and arms, diseases of the sphenoid sinus and all possible affections in the retromediastinal space (see chapter on "Pains in the Neck"). Only a careful scrutiny of all these conditions can assure us of a correct diagnosis.

If a patient complains of intense pains in the head which come on in sudden periodical attacks, last sometimes for two or three weeks on a stretch and are of a neuralgiform, shooting and piercing character, we should promptly suspect a case of *lancinating* pains in tabes cerebralis. Beyond the customary symptoms we may expect no nervous sensitiveness to pressure pain, no nervous pressure points, but rather objective sensibility disturbances in the sense of hypo- or anesthesia, resp. analgesia.

Headache of a neuralgic character may be a manifestation of a local lesion in any part of the trigeminal plexus or of any other encroachment in the frontal region. Circumscribed, chronic, local meningitides, basilar cranial and cerebral tumors and those of the angle of the cerebellar pons will surely give rise to neuralgiform headaches. The diagnosis results from the local and general symptoms.

Myalgia capitis is rheumatism of the scalp and the cause of acute, continuous, exacerbated attacks of headache. The diagnosis is guided

by the presence of hard, painful, lumpy indurations along the linea semicircularis where the muscles are attached to the cranium, especially the splenicus and m. cucullaris, also the sternocleidomastoid and scalenus. Very sensitive nodules in the panniculus cavernosus of the occiput and of the nape of the neck may also be felt. There is pressure pain in the periostea of the superior vertebræ, especially in the transverse processes.

The patient complains of a tearing pain in the head, exacerbated when chewing or speaking (also a symptom in neuralgia of the trigeminus), or by the pressure of a hard hat, or by combing the hair. The pain is sometimes relieved by a brisk movement of the head, but it may be also aggrevated by it.

In bald-headed people myalgia capitis is not uncommon, likewise in women who contract a cold from washing the hair, or when the hair gets wet through some accidental cause, going from a warm room into a colder atmosphere, or exposure to drafts or cold winds. The pain prevails chiefly in the back part of the head, rarely in the temples or forehead. A dose of a salicylic preparation, massage and wet bandages frequently bring relief.

The *crural part* of the *cranium* may be a source of headache. I mention here syphilis of the bones or of the periosteum. When osten-

sible gummata exist, the diagnosis offers no difficulties. But when these are not in evidence, we should look for the nocturnal appearance or aggravation of the pains especially before or around the midnight hour—always a strong hint for the syphilitic genesis of the aches (but not pathognomonic).

Such *headaches with nocturnal habits* are also found in *tumors of the brain,* especially in *aneurysms* of the *vertebral* and *basilar arteries,* in *uremia, arterial hypertension, dental affections* (caries), *glaucoma,* and also in *diabetes, hysteria* and *migraine,* not to speak of patients who work in the night time. Their nocturnal character together with the complementary corrective reaction and the results obtained from therapeutic measures should easily put upon them the stamp of *dolores osteocopi luetici.*

Osteocopic pains mature also in other morbid conditions of the osseous portion of the cranium, viz., *hyperostosis* (*leontiasis ossea*), in *neoplasms* (*chloroma, carcinoma, sarcoma*), *myeloma, famine osteopathy* (sensation of hammering in the skull). The pains are generally in the shape of attacks of an intermittent character, while in some cases they do not appear at all. *Caries* of the *cranial bones* and *cold abscess* also belong here.

Pyrgocephalus is only mentioned here casually. Fuller details will be given later on, be-

cause the pains in it are more in the form of migraine.

That in *traumatic periostitis* headaches prevail is self-evident, for in it impressions of the osseous cranium can be easily demonstrated by radiology. The cause of headache in cranial trauma is frequently focused in intracranial conditions. In some cases these pains pass away quickly and may be attributed to a transient minor circulatory disturbance or to some cellular encapsulation. In other cases the root of the evil may be traced to cerebral commotion, compression or contusion or to some hematoma of the dura or a traumatic rupture of the median meningeal artery, an intermeningeal hemorrhage or an abscess in the brain or a traumatic neurosis. The diagnosis is irrelevant in this place.

What is of interest to the internist is the fact that headaches with giddiness—especially when stooping down—nausea, abnormal irritability or fainting, also loss of consciousness and intolerance for alcohol are the inevitable after-effects of a previous concussion of the brain. This knowledge would forestall an erroneous diagnosis of "simple" traumatic neurosis in many cases. Headache with increasing lumbar pressure indicates very often chronic serous posttraumatic meningitis, even if only as a localized condition. An important symptom in all these cranial affections is a persistent hypersensitive pressure

zone endowed with the sensation of a hot sponge wiping over it.

We now pass over to that chain of intercranial affections without trauma which may be the source of headaches. With the exception of cerebral atrophy all other diseases that occur in the cranial cavity, of whatever nature, are here included. We shall only deal now with those endocranial headaches that are determining diagnostic factors.

The diagnosis of *chronic periostitis* can be made absolute by the X-rays, unless there are external perceptible signs of osseous changes or some definite symptoms of a cerebromeningeal lesion. The same may be said of intracranial osteoperiostitis. In fact it is the only means at our command whereby we may obtain undoubted results, for a differential diagnosis between syphilitic ostitis, between the dura and the roof of the cranium on the one hand, and pachy- or gummatous leptomeningitis on the other can hardly be established.

The diagnosis of *internal hemorrhagic pachymeningitis* is beset with many difficulties. It occurs in marantic individuals, in nephrosclerosis, in heavy drinkers and in old people. Apart from the conditions mentioned above we find the following: the patient complains of headache, periodical vomiting, befogged consciousness, paretic conditions of certain cerebral nerves, pupillary

differences and disturbances in speech. The body temperature remains unchanged. Important for the diagnosis, especially for the differentiation from an intracerebral affection is the fact that there is a marked contrast between the cerebral paresis or paralysis of the extremities and that of the respiratory muscles of the thorax, and that the patient breathes with more ease on the affected side.

Of course, this does not establish a differentiation from a convexity leptomeningitis which runs a course similar to pachymeningitis. But worthy of note is that *internal hemorrhagic pachymeningitis* has a partiality for clinically one-sided external, irritating symptoms, whilst in the other forms of convexity meningitis these manifestations bear a much more generalized character. Hemorrhages do not always occur in any of these varieties, especially not in the earlier stages of the diseases. Then there is the diffuse form with a limited partial symptom complex, basic in a stronger, unilateral meningeal progression or also in a partial encephalitis or vascular lesion (thromboarthritis).

Still more distressing are the cephalic pains in all forms of *leptomeningitis.* The accession of throbbing pains is a telling symptom.

Cephalalgia is a frequent initial symptom in acute (*epidemic*) *encephalitis* and one of its after-effects.

In *multiple sclerosis* the pains come in recurrent attacks, now from a frontal, now from a vertical, or again from an occipital direction. At times they are of a very intensive character and accompanied by vomiting and giddiness.

The acme of pain in the head is reached in three other intracranial morbid processes, viz., in *tumor of the brain* (including cysticercus and pseudotumors), in *chronic abscess of the brain* and in chronic *serous meningitis.* When the intracranial pressure, which is generally associated in a violent form with these ailments, is, however, of a more moderate character, the pains will also be lighter in proportion. This puts the stamp of a weighty symptom on the initial headaches in *acromegaly.* It assumes here mostly the form of a dull pain in the frontal region, perhaps only on one side, whilst in other hypophyseal diseases the occipital portion is rather involved.

Cerebral vomiting frequently occurs in all these affections and is a stereotyped signal for the diagnosis. The pain is aggravated by other accompanying symptoms such as muscular exertion in coughing, sneezing, defecation or stooping down, all of which tend to increased pressure on the cranium from within. Another source of irritation is the intake of alcoholic stimulants. In chronic serous meningitis the pain is more diffused, whilst in cerebral tumors it either

spreads over the entire cranium, or at any rate, if localized, is more intensely punctuated in a certain spot, in which case, however, it must not be accepted as an indication of the real seat of the tumor. Even a neuralgiform pain in the trigeminal zone is not an unconditional local symptom. In cerebral abscess the site of the pain corresponds with the local suppuration thus pointing out the seat of the disease, but not invariably. In both, tumor as well as abscess of the brain, the pains are nearly always of the deepest intensity when located in the posterior portion of the cranial cavity they encroach on the circulation of the vena magna *Galeni* and in the aqueduct of *Silvius*. *Magendie's* foramen being occluded a similar effect results in meningitis.

The impairment of the venous circulation, often of diagnostic significance, in tumors of the brain, only partially explains why the pains are aggravated when the head is held in a certain position. This is particularly so in cerebellar tumors. When the patient rests the head on the affected side the pains set in, at least in the beginning of the sickness. This is also the case in cysticercus of the fourth ventricle. The patient gives his head a forced position, sometimes to the right or left, sometimes forward or backward, in order to make the pain more tolerable. The same happens also in renal headaches.

When, however, an occipital pain is very much exaggerated by a forward inclination of the head, when the patient has the sensation as if the skull would burst, we must look for another cause. We should be prepared to find a compression of the aqueduct of *Silvius* or an acute hydrocephalus of the fourth ventricle.

A point of value for the diagnostician is the observation that in some rare cases the pains are of a strongly remittent even intermittent character. An erroneous diagnosis of neurosis is here not excluded.

There are many cases of this kind which are very difficult to diagnose. We should never be rash in diagnosing neurosis from remittent or intermittent violent attacks of headache persisting for days or weeks, but rather direct our thoughts to serous meningitis or brain tumor or cerebral chronic abscess, even when the ailment drags through several years. The same idea should be followed when an undoubted trauma has preceded the apparently "neurotic" symptom complex. Every practitioner with some clinical experience has seen cases in which chronic serous meningitis followed on the heels of some cranial traumatic affection running its course exclusively under neurasthenic conditions such as persistent or else periodic headache, backache, giddiness, abnormal irritability and psychic emotions. And does that not happen also in

chronic post-traumatic abscess of the brain? Even light, continued headaches may constitute the solitary symptom of a hitherto latent cerebral abscess.

There are other forms of periodic headaches which build up from ascending intracranial pressure. More about this when we are dealing with *infectious diseases.* In passing I will only mention here that in an analogous manner certain cephalalgic attacks in *chronic alcoholism* may be explained, especially when they are associated with tinnitus, stupor, nystagmus, amblyopia without ophthalmic conditions at times aggravated by coughing or sneezing. They yield, as a rule, to lumbar puncture. The etiologic diagnosis is made positive when tremor of the hands and tongue, sleeplessness and morning sickness present themselves.

Headache in *hysteria* or *neurasthenia* cannot be accepted as a pathognomonic manifestation *per se.* In the majority of these cases the pain is nothing more than an indefinite cephalic pressure covering either the whole circumference or only portions of the skull (forehead, temples or occiput), although in neurasthenia some patients complain of external constraint such as might be caused by a hard hat, or a vice or a circular band, while in hysteria the feeling is more as if the head would burst from within; there is also hyperesthesia of the scalp and hairache.

Hysterical headaches may also assume the form of clavus—a sign which is unknown in neurasthenia—aroused sometimes by the rays of a glaring light or some loud noise.

But there are cases of hysteria in which the headache simulates the character of a neuralgia by running along the nerves of the cranial roof into those of the occiput and branching out even as far as the nape of the neck or else into the frontal section and the face itself. The differential diagnosis may be drawn from the fact that the headache is not confined to the path of the nerves but radiates into adjacent ground, that the head feels as if ready to burst in two from within and that classical pressure points are missing—all characteristic symptoms of hysterical and non-neuralgic conditions.

Neurasthenic headache sometimes attended with slight excitability or weariness or wakefulness is not uncommonly the sole symptom of neurasthenia. But there are other signs which may help in the diagnosis, viz., mental overexertion, psychic or sexual hyperexcitement, late hours, and changeable weather conditions, more acute in the early hours of the day; also the fact that unlike constitutional forms of headache, e.g., migraine or brain tumors, it is improved by or disappears altogether with open air exercise or walking or riding bareheaded.

Hysteroneurasthenia should not be overlooked

in this connection. *Arteriosclerosis* of the *cerebral vessels* presents not only headache but also other cerebral symptoms which are analogous to neurasthenia. The headache is chiefly localized in the frontal region and is of a piercing, boring, shooting, dull character, accompanied by giddiness, loss of memory, sleeplessness, irritability and fatigue. In all these qualities it strongly resembles neurasthenia, a circumstance that makes the diagnosis difficult. The minor symptoms such as dizziness, paresthesia of the extremities, inhibition of speech, changes in the handwriting, vivaciousness, relate rather to arteriosclerosis, although they are not foreign to neurasthenia. Arteriosclerotic headache may easily be caused by a physical strain in coughing, sneezing or during defecation, that is to say by every form of increased blood pressure, while on the other hand this does not seem to be the case in neurasthenia.

Other distinguishing signs are: it mostly comes on in the early morning hours, starting the patient out of his sleep, like a thunderbolt, sometimes it bears the character of migraine. In spite of all this, I mean to say that the determining factor will always be found in the etiology of the case, especially in face of the fact that the presence or absence of peripheral arteriosclerosis does neither prove nor gainsay the existence of a sclerosis in the cerebral vssels. If we fail to

discover the causative force of the apparently neurasthenic conditions (mental overstrain, psychic emotions), and if the patient is advanced in years (beyond the fifties), I should be in favor of diagnosing pseudoneurasthenia basic in arteriosclerosis resp. pseudoneurasthenic headache. Nevertheless, arteriosclerosis has been observed in younger persons, especially after some preceding infectious disease such as syphilis, malaria and toxic conditions (lead poisoning). Neither must we overlook the fact that a genuine attack of neurasthenia may spring from a preexisting demonstrable *arteriosclerosis* of the *afferent vessels.* Still the diagnosis of such a combination would not be justified unless there is unmistakable proof of the provocative cause.

If the patient begins to complain of headache we must consider the likelihood of an additional cerebral complication such as *thrombosis* of the *cerebral arteries* or *cerebral hemorrhage.* Either of these may occur in older persons with a contracted kidney, and in younger individuals with arterial calcification. Such headaches are, moreover, a valuable symptom for the differential diagnosis between thrombotic softening of the brain and cerebral hemorrhage. If the pains have prevailed for hours or days before the attack of cerebral hemiplegia set in, they point to thrombotic softening of the brain. In arterio-

sclerosis the headache does not always bear the neurasthenic character but rather assumes that of a boring, shooting or stitching sensation, especially after psychic emotions.

Similar manifestations are observed in the incipient stages of *syphilitic endarteritis* of the brain; likewise in progressive paralysis, the earlier epochs of organic tuberculosis—chiefly pulmonary — in *Addison's* disease, contracted kidney or in some of the toxic affections. The pains may mimic neurasthenia and thus create a pseudoneurasthenic symptom complex.

Sclerosis of the cerebral arteries frequently opens the road to the formation of aneurysms, especially in the sphere of the basal vessels. The evidence of combined general pressure and local compression symptoms (e.g., in aneurysm of the vertebral or basilar artery of bulbar symptoms) is of great value for the diagnosis. If blood or clods are found in the fluid obtained by lumbar puncture the diagnosis is absolute.

Dementia precox, progressive paralysis and other mental affections are capable of creating a "pseudoneurasthenic" symptom complex with distressing headaches. It is often clinically difficult to distinguish between neurasthenia and an early progressive paralysis. The character of the headache may here be a determining factor. In neurasthenia the pain is rather in the form of pressure, although psychic emotions or fatigue

may give it the nature of a real ache; it is sharper in the morning, tapers down as the hours pass by and vanishes with the setting sun; it obsesses the patient with the fear of "going mad."

In paralysis the pressure in the head is not so predominant, but we find rather a progressive weakening of memory and striking changes in the personality of the patient. And yet, progressive paralysis may wear the mask of a cerebral asthenia and may be accompanied by sleeplessness and very severe pains in the head. But etiology and the somatic symptoms should here help us out, viz., change in the pupils, the missing light and pain reaction, weakening of the patellar reflexes, trembling of the lips, anomalies of speech, change in the hand-writing, not to forget lumbar puncture. *Nonne-Appelt, Wassermann,* plyocytosis, all positive. Nevertheless a positive *Wassermann* reaction should be accepted with caution, for syphilitic patients are frequently neurasthenic.

In *dementia precox* there is more pressure, a fullness of the head, than direct pain. Abnormal fatigue, impaired capacity for work, noticeable deterioration of the mental faculties, are important points to watch. The anomalous hyperexcitability of the neurasthenic is lacking. While in neurasthenia nervosity runs parallel with external stimulation, the nervous spells in de-

mentia precox come on like explosions to make room immediately after for dull inactivity.

Of course, all *melancholic* and *depressing psychoses* carry parasitic headaches.

Anemia, no matter what its genesis may be, *and hyperemia* of the *brain* arouse pains in the head. In anemia the pains are not often of a very intensive character, but may appear in different localities, and in company with other manifestations such as vertigo, fainting fits, tinnitus aurium, nausea, especially when the patient suddenly sits up. They are aggravated when the head is held erect or when the abdominal muscles undergo a strain, but soften down with a horizontal position or deep inclination of the head. In anemic women this headache is a steady visitor during the catamenial period. This is important for the reason that such a pain, localized as it is in the occipital region and associated with the other symptoms already enumerated, may very well point to some internal hemorrhage.

If we find, however, very severe headaches in the anemic patient we should at once suspect higher pressure in the cerebrospinal fluid, or some complication such as thrombosis of the cerebral sinus especially when other symptoms, impaired consciousness, fainting fits, accede.

A special form of cerebral anemia with headache is caused by *insufficiency of the aorta.* The

quick pulse of the cerebral arteries, the in- and outflow of the cerebral blood produce the sensation of pressure simulating neurasthenic pains in the head which are of a throbbing and very distressing character, very much in the nature of similar throbbing epigastralgias in failure of the cardiac valves. Since the patients afflicted with the latter diseases are likewise abnormally excitable and suffer from sleeplessness, we have again a pseudoneurasthenic symptomatic picture before us.

Anemia of the brain need not necessarily be a partial manifestation of a general anemia, nor the result of defective organic circulation, but may result from local vesicular changes. A considerable *contraction* of the *os art. anonymæ* or of the *carotis sin.* either due to sclerosis of the aorta, syphilitic arteritis or complete thrombosis of these arteries, will surely lead to anemic headache with vertigo.

Hyperemia of the *brain* is another source of headache, but only in those cases in which there is *polycythemia rubra.* The patients complain of pressure and fullness in the head, the pains are sometimes very severe, sometimes migraine-like, or of a throbbing character, often there are congestions combined with scintilating scotoma, tinnitus aurium, giddiness, sleeplessness, abnormal excitability, in other words all the "pseudoneurasthenic" symptoms again as men-

tioned before. Blood test and a proper clinical examination should make the diagnosis clear.

The situation is very much the same when there have been reiterated *ruptures* of *bloodvessels* followed by pain and dullness in the head and clumsiness in the performance of the daily tasks. We find it frequently in patients suffering from hemorrhoids. A good hemorrhage from the piles gives relief all around and clears the head of painful conditions.

Passive hyperemia of the skull can only then be considered a cause for giddy headache when it is of a chronic character. The pain as a rule is light, but may become bothersome when it spreads over a larger area which is really the case in *venous hyperemia* owing to insufficiency of the right ventricle. In other words we are dealing here with a high grade cardial congestive cyanosis of the brain, mainly by relative insufficiency of the tricuspid valve and by direct restriction in the circulation of the vena cava superior, no matter whether this restriction is caused by pressure from a mediastinal tumor or a chronic mediastinitis or a thrombosis of the vein or of the right vestibule. The remaining symptoms of the congestion of the vena cava sup. (see Cyanosis and Edema), the direct mediastinal manifestations, the *Oliver-Cardarelli* symptom and the Roentgen-ray should suffice to trace the real cause of the headache.

It is not hard to understand how a *steadily rising pressure in the brain,* i.e., increased venous stasis (abdominal pressure, coughing, inclining the head forward, or stooping, etc.) aggravates the pain sometimes to a point where the patient feels as if the head would split. We can observe this in every cerebral cephalalgia, in arteriosclerosis in chronic alcoholism, in vasomotoric affections, in cerebral arteriosclerosis, in affections of the nasal cavities, pressure in the cerebrospinal fluid or in the arteries or in cerebral hyperemia. In conditions due to visual refraction these pains do not seem to occur.

Men who wear high collars, too narrow around the neck, and women who lace too tightly are likely to suffer from headaches owing to restricted venous circulation.

In acute venous cerebral congestion headaches do not, as a rule, play a prominent part. The complaint is more of pressure and dullness than a veritable pain.

But in *phlebitis* and *thrombosis* of any particular cerebral sinus severe headaches are symptomatic, for here we are confronted in nearly all cases by pseudomeningitic conditions. The diagnosis is greatly assisted when we find a collateral edema on the outside of the skull—in thrombosis of the longitudinal sinus a swelling of the veins at the roof of the skull, in thrombosis of the cavernous sinus an edema of the

eyelids, in thrombosis of the sigmoid sinus an edema of the mastoid process—and when we give due consideration to the appurtenant etiologic factors (nasal and aural and accessory cavities, erysipelas of the scalp, marantic, anemic thrombosis).

Periodic fluctuations in the blood stream give rise to *vasomotoric headache.* In the *vasoparalytic* form it assumes the nature of rushes to the head caused by certain *acute intoxications, alcohol, chloroform, nitrites,* especially *amyl nitrite,* and *chronic theism.* Furthermore there are other morbid conditions in which vasomotoric changes are liable to occur, viz., neurosis in both sexes, especially the climacteric form in woman, neurathenia, hysteria, traumatic neurosis, *Basedow's* disease, chlorosis, etc. The pain in these cases generally concentrates in the region of the vertex and is accompanied by various vasomotoric manifestations (erythema, dermographism, red streaks where the garments pinch the skin, changes in the complexion), cold hands and feet, chilliness, heat, inclement weather conditions. The vasomotoric hyperirritability of the skin accompanying these headaches is also peculiar to serous meningitis.

The *climacteric headache* is generally localized in the occipital area. Its true signs are easily recognized: its seasonable advent (sometimes months or years before the cessation of

the menses sets in), congestion, rush of blood to the face and head, unprovoked breaking out of profuse perspiration, irritability, anxiety, worry and general depression. Quasi climacteric neuroses may also occur in the male.

There is also a *vasoconstrictor headache* of an acute (mainly pressure in the head) as well as chronic character, e.g., *nicotinism*. No doubt, some of the passing symptoms of *cerebral arteriosclerosis* may very well be reduced to some vasoconstriction. We all know that in arteriosclerosis the nerves of the vessels, especially the vasoconstrictors, are much easier stimulated than is normally the case, and that functional hyperirritation is often a prominent feature in an anatomical affection of the vessels. Vertigo and periodic headache are part of the symptoms of cerebral arteriosclerosis which manifest themselves in the union of these two factors. This explains also the fact that in abnormal vasomotoric conditions both vasoparalysis and vasoconstriction may give rise to the same manifestations of headache. That vasoconstriction is the provoking cause is proved by the fact that fainting is often associated with it.

Abnormal innervation of the vessels, abnormal distribution of the blood either due to anemia or hyperemia of the cerebral arteries, are indubitably the originating factors of that headache which is so common in *overworked men and*

women in whom no anatomical affection exists. The slightest excess, even a small dose of alcohol, or a pipeful of tobacco may bring on the headache in such people.

Vasomotoric disturbance is the excitant of headaches in *menstruating women,* but the characteristics of a concomitant migraine are not present in these cases. We observe the same in young girls at the time of pubescence, when vicarious bleeding from the nose is coupled with the pains in the head before the menses have assumed their proper rhythm. Nausea, vomiting, giddiness come along with the headache and disappear with the epistaxis.

An anatomical *lesion* in the *sympathicus* is another originator of vasomotoric pains in the head. If the complaint is on the right side of the head and is combined with pains in the right arm and hypodrosis in the right axilla, then in a case of aortic aneurysm it can only be due to pressure on the marginal trunk of the right sympathicus or to perineuritis of the sympathicus arising from periaortitis.

In a case of *ulcus ventriculi* I had occasion to observe the following symptoms. The ulcerous attack, came on year after year with increasing virulence; with them pains in the right inside of the head set in, but disappeared with the removal of the intestinal trouble by gastro-enterostomy.

Some patients who suffer from *muriatic anacidity* are subject to headaches when the stomach is empty. It goes away with the intake of food.

Still another cause for headache we find in *arterial hypertension* and in all morbid processes that lead to it no matter whether the hypertension itself is essential or deuteropathic, painful or painless in its nature. The seat of the pains in the head may vary according to circumstances. If they are connected with a protracted case of arterial hypertension they are wont to set in after midnight, i.e., in the early morning hours or at awakening. In some cases they are the only sign of the existing disease, but in others they are associated with sleeplessness, dyspnea, precordial pains (pressure, anginoid troubles, polyuria).

Headache in *arterial hypotension* combined with weak heart action or venous congestion I have never been able to observe.

If, what is today called *"pseudouremia,"* comprises also the vessel crises described above, then it includes also the same list of symptoms (headache, giddiness, transitory amaurosis, aphasia, strokes, cramps, *Cheyne-Stokes'* respiration, mental disturbances), and also increased cerebral pressure owing to edema (chloremia) which may be spotted by lumbar pressure.

Needless to say headache is a steady com-

panion of epilepsy. It comes at times in the shape of a sudden stroke on the head, and travels in company with dizziness, nausea, vomiting, temporary loss of consciousness. Without these symptoms we have rather a case of *petit mal* before us. The pains in the head, as a rule, go away after the epileptic seizure. I may point out here the fact that cocaine is contraindicated as its administration may provoke an epileptic fit or at any rate an epileptic aura including giddiness, nausea, vomiting, palpitation of the heart, dyspnea, tremor and sever headache.

Pain, especially in the occipital and vertex region, is a frequent and lasting complaint during the whole time of mental stupor in *myxedema.* The evidence of psychic and cutaneous changes, hypothermia, the thickened tongue and the effects of thyreoid extract should clear the situation without much delay.

The same holds good in cases of *hypothyreoidism* which often simulates neurasthenia or anemia. Dryness and roughness of the skin, trophic disturbances in the hair and the nails, lassitude, hypothermia, insufficient perspiration, moodiness, and treatment with thyreoid extract, apply here also.

Adrenal insufficiency and its substitute *Addison's disease,* follow in line. In both diseases headache is of a very severe, piercing character not only during the whole course of the disease

but especially also in the "pseudomeningitic" final stadium.

True (functional) *orthototic albuminuria* in youthful persons lays claim to headache, early fatigue, despondency, giddiness and proneness to fainting. The headache in these cases may have in part a vasomotoric basis, but it seems to me rather due to some abnormal, constitutional, degenerative condition. The diagnosis is extremely easy to make, but according to my own experience it is rarely made. If a differentiation is to be made between the lesional and merely functional forms of the disease we should look for granulated or epithelial cylindroids, large numbers of erythrocytes and a typical chondritis.

Headache Due to Distant Causes

Headaches due to ocular conditions. Overstrained accommodation in refraction anomalies, in hypermetropia and astigmatism, abnormal convergent straining (exophoria), inflammatory affections of the eyes, especially glaucoma, working by bad light, all these are possible causes of pains in the head. In arthenopia the patient complains also of pains in and above the eyes, also in the forehead, that the headache is aggravated by prolonged work, stops during the night time, and when present resembles a supraorbital neuralgia. When in glaucoma vomiting

is witnessed the erroneous diagnosis of intracranial disease with increased cerebral pressure is apt to creep in much to the detriment of the patient. It is not unlikely that some cases of adolescence and puberty headache are attributable to overstrain of the visual apparatus.

The same as in eye troubles, so the pains are located in the forehead when the *frontal sinus* is implicated either in a suppurative or inflammatory process. They are apt to create the impression of a supraorbital neuralgia and are of a diffuse character, generally felt at the root of the nose between the eyebrows, chiefly in the early morning hours. We shall not go astray in the diagnosis if we keep a careful watch on the following points: not only the nerve along its course is tender to pressure, but the whole frontal area is sensitive on percussion, the patient complains of a raging, throbbing pain in the forehead at large, the nose is obstructed, previous or parallel *affections of the nasal cavities* (acute or chronic rhinitis, the usual secretions are hardened, polypus, spurs, spikes or other deformities of the septum or bones). Adrenalin or cocaine give relief. Thorough examination of the nasal cavities and transillumination of the frontal sinus are prerequisites.

All this applies with like force to acute as well as chronic diseases of the *sphenoid* and the *turbinated bones,* also to any affections of the

accessory nasal cavities. In catarrhal inflammatory conditions the pains are sometimes very severe and distressing. Influenza is especially marked by dull pains in the vertex region when they are due to ethmoiditis, but when arising from the sphenoid sinus they settle behind the eyes and are made worse by mental work or physical exertion.

Pains in the anthrum of Highmore radiating to the head are likely due to some suppurative process in the maxillary sinus giving rise to meningeal or phlebitic complications.

Diseases of the outer or the inner ear (furunculosis, foreign bodies) always afflict the patient with pains in the head. Sometimes they are only of local signficance within the radius of the affected part of the ear, but, as a rule, they permeate the whole of the corresponding side of the head. If of a diffuse nature they settle also in the occiput, rarely involving the entire cranial cavity. And again, they attack the region of the visual organs with local pressure and delicacy to percussion around the ear, especially above the mastoid process in inner affections. In acute, and more so, in chronic lesions of the middle ear (cholesteoma, suppuration of the bones of the ear) they involve the brain, and form, perhaps, the primary symptom of a sinus phlebitis, cerebral abscess, or purulent meningitis. Lumbar puncture, or eventually trephin-

ing, often stop these very distressing headaches efficiently.

Malignant growths and other affections in the *larynx* or *pharynx* are also originators of bothersome cephalalgias.

Retronasal angina and morbid conditions of the tonsils and of the teeth belong here. Acute occipital neuralgia with pains ranging from the nape of the neck to the occiput point to acute angina. Chronic pharyngeal affections (adenoids, tuberculosis or carcinoma of the pharyngeal wall) give room to chronic pains in the occipital or frontal regions. Headaches arising from sick teeth, especially from caries of the molars or the difficult passage of a wisdom tooth, are generally localized in the temporal area, but may also be in the ear itself (simulating an attack of otitis) or in front of the ear with irradiations into the corresponding cheek, and often supersede in violence the toothache itself.

Among the affections of the oral cavities I wish to mention chronic infectious conditions such as chronic purulent tonsilitis, chronic dental stasis, all of which may lead to chronic sepsis, loss of appetite, pallor, subfebrile temperature, chills, sweats, and a general feeling of indisposition and in consequence to toxico-septic headaches which disappear with the removal of the originating cause.

Another focus from which headaches derive their origin we find in the internal organs, especially those in which urinalysis is demanded.

In *kidney affections* headache easily proves the distinguishing sign of *uremia,* pointing often to the probability of an existing cerebral edema running a parallel course with the edematous condition (nephrosis), or of cerebral arteriosclerosis (pseudouremia) or of azotemia. The occiput is the habitat of uremic headache which, like all the other symptoms of uremia, is associated with vomiting, and generally comes on in the morning. That is the reason why it is so frequently mistaken for idiopathic migraine. A proper consideration of the other uremic symptoms, urine, blood (residuary nitrogen) and blood pressure tests should obviate all errors. For quite some time I have held the opinion that headache in chronic nephritis is of toxic origin. No doubt a good deal of it is due to high pressure tension. Moreover, in nephritis with or without uremia there are many complications which may give rise to pain in the head, such as uremic meningitis, uremic cerebral edema, cerebral hemorrhage, the possibility of encephalomalacia of thrombotic or embolic origin, inclination to internal hemorrhagic pachymeningitis. Headache may also set in at the incipient stages of acute glomerulonephritis as a manifestation of acute infection.

Headache will also occur as an "anaphylactic" phenomenon in the rapid *resorption of hydropic fluid in edemata* and in hepatic insufficiency (hepatargia, anhepathia). In the latter case, however, they are, according to my own experience, more of secondary importance than in the other cerebral disturbances (numbness, cramps) and in the manifestations of hemorrhagic diathesis.

In every kind of *gravidity toxicosis* headaches will be present. They are complementary symptoms in *hyperemesis gravidarum,* and still more so in eclampsia gravidarum and atypical toxicoses in the pregnant. The latter, as a rule, starts in with cerebral symptoms, with icterus, oliguria, even anuria and also hematuria, nearly always accompanied by albuminuria and cylindruria and—in contradistinction to eclampsia—without spasms leading to coma. But I have my doubts whether these headaches which manifest themselves in some women at the beginning of every pregnancy, can be always reduced to toxic origin.

In *diabetes mellitus* some patients suffer from headache. Sometimes it is a generalized pressure in the head, but frequently it assumes the character of a well defined pain, mostly in the frontal region, often enough, indeed, in the very center of the cranium. At times it resembles neuralgic pain—all diabetics are inclined to neu-

ralgia. This is mainly an expression of the existing acidosis often making its appearance only in the night time or at any rate with more pronounced severity at that period. It is worthy of note that very severe, at times, insufferable pain in the head in the course of diabetes mellitus is often the forerunner or admonitory sign of a diabetic coma. The diagnosis will be guided by the concurrent clouding of consciousness, by epigastralgia diabetica, meteorism, deep, retarded breathing and above all, by a correct urinalysis showing the presence of acid intoxication of the organism (acetic acid, oxybutyric acid, increased ammonia content).

The situation may become more complicated when the coma is compensated by an acute affection which alone gives rise to headache. I remember a patient who had been suffering from diabetes mellitus of long standing and who suddenly became feverish owing to an acute periostitis in an upper molar. The second day he complained of violent pain in the head attributing it to the diseased tooth. But a careful clinical observation soon showed that the real cause was a coma which developed during the next twenty-four hours. On the other hand an internal hemorrhagic pachymeningitis or a tuberculous meningitis may be the inciting element of terminal headaches in diabetics.

Chronic obstipation is another source from

which pain in the head may spring in the shape of a feeling of pressure, neuralgiform or real, violent neuralgic pain either in the trigeminal or in the occipital region. Many of these obstinate cases yield to an efficient rigorous cathartic treatment.

In cases where obstipation is in the main only coupled with pressure in the head a diagnosis for intestinal auto-intoxication is in my opinion not warranted. I would rather seek the cause in an existing neurosis, which would also explain the complaints of lassitude, dizziness, restless nights, etc.

Retention of flatus, intestinal flatulent dyspepsia following chronic intestinal catarrh are apt to produce pain in the occipital region. Diagnosis is rendered easy by means of adjuvants.

Gout, resp. uratic diathesis, is not of great moment in this connection. In my own practice I have not been able to substantiate the existence of specific gouty headaches or neuralgias either in the occipital or trigeminal region. When headache and gout run concurrently I should prefer to attribute the headache rather to neurasthenia which thrives in gouty subjects, or to arteriosclerosis of the cerebral vessels, or to disturbances in the intestinal canal, to arterial hypertension or insufficiency of the kidneys, unless other causes (alcohol, nicotine, etc.) are

in evidence, barring, however, headaches which are the forerunners or companions of acute articular gouty attacks (podagra).

Headache is also conditioned by not a few of the *exogenous toxins* (*coffee, tea, nicotine, lead, arsenic*). All poisons which produce cerebral morbid symptoms will also cause headaches (narcotics, carbonic oxid, botulism, etc.). Similar excitants of the so-called vasoparalytic headache, are alcohol, chloroform, amylnitrate, nicotine. The prolonged use or misuse of certain toxic substances may lead to headaches (tea, coffee). They all are the producers of pseudoneurasthenic troubles, i.e., pain in the occipital region, pressure in the head, dizziness, anginoid attacks, fainting fits, profuse perspiration, gastralgic crises, pallor, emacition, obstipation, aphasia, hemiparesthesia, etc. Presence of tremor, dislike for tobacco, etc., should assist the diagnosis. (That first smoke!)

Excess in the use of the weed may also be the father of other pains in the head such as migraine, neurasthenic, hysterical or arteriosclerotic pains, likewise of headaches caused by cerebral affections, e.g., meningitis serosa.

Chronic lead poisoning may easily be mistaken for some kind of neurasthenia as both have in common the manifestations of slight pressure in the head, sensation of oppression, weakening of memory and general lassitude. This error will

creep in when the possible cause of chronic lead poisoning is far removed from the minds of patient and physician as well, for in many trades lead is only used in small quantities, for instance, in cosmetics. But findings of the blue line, pallor, presence of punctuated erythrocytes, of colicky pains in the bowels or of arthralgia, likewise muscular weakness in the radial region should decide the correct diagnosis. These symptoms must also be watched when saturnine encephalopathy lies in the wake of plumbism. In the differential diagnosis between lead poisoning and leptomeningitic headache is of little moment, but in progressive paralysis (alcohol paralysis) it becomes a determining factor, viz., presence of pseudoneurasthenic, moderately acute pressure in the head, as against most violent headaches in the former.

Arsenic poisoning, especially with arsenous hydrogen, is also associated with headaches, especially in the forehead, with lassitude, dizziness, sleeplessness and loss of appetite. The presence of inflammatory lesions of the conjunctiva and respiratory mucous membrane, gastrointestinal symptoms, neuritic manifestations preferably in the lower extremities, with well-defined strong pains and trophic disturbances in the skin, and dermoid formations, and in herpes zoster awaken the thought of arsenic poisoning. Laboratory tests of feces and urine furnish the final proof.

I remember the case of a patient who was afflicted with severe arteriosclerosis and a granular kidney and strong arterial hypertension. Periodically he suffered for weeks from want of appetite, nausea, vomiting, headaches, cramps in the calves, a peculiar sweetish taste in the mouth. The patient who possessed unusual intelligence suggested arsenic poisoning. I myself diagnosed chronic uremia because the patient's breath had the characteristic uremic odor. The laboratory tests showed traces of arsenic in feces and urine. Upon closer investigation the real cause of the trouble was discovered. He had a number of stuffed birds and also upholstered chairs in his room all of which contained arsenic as a protection against moths. Still I adhered to my diagnosis of chronic uremia but complicated with arsenic poisoning.

Mention must be made here of *nitrobenzene* poisoning (an imitation oil of bitter almonds, also called essence of mirbane, used in the manufacture of perfumes). Predominant signs are: headache, transitory disturbances in the central nervous system and manifestation of hemolysis (icterus).

Very stubborn headaches will be encountered in workers with *vanilla, carbon disulphid* and *quicksilver.*

Raging headaches will at times follow *spinal*

anesthesia, generally accompanied by sleeplessness and delirious or pseudomeningitic affects.

It is proper to mention here also poisoning by *carbonic monoxid* gas. It develops from leaky gas pipes in the gas works (for heat or light). The symptoms are: headache, dizziness, lassitude, sleeplessness, numbness, epileptoid attack, and also glycosuria. It is of value to know that the pains in the head endure in the winter, but disappear in the summer time. Spectroscopic examination of the blood—it need not be positive—will confirm the finding. Similar conditions will be encountered in laundries where gas is used.

Reverting to the internal organs again, headaches will be prominent in acute and chronic dyspepsia or in gastritis. In constant or only sporadic dilatation of the stomach the pains are by preference localized in the forehead; by icterus (no matter of what origin) they consist more of pressure in the head. It is selfevident that in dyspepsia and gastritis they possess no differential diagnostic significance so far as neurosis of the stomach is concerned, because headache and pains in the lumbo-sacral region are common companions in all kinds of organic neurosis.

Chronic cholelithiasis (gallstone in the gallbladder) deserves special mention here. It appears often in the frame of a chronic dyspepsia.

There is a feeling of pressure in the stomach, generally one-half to one hour after meals, especially after a heavy banquet, of surfeit, followed by belching, ructus, oppression and headache, dizziness and the sensation of cold pervading the body, and chronic obstipation. (Particulars will be found in my book "Abdominal Pain," Rebman Company, New York.) The same may be the case in relapsing *cholecystitis.*

Intestinal parasites (tenia, trichocephalus, ascarides, oxyuris) also cause chronic dyspeptic troubles (loss of appetite, bulemia, hungerpains, vomiting, diarrhea) with accompanying pseudo-neurasthenic manifestations, principally headaches, also vertigo, palpitation and emaciation. Repeated tests of the feces for the eggs of the parasites will clear the situation.

Headaches play a prominent rôle in the intermittent *flow of gastric juice* (intermittent hyperacidity of the stomach) called by *Rossbach* "nervous gastroxynsis." Violent headaches, severe gastralgias, copious vomiting of hyperacid stomach contents are the principal symptoms. Some of these cases, it seems to me, are only a migraine with prominent gastric manifestations, whilst others rather possess the dignity of gastric crises (for instance in tabes) pointing ultimately to some functional or anatomical disorder.

In affections of the *respiratory tract* headaches

are not of frequent occurrence. If present they are very likely due to some vascular stasis conditioned by repeated fits of coughing, defective pulmonary ventilation or by insufficiency in the right heart.

If headaches make a sudden appearance in bronchiectasy, or in chronic interstitial pneumonia, or perhaps also in cladothrix, they will remind us of possible complications with an abscess in the brain, chiefly in the cerebellum.

Angina pectoris also brings about headaches, chiefly in the left hemisphere. The fact that they nearly always emanate from the retrosternal pain center spreading thence over nape and neck into the occiput will render the diagnosis easy. Unilateral headaches will also be observed in vasomotoric angina pectoris. Cf. chapter on "Pains in the Heart."

Headaches coming in successive attacks should direct our thought to *cardiac dissociation.* But if we are confronted by a functional disturbance of the heart which may also lead to similar cerebral symptoms such as heartblock (see "Disturbances of the Heart Rhythm"), then headaches accompanied by dizziness, befogged consciousness and nausea may constitute the sole sign of a cardiac affection. They would then be due to a defect in the blood supply of the brain, i.e., a periodical cerebral anemia. It is acute ischemia of the brain that produces the pain.

Headache, particularly in the occipital region, sometimes only in the form of a feeling of pressure in the back of the head, often coupled with dizziness, tinnitus and fainting fits, may be the initial sign of incipient chronic *insufficiency of the heart.* (Cf. under that heading.)

Headaches during the period of pubescence, *cephalea adolescentium,* chiefly in the male, are principally located in the frontal region, milder in the morning but more acute in the evening, very bothersome and interfering with even light mental work. They may originate from adenoids. Otherwise we must consider age, abnormally quick physical growth, disturbances in the heart (cardiac debility, palpitation, small caliber of the arteries) and vascular innervation, dizziness, epistaxis, early fatigue in bodily exercise, orthotic albuminuria, in some cases asthenopia, in others insufficient arterial circulation in the brain, retarded cerebral development. All these conditions may be the active cause of "pseudoneurasthenic" cephalic pains. They are called *school headaches* and may persist during the whole scholastic life. This "school anemia," as it is also called, is, no doubt, due to an abnormal rigidity of the arteries originating from an increased vascular muscle tonus. Palpation of the radial arteries will prove this. These pains are also accompanied by other vasomotoric disturbances, among them cold feet and hands,

palpitation of the heart, cardiac pains and fainting spells, even anginoid troubles. In overexertion of the mental faculties a peculiar pain will be sometimes observed. The cause for this may be found in the developing process of the *cranial sutures* and may assume the form of a high grade pressure sensibility (*sutural neuralgia*).

Headaches in childhood may be caused by any of the following conditions, viz., *defective hygienic conditions,* badly ventilated dormitories or bedrooms or classrooms, sitting too near the heating apparatus, too early rising in the morning, insufficient sleep, underfeeding at the breakfast table or going away with an empty stomach, and last but not least to overstrain of the eyes in reading. These conditions may have the same effect even on grown up people.

Persons who work by artificial light especially when the *gas jet or the electric bulb is too near the crown of the head* very often complain of headaches. These pains belong to the same category as *sunstroke* or *heatstroke* and are frequently accompanied by the same symptoms, dizziness, tinnitus aurium, vomiting, trembling of the hands, fibrillary muscular contractions or twitchings, even convulsions, subsequent retarded pulse, unconsciousness, stertorous breathing, delirium. Solar dermatitis is not excluded.

Violent hammering in the cranium and a feeling of pressure in the head are concomitant

symptoms of heatstroke together with dryness in the mouth, physical debility, mental fatigue, mental numbness, indifference to surroundings, rise in body temperature, unsteady gait, bluish tint in a puffed face and hot, dripping skin. Sudden coma and utter collapse, spasmodic muscular movements, anuria, arrest of perspiration, feint, thready, finally disappearing pulse, suppressed breathing, vomiting, diarrhea and general convulsions. Persons who recover from an attack of sunstroke frequently complain for a long time afterwards of headaches and nervous (pseudohysterical) disorders.

Reflex actions from the sexual sphere may also cause headaches. Acute and chronic *infectious diseases* are often accompanied by headaches which are very intensive in some of these affections, e.g., in *pernicious* or *tropical malaria, plague, yellow fever, blackwater fever, recurrent fever, exanthematous fever.* In the last named disease the pain is at times so severe that the patient complains about nothing else than that "terrible pain in head" especially in the top and the frontal region. These pains are at times coupled with vertigo and broadcast into the lumbar and sacral regions, into the calves and articular sections.

Infectious *cerebral* and *spinal* diseases, *erysipelas, smallpox, epidemic parotitis, Wolhynian* fever, abdominal *typhoid, paratyphoid* and

influenza, all are originators of headaches. In typhoid and influenza the pains in the head set in with the incipient stages of the disease—in typhoid they affect also the hearing—and are of a purely toxic infectious character. Recent research ascribes them to hypertension of the cerebrospinal fluid. In consequence they are much ameliorated by lumbar puncture, but they continue with the progressing infection until the clouding of the sensorium renders the patient less susceptible to pain. But if the patient despite this numbness of the sensorium continues to complain of steadily increasing headaches it will be advisable to look for complications in the cranial sphere. Influenzal cephalalgia bears the character of inflammatory pain due to infectious lesions in the nasal cavities, or that of neuralgic pain.

The differential diagnosis between influenza and *hay fever* depends upon the consideration of the pain in the head. Quite true, the hay fever patient also complains about dullness and pain, mainly in the forehead, severely exacerbated by the typical attacks of sneezing. But they are surpassed by the initial burning and itching in the eyes and nose, whilst in influenza the headache together with the rhinitic symptoms will be found among the predominant manifestations.

In *Pappataci* fever, which also may be mis-

taken for the grippe, the streaky injection of the conjunctiva, congestion of the face, leucopenia and the local conditions will facilitate the diagnosis.

Infectious meningitis is another source of headaches. In the abortive forms of *epidemic meningitis* mild headaches are about the only complaint made by the victim. That circumstance renders the diagnosis rather difficult, particularly so when the existence of such an epidemic has not yet been established. Nevertheless, it remains an important sign.

In these cases it is well to watch the other symptoms even though they also be developed in a rudimentary form only. Among them: a slight hammering in the cranium, stiffness in the neck, a touch of pain in the scruff, indication of *Kernig's* signs, percussion in the spinal column, leucocytosis, slight angina, bronchitic or gastric disturbances. Our suspicions will be confirmed by bacterial examination of the nasal and faucial secretions and lumbar puncture (increased lumbar pressure, turbidity in the spinal fluid, intracellular meningococci). But we must not overlook the fact that in these very cases the meningococcus is generally not present at all or at least not in its characteristic form and that the lumbar fluid, though rich in albumin, is quite clear in appearance. The final diagnosis must be made at the bedside.

The recurrence of intensive pains in the head during the time of an epidemic of meningitis points to the development of hydrocephalus, or, let us say, to that of a serous chronic meningitis as a sequel to acute epidemic meningitis.

There are other infectious diseases in which headache is one of the foremost typical manifestations, for instance, *walking typhoid.* Many of these cases are at first diagnosed as influenza or lobular pulmonary catarrh, until a severe ambulant melena throws the patient upon the sick bed or even into a premature grave. We shall do well if in such doubtful cases we keep our eyes open for initial nosebleeds, obvious bradycardia, a possible minor splenic tumor, positive diazo reaction, a rapidly developing leucopenia, a positive *Widal* serum test and the necessary bacteriological examination of the blood. It is my opinion that a stubborn, conspicuous, severe headache devoid of definite signs of infection should always remind us of a possible underlying typhoid condition.

We find these mild cases among the persons who have been vaccinated with *typhoid serum.* But the vaccination itself is often enough connected with fever during the first week, articular pains, lassitude, slight enlargement of the spleen, leucopenia, sometimes a light attack of bronchitis and very often severe pains in the head.

What has been said of typhoid refers in the

same measure to *paratyphoid,* in fact to every kind of *septic infection* in the widest sense of the term. Every disease kindred to typhoid is accompanied by headache. Mild forms of chronic sepsis so frequently met with in chronic infections of the tonsils or other organs of the oral cavities belong here. Likewise *Malta fever* and *miliary tuberculosis.* In the latter initial headaches are frequently present even though the meninges do not participate in any way in the tuberculous process. *Epizootic* and *malignant pustule* must be mentioned in this place. In epizootic the headache is generally localized in the lateral region of the cranium. Anthrax may set in with very severe pains in the head even then when the meninges are either not at all, or at any rate, but slightly implicated (capillary embolism, hemorrhages).

Spotted typhus — as the World War has taught us—may also occur in the "walking" form, the affection being centered in very severe headaches. The sudden rise in the body temperature mainly due to intermittent chills (ague), inflammation of the conjunctiva or of the mucous membranes of the respiratory organs (laryngitis, bronchitis) or of the intestines (diarrhea), intumescence of liver or spleen, meningeal irritations and epidermological conditions will demand an early *Weil-Felix* agglutination test (which need

not be always positive) in order to establish a correct diagnosis.

In *malaria* nearly every attack is accompanied by headache. In fact it is in my opinion even with only a slight rise in the temperature the most salient feature of this infection which in its chronic form is often the originating cause of typical, periodic, cranial neuralgias.

Syphilis in the *secondary* as well as in the *tertiary* stage frequently gives rise to severe, at times also nocturnal, headaches. On account of the accompanying articular pains and fever it is sometimes mistaken for influenza. I call attention here to the differential diagnosis between typhoid and secondary syphilis described in the section on typhoid fever.

In *occult syphilis* the intermittent headaches, frequently setting in towards evening or during the night are an important symptom, no matter whether they are due to some specific disease of the bones, to meningeal infiltrations or to some vascular disorder. Emaciation, pallor, sleeplessness, splenic tumor, nervous disturbances together with the anamnesis and a positive *Wassermann* reaction mould a safe diagnosis.

That long list of "pseudotyphoid" diseases is in every instance associated with headache, sometimes of an almost unbearable nature, e.g., in *acute leucemia.*

Headaches are also the companions of all

those infectious disorders which bear the stamp of *meningism.* They are apparently due to increased pressure of the cerebrospinal fluid. *Lobar pneumonia* (simulating symptomatically meningitis) and *acute articular rheumatism* belong to this category. In the form of *cerebral rheumatism* the latter produces most violent headaches and hyperpyrexia, followed by cerebral excitation and coma. In lumbar puncture performed in this connection no pathological conditions have as yet, so far as I am aware, been found.

In *pulmonary tuberculosis* headache seems to be of minor import, unless there are further complications present, for instance cerebral conditions, tubercle, meningitis, pachymeningitis, sinus phlebitis or caries, i.e., conditions which in themselves lead to headaches (heart, kidney), or violent fits of coughing. Still an initial pulmonary tuberculosis may creep in with headache, dizziness, lassitude, all of which are symptoms of toxic origin and constitute, as it were, a clinical fact, when we take into consideration that frequently we are called upon to differentiate between chlorosis and incipient lobar or glandular tuberculosis. On the other hand these headaches in pulmonary phthisis are often enough caused by a chronic serous meningitis of tuberculo-toxic origin. Lumbar puncture will furnish the proof.

In *pellagra* intensive pain in the head, but variable in its location, is, in close connection with pains in the back and paresthesias in the extremities, the primary symptom, showing how the nervous system is implicated even in the earliest stages of the disease.

We now come to the *hereditary* headaches, the *familial* or *family headaches* handed down by the parents or ancestors who suffered or still suffer from headaches or migraine basic sometimes in chronic alcoholism or lead poisoning, to the offspring. They make their appearance in early childhood or at the time of puberty and are kindred to *migraine* (hemicrania). The characteristic sign of these pains is that they generally occupy only one side of the head; but they may be also bilateral in the sense that they move from one side to the other. In some cases they affect both sides simultaneously, more pronounced on one side or of equal severity in both.

They are of a boring quality, chiefly in the temporal, more rarely in the frontal or occipital region, spreading at times over the whole hemisphere, accompanied also by flittering scotoma or teichopsia or facial disturbances. In some rare cases there is paraesthesia in the extremities. They set in at the early awakening, steadily increase in violence and cause the sufferer to avoid all bodily motion, he feels ill and jaded. Hyperesthesia of the visual and auditory organs

accompanies the pains. Marked irritability, moodiness, dislike of or even unfitness for work, vasomotoric manifestations in the face (pallor, flushing), commonly go with the attack. As a rule, the patient recovers quickly after an attack of vomiting or purging.

Of importance for the diagnosis is the fact that migraine predominates in the female sex and often sets in with the menstrual period. In many cases it disappears spontaneously in the climacteric stage or, at any rate, loses much of its virulence. In pregnancy—mainly after the second month—and during lactation it generally ceases altogether. Migraine is an inherited evil and crops up in early youth. If it makes its appearance only at an advanced age—beyond the forties—it is not a true, but rather a symptomatic migraine.

I have mentioned above that migraine prevails among the gentler sex and often accompanies the menstrual flow. Valuable though this knowledge be, we should not lay too much stress upon this fact. There are other conditions which produce headaches and at the very time of menstruation. I refer to what I have said about headaches in chronic serous meningitis and in abscess of the brain.

There are other affections which show similar symptoms and are often very misleading. The *uremic* or *urotoxic* headache is also onesided,

the same as in migraine; it is also accompanied by vomiting and has the habit of starting in the morning. Of course, when we have unmistakable proof of a kidney disease through urinalysis in cases where this pseudo-migraine makes its appearance in advanced age only, the diagnosis cannot go wrong. But in the abscence of albuminuria mistakes will be made. A correct diagnosis cannot be missed if the following factors are carefully weighed: evidence of permanent polyuria (this may also be present in true migraine. but only in a transient form), pollakiuria, nycturia, examination of the fundus oculi, accentuation of the second aorta tonus, condition of the peripheral arteries, intensity of the blood-pressure, uremic habitus, and all the other symptoms of the so-called "minor uremia," cramps in the calves, *Raynaud's* disease, loss of appetite, itching of the skin, thorough test of the renal functions. This *"uremic"* migraine occurs not only in nephritis but in every kind of renal insufficiency; it is preeminently an early symptom in all urinary intoxications caused by chronic congestion in the urinary tract, e.g., in strictures.

Exogenous toxins may occasion recurrent unilateral headaches strongly resembling migraine, chiefly chronic nicotinism. The diagnosis should offer no difficulties when proper attention is paid to the other symptoms: cardiac disorders (palpitation, arhythmia, anginose troubles), disturb-

ances in the visual organs (impaired visual acuity with central scotoma for red or green, tobacco amblyopia, retrobulbar neuritis), brown discoloration of the teeth and fingers (cigarettes), chronic laryngeal, pharyngeal, bronchial catarrh, disturbances of the nervous system (generalized and neuritic).

Chronic especially *spastic obstipation* of toxic origin may also cause migraine-like pains in the head. It is not often confined to the lateral region, however. But a true migraine may often enough go hand in hand with headache in such cases, and it will be our task to differentiate between the two. This may be the proper place to point again to the fact that a genuine migraine is sometimes eased, if not entirely stopped, by therapeutic means such as purging, or scouring. The differential diagnosis should not meet with obstacles when we keep before us the hereditary and familial moment, the concomitant symptoms (vomiting, hyperesthesia), the matutinal or nocturnal beginning of the attack, and the duty of adopting therapeutic measures for relieving the headache due to chronic obstipation.

Neither must we neglect to remember that such headaches might be traced to *intestinal parasites.* Hence the necessity of proper examination of the stools.

Nervous gastroxynsis (*Rossbach*) in connection with migraine has already been mentioned.

Pains in the *frontal sinus,* in the *nasal cavities* and *toothache* belong here also. Sometimes even a casual examination will determine the differential diagnosis between these aches and migraine in many cases. The absence of visual symptoms, of vomiting and of hereditary idiosyncrasies, the fact that migraine will persist sometimes through weeks or crop up only at intervals as isolated attacks, should make the diagnosis obvious irrespective of local conditions. Similar observations may also be made in affections of the nasal organs especially in women during the menstrual period. But in these cases the seat of the pain is rather in the root of the nose or in the eye.

To misjudge a *neuralgia* of the *trigeminus* or of the occipitalis for a migraine seems to me well nigh impossible. The very fact that in this case the typical pressure points are present, but never vomiting, should guarantee a correct diagnosis.

Unilateral headaches, perhaps with giddiness and nausea and a solitary or repeated attack of vomiting should always point to the existence of an *arteriosclerosis* in the plexus of the *cerebral arteries.* If the headaches are of an intermittent character we may give thought to an intermittent angiospasm of the arteries which have already been impaired by arteriosclerosis. If the pains continue for days or weeks they are a sign of thrombosis in a cerebral artery. Visual and

skin reflexes (exacerbation of the former, absence of the latter), the *Babinski* toe phenomenon, hypersensitiveness of the sensory and motoric faculties (paresthesias, pains), muscular contractions in the extremities contralateral to the lesion, or manifestations of minor defects should be auxiliaries of the diagnosis. In many cases this can be made merely on the ground of the age of the patient, symptoms of arteriosclerosis of the cerebral or other arterial regions. Sufferers from migraine may also become the victims of cerebral arteriosclerosis at a later time in life. The anamnesis, the periodical succession of the manifestations, the point that migraine, with exceptions, of course, generally lapses into desuetude with the advent of the climacterium, i.e., to put it rudely, the season for cerebral arteriosclerosis, aught without default invariably clear up the situation.

An *acute glaucoma* has often enough been mistaken for migraine. Both have in common unilateral recurrent headache, pain in the eye which lies in front of the headache, subjective disturbances in the visual apparatus, and likewise vomiting. If in the bargain the patient suffers from migraine already, the error becomes still more excusable. Only a thorough test of the bulbar tension, in other words a careful ophthalmoscopic examination will remove any doubts that may exist.

Pronounced unilateral headache with vomiting and strongly resembling migraine occurs also in another combination; I refer to the so-called *periodic oculomotor paralysis.* It makes its appearance either before or simultaneously with the attack. In its pre- or coexistence and in the anamnesis (appearance in childhood or early youth) we may find the key to the diagnosis.

Myalgia capitis is at times erroneously taken for migraine because it also is associated with severe headaches although they generally develop on both sides of the head. But the fact that the pains in myalgia are continuous, though at times of a milder character, should be the determining element, especially if the symptoms typical for each of these two affections are carefully considered.

If in migraine a so-called *status hemicranicus* results from cumulative attacks, the differential diagnosis will be surrounded by difficulties. One attack follows the other with intervals of a possible restful night. Sometimes the patient is distracted by continuous pain for six or seven days on a stretch. Visual symptoms and vomiting are not always present. In such cases the diagnosis must rest entirely on the anamnesis.

When the diagnosis of migraine has been firmly established it will yet be necessary to remember, that not all cases of a true migraine are a *protopathic vasomotoric neurosis.* They

may be of a secondary nature, especially when the first attacks arrive only at an advanced age and bear no signs of familial or hereditary origin. A migraine accompanied by visual symptoms, *migraine ophthalmique,* a combination of unilateral headaches and a clouding of the visual field, should always remind us of a localized affection of the brain, chiefly of the *hypophysis* or the *occipital cranial fossa, a cerebral tumor,* also of *acute* or *chronic serous meningitis, aneurysm* of the *cerebral arteries, progressive paralysis, taboparalysis,* syphilis of the brain, *multiple insular sclerosis* or *tuberculous headache.* The ophthalmic form offers difficulties before the typical symptoms have been fully developed.

Useful points to remember are: in migraine vomiting brings relief, often the end of the attack; in tumor, resp. meningitis the patient feels weak, has a jaded look after vomiting. In migraine absolute rest, the exclusion of light and noise have a soothing effect; but not in tumor, at least not to any extent. The tumor patient is indifferent to his surroundings, mentally benumbed, but not so in migraine. But migraine may also in due time develop a cerebral lesion.

In *pyrgocephalus* we frequently find migrainic conditions. The characteristic formation of the head, the bulbar protusion, atrophy of the optic nerve, the Roentgenogram are typical enough for diagnostic purposes.

Epileptic migraine is not always so easily recognized. An epileptic coma may very strongly resemble migraine. Even an atypical attack of epilepsy may have many symptoms in common with a migraine such as headache, paresthesia in the arms and optic disturbances. But we shall find a strong hold in the contemplation of the hereditary conditions, of the convulsive muscular reactions, the rotation of rudimentary attacks and genuine epileptic fits, and also occasional enuresis.

A deuteropathic status hemicranicus may also be observed in another form of neurosis, I mean *cyclothymia.* If depressing moodiness is strongly marked, the diagnosis will be easy. But when, as will happen, the migrainic attacks are associated with a scintilating scotoma and other somatic disturbances, such as periodic sleeplessness, dyspepsia, diarrhea and urticaria, then only a competent psychanalysis will uncover the true state of affairs.

Quincke's edema (hydrops hypostrophos) may likewise lead to a secondary migraine coupled even with external opthalmoplegia (abducens oculomotorius). Such cases are so obvious, however, that they leave no room for doubt.

The same may be said of *erythromelalgia.*

Uratic migraine has been mentioned by some French authors in this connection, not only in the sense that migraine may be basic in gout,

but also that an acute attack of gout might be substituted by an attack of migraine. I have never been convinced of this. Yet it strikes me that migraine, not unlike neurasthenia, finds in gouty conditions rather a fertile ground. Quite recently the opinion of our French colleagues has been maintained by several specialists who are well-recognized authorities on this subject.

Polycythemia rubra may also give rise to migraine-like headaches.

The same may be said of *essential arterial hypertension.*

Hysteria may also simulate migraine. Even typical hysterical stigmata do not definitely exclude a state of genuine migraine, for both diseases may very well co-exist. The presence of a scintilating scotoma and hereditary taint should furnish the final proof.

From what I have placed before my readers it will be clear that a definite localization of headache cannot be reduced to a definite genesis. But the following auxiliary deductions may be garnered from the perusal of the foregoing pages: pain in the occipital region points to induration headache as the originating cause or to occipital migraine or uremic cephalea or an affection of the heart or the fauces. A circumscribed headache in the frontal region may arise from an optic disorder, from the nose or from the frontal sinus. Strictly defined pain in the

vertex directs our attention to hysterical etiology. Benumbing pain in the vertex leads to ethmoiditis and pain in the temporal region around the ear to otological conditions or affections of the teeth as the causal factors. For the local diagnosis of a cerebral tumor headache is one of the strongest indications, especially when it is accompanied by the sensation of humming in the cranium and tympanitic percussion sound in this region with *bruit de pot fêlé.*

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