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BIRTH INJURIES  
OF THE CHILD





# BIRTH INJURIES OF THE CHILD

BY

HUGO EHRENFEST, M.D., F.A.C.S.

PROFESSOR OF OBSTETRICS AND GYNECOLOGY, ST. LOUIS UNIVERSITY SCHOOL  
OF MEDICINE; OBSTETRICIAN AND GYNECOLOGIST, JEWISH HOSPITAL,  
MEDICAL DIRECTOR OF ST. LOUIS OBSTETRIC DISPENSARY;  
CONSULTING OBSTETRICIAN, ST. LOUIS MATERNITY  
HOSPITAL; FELLOW OF THE AMERICAN GYNE-  
COLOGICAL SOCIETY; ETC.

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## PREFACE

A serious charge against the present status of obstetrical practice was made in 1917 by Grace Meigs in her report on Maternal Mortality in the United States. "Every year at least 15,000 women die in the United States from conditions, almost entirely preventable, caused by childbirth." Ever since, this statement has been reiterated practically by every writer and speaker who felt that he could offer a valuable suggestion towards the betterment of this truly appalling situation. The stereotyped and almost exclusive use of this one argument in all the varied efforts to supply the women of this country with "better obstetrics" would seem to imply that conditions are satisfactory as far as the newborn infant is concerned.

Dr. Meigs has irrefutably demonstrated that up to 1913, childbirth has remained as hazardous to the mothers as it was in 1900. How about the danger of birth to the infant? How great is it? Is it increasing or decreasing? Where rests the responsibility for this risk? Can anything be done to reduce it? To answer these and many other questions pertaining to the serious problem of Birth Injuries of the Child is the purpose of the volume here presented.

Wider interest in the causation and prevention of parturitional injuries of the infant is of relatively recent date, but it is growing rapidly. This must have become evident to any one conversant with modern obstetrical literature. To-day we are facing the surprising fact that "in at least 40 per cent of all autopsies, properly performed on all stillborn infants and those dying within the first few days after birth, intracranial traumatic lesions of some sort are discovered," while in official and unofficial tabulations of the causes of death in earliest infancy or of stillbirth "injury in birth" is assigned a most inconspicuous place. To quote but a few examples selected at random: Very exhaustive field studies have been made in various communities by the Children's Bureau concerning Infant Mortality. What do they show? In Manchester, N. H. (Infant Mortality Series No. 6, 1917), out of 34 live born infants who died under two weeks old, the death of but one was reported as due to

a birth injury; in New Bedford, Mass. (Inf. Mort. Ser. No. 10, 1920), of 84 infants dying under the age of two weeks, of whom 41 had died in less than 24 hours, the cause of death was given as injury in birth in 10 cases, and in Akron, Ohio (Inf. Mort. Ser. No. 11, 1920), in 6 out of 73 live born babies dying under two weeks of age.

These statistics, though they seemingly manifest, within three years, an increasing appreciation of birth injury as the cause of death, are evidently incorrect. The majority of the actual cases of fatal traumatism still are included in the figures assigned to prematurity, congenital debility, and most of all to asphyxia. The inaccuracies of these and similar statistics are due to definite causes.

Not only many of the intracranial, but also most of the fatal injuries of the vertebral column, spinal cord or abdominal viscera, can be discovered only at autopsy—an autopsy, as must be emphasized, in which the skull is opened in a specific manner and the routine examination includes the vertebral column, the spinal cord, and such abdominal organs as, e.g., the suprarenal bodies.

Without an autopsy the cause of death so often is incorrectly ascribed to asphyxia because the majority of seriously traumatized infants exhibit a clinical picture which closely resembles that generally considered as typical for deep asphyxiation.

The attending physician excusably hesitates to pronounce the infant's death the result of a parturitional injury, because, unfortunately and unjustly, it has remained the prevalent conception, both among the profession and laity, that fatal injuries are the result of, and thus signify, a lack of proper skill on the part of the accoucheur.

Practically without any exceptions every known type of parturitional traumatization has also been observed subsequent to spontaneous labors. The term "spontaneous labor" in this connection does not necessarily mean, in its customary application, a non-artificial and noninstrumental labor, but as well, in the strictest sense of the word, a labor in which the child was expelled without any extraneous aid of any sort. This fact deserves wider appreciation, not only by physicians and the laity, but also by the legal profession. Only recently the Supreme Court for New York County (184 New York Supplement, 337) has determined that a child injured for life, through negligence on the part of the attendant at its birth, can recover damages for injuries sustained.

No physician should feel any reluctance in pronouncing and reporting the infant's death as the result of birth injury if, in his

opinion, this is the case. If the obstetrician could furthermore be induced to suspect and search for a possible injury whenever the newborn child seems to act abnormally, prompt and proper therapeutic interference certainly would save many children now doomed either to immediate death or possibly to a life sadly marred by a preventable physical or mental defect.

This newer knowledge, that the child can be more or less seriously injured in the course of a normal and spontaneous delivery, however, does not relieve the physician from all responsibility for such injuries. In by far the larger number of instances severe traumatism is observed after labors terminated artificially or by means of instruments. In this large group of cases lack of skill plays an important rôle. It may be lack of diagnostic ability, a lack of judgment or mere awkwardness of technic, not rarely superinduced by bewilderment or entirely unnecessary haste. It is this fact which naturally suggests still another question. Is the pronounced operative trend of modern obstetrics increasing, or likely to increase, the incidence of birth injuries of the child? Statistical figures seemingly indicate a steady increase. In part this is certainly due to the evergrowing interest in, and the more systematic search for, such injuries. However, careful consideration of the various mechanical factors directly responsible for the traumatization of the child would seem to lead to the inevitable deduction that a higher incidence of injuries must be looked for, if the teachings of some of our ultramodern obstetricians should prevail. No amount of personal technical skill, unavoidably acquired at the cost of many fetal lives, in my belief, could neutralize the augmented risks to the child of a routine version followed by immediate extraction, of forceps extractions seriously recommended even on the high head for the sole purpose of shortening the suffering of the parturient woman, or made necessary by the elimination of important accessory expulsive force in twilight sleep. No personal effort of the accoucheur could obviate the dangers of a sudden and excessive compression of the fetal head quickly forced through an unyielding birth channel by a large dose of pituitrin.

Most clearly it has become the duty of those who advocate the artificial termination of labor under general anesthesia in order to overcome the unquestionably slower and more painful process of spontaneous delivery, to prove that such methods do not imply a greater immediate and later risk to the life and health of the infant.

Mere mortality figures will not suffice to settle this complex problem. Slighter intracranial lesions, fractures practically of every bone of the skeleton from skull to femur, hemorrhages whether in the eye or in abdominal viscera, are not necessarily fatal, but they are fairly common and, as a rule, are overlooked if not revealed by later sequelae. To be sure, in the overwhelming majority of instances these minor injuries probably inflict no permanent harm on the infants. There is, however, much evidence rapidly accumulating which tends to show that many of the conditions, still rather loosely classified as congenital, as a matter of fact are but the later manifestations of traumatic lesions sustained at birth, lesions which remained unrecognized in the newborn, possibly because their symptoms were insignificant, and more probably because the obstetrician failed to examine the newborn with the necessary care, or left, as is the custom, the observation of the behavior of the infant during the first few days of life entirely to a more or less competent nurse.

Modern conceptions of the causation of birth injuries of the child place on the obstetrician, not only the task of preventing them so far as this is possible, but also the responsibility for recognition of their presence at the earliest possible moment. He must cease to regard the newborn as the "unavoidable by-product of his essential function of separating the mother from the fetus" as facetiously but with a justification recently remarked by a leading pediatrician.

In theory it would be the ideal to have the expert pediatrician assume responsibility for the child immediately after birth. In practice this is not feasible outside a properly staffed general hospital. The prompt recognition of a birth injury for the present remains the duty of the obstetrician. To help him in this task, the author has attempted to present in this volume all available information concerning the pathology, etiology, and symptomatology of all known parturitional injuries of the child.

The discussion of the etiology of each type of traumatism offers the opportunity for pointing out certain details in the technic of recognized operative procedures and nonoperative manipulations which have been found to imply special dangers to the fetus. In the consideration of the symptomatology and diagnosis an effort is made to set forth clearly the points of differentiation between unpreventable injuries, those caused by inexpert or actually reprehensible procedures on the part of the attending physician or midwife, unin-



tentional traumatization of the child by the mother delivering herself without aid, and finally injuries inflicted on the born child with criminal intent.

The discussion of treatment is deliberately limited to methods which are simple or might prove immediately life saving. It is my sincere conviction that further treatment is preferably left to the respective expert—the pediatrician, neurologist, surgeon, or orthopedist.

No obstetrician could ever be expected to accumulate sufficient personal experience to write authoritatively on every form of injury which the infant is likely to sustain at birth. Of necessity, therefore, much of the material for this monograph had to be compiled from a great variety of sources, including practically every special branch of medicine.

Having studied in the original, as far as was possible, the contributions selected for citation in the volume, I feel certain that I am quoting correctly the observations, investigations, and theories of the large number of men who have seriously thought about the manifold aspects of the problem of birth injuries. The list of references appended to each chapter clearly indicates the source of my own information, but it also is expected to prove useful to those who are seeking enlightenment on minor details for further investigations. I am taking the liberty of directing here and there attention to mooted points and discrepancies of opinions which, in my belief, could be settled by studies and observations along certain lines.

To my best knowledge the Birth Injuries of the Child never before have been dealt with in so exhaustive a manner as in this volume. There was no precedent by which to determine what constitutes a birth injury. I have confined myself to a consideration of the traumatic injuries sustained during labor and delivery and in the common and necessary manipulations of the newborn. Unquestionably the infant might also be chemically injured if during a twilight sleep a total of five-sixths grains of morphin and eleven ampules of hyoscin hydrobromid (the maximum of which I have personal knowledge) are administered to enable the attending obstetrician to keep his promise that the patient will not know anything about her labor. There was a question in my mind whether this monograph should not dwell upon injuries of the umbilical

cord. As will be seen I decided to consider only traumatization of the umbilical vessels within the abdomen of the infant.

The discriminating reader presumably will discover other omissions, almost unavoidable in a first effort to deal with so complex a problem. Nevertheless, I hope that, not only every physician attending women in labor, but also those limiting their work to a special field of medical science, including the branch of forensic medicine, will find in this volume something that will prove of interest and practical value.

HUGO EHRENFEST

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## CHAPTER I

### CAPUT SUCCEDANEUM

Pathology—Etiology—Diagnosis—Prognosis—Treatment.

While the presenting fetal part, after rupture of the membranes, is gradually pressed down through the lower portion of the birth canal, a localized, soft, boggy swelling forms in that portion of the skin surface which is exposed within the area of the dilated cervix. On account of the decided prevalence of cephalic presentations, this edematous swelling, as a rule, is located somewhere on the fetal head, and it is for this reason that it is called caput succedaneum. Obviously in other than cephalic presentations the identical swelling appears on one or both feet, on the buttocks, then usually extending over the external genitalia, or on a shoulder.

**Pathology.**—The caput formation on microscopic study is found to be the result of an infiltration of the subcutaneous connective tissue and of the tissues beneath the galea with a sero-sanguineous transudate, without exceptions, accompanied by ecchymoses in the skin, and smaller and larger hematomas in all layers of the scalp. Skin and underlying tissues are swollen to twice and three times their normal thickness. Often there are flat hemorrhagic areas underneath the pericranium, and within the extent of the caput the underlying bone and meninges may exhibit a marked dilatation of the blood vessels. This description, first furnished by Werth, has been later confirmed in every detail by the investigations of Loennberg, and quite recently by Schwartz who found this hyperemia with small hemorrhages often also extending into the cerebral substance.

**Etiology.**—A detailed discussion of the mechanical factors presumably accounting for the formation of the caput is desirable in view of the fact that these same factors also are supposed to play

an important rôle in the causation of intracranial lesions of the newborn.

It is now rather generally assumed that the caput is the expression of the difference between the atmospheric and the intra-uterine pressure greatly increased during each uterine contraction. Only within the area of the dilated cervix the neutralizing counterpressure exerted by the contracting uterine wall is wanting. To a certain extent a similar situation prevails if unruptured membranes during a uterine contraction bag deeply down into the vagina. Therefore, occasionally, though rarely, the development of a caput can be noticed before the escape of the amniotic fluid.

Some authors explain the formation of the caput in another manner. They believe that the presenting part during a uterine contraction is so firmly pressed against the comparatively rigid ring of the partially dilated cervix that the deflux of the venous blood is interfered with. The resulting passive congestion causes the local edema. The main arguments in support of this theory are the usual absence of a caput before the rupture of the membranes, and especially the evident fact that large swellings, as a rule, are found in the cases of marked rigidity of the cervix. Obviously this latter theory of caput formation implies the necessity of active circulation of blood through the affected skin area at the time the caput is formed. It was held, and, indeed, is still claimed by some medico-legal writers, that the presence of a caput succedaneum proves beyond any doubt that the fetus was alive during the second stage of labor. The error of such a belief seems now well established by undisputed observations of a typical caput on fetu born in a macerated condition, and also by experiments first made by Lesser and later confirmed by Holzapfel. By applying a breast pump to the skin of fresh stillborn infants they succeeded in producing circumscribed edematous swellings which in every respect seemed identical with the typical caput. These experiments furnish strong support for the entirely plausible theory that the edema develops as a result of that difference between internal and external pressure which exists during a uterine contraction within the area of the dilated cervix.

The size of the caput succedaneum in general is proportional both to the strength of the uterine contractions and the length of time elapsed between rupture of the membranes and expulsion of the child. Since abnormal rigidity of the cervix and pelvic contrac-



tion obviously lengthen the duration of expulsion, they do play an important, though only indirect, rôle in the etiology of the large caput.

**Diagnosis.**—This soft, doughy swelling, at its edge gradually rising from the level of the surrounding skin surface, is present at the moment of birth. Its location is definitely dependent upon presentation and position of the child during labor, and, therefore, the caput immediately after delivery can be utilized to determine the mechanism of labor. This is a point of considerable importance in certain medicolegal cases. In all head presentations the caput lies the farther away from the occiput and the nearer the face, the less completely the fetal head was flexed. In all head presentations the caput is situated on the right when the back of the fetus was on the mother's left side during descent, and vice versa. In pelvic end presentations the caput appears on the right when the fetal back was on the mother's right.

The swelling is the largest at birth and begins to reduce immediately, as a rule, disappearing within a few days.

No localized edema in the skin of the newborn which in its location does not exactly correspond to the known mechanism of labor can be diagnosed as caput.

**Prognosis.**—The simple, uncomplicated caput succedaneum, even if large, in general is of no clinical significance. When in a face presentation it occurs over the infant's entire mouth, the edema of lips and tongue may prevent suckling for a few days.

There are only five or six cases on record in which excessively large swellings became necrotic and finally resulted in extensive scar formation (Nebesky). In one other instance a child succumbed to the protracted suppuration of a large caput.

Medical, and especially medicolegal, literature mentions several instances of more or less serious injuries produced in the attempt to open the amniotic sac when a large, soft caput was mistaken for the protruding unruptured membranes.

**Treatment.**—Though in itself practically devoid of any danger to the infant, the very large caput has so evident an obstetric significance in the protection of the fetal life that it seems justifiable to speak of its prophylactic treatment.

An unusually large caput, as already stated, is chiefly the result of a protracted labor in the presence of, or as one could more appropriately say, in spite of, strong uterine contractions. Therefore,

the formation of a large caput in the course of labor in most instances is indicative of some mechanical obstacle, as a rule, offered by the cervix or the pelvis. Rapid increase of the swelling during the second stage calls for a thorough study of conditions to prevent the passing of the proper time for intervention.

A quickly growing caput often deceives the inexperienced, who erroneously interprets the gradual descent of its lower pole as a sure proof that labor is progressing satisfactorily.

It has been claimed by some writers that a marked decrease in the consistency of the caput in the course of labor strongly suggests the death of the fetus.

The not uncommon abrasions and small ecchymoses of the skin over the caput call for protective measures against possible infection. Sterile vaseline covered with a layer of sterile gauze will prove sufficient for this purpose, and should never be omitted in cases in which the swelling involves mouth, tongue, or the eyes of the newborn. However, in the overwhelming majority of cases the caput succedaneum demands no particular therapeutic attention.

#### LITERATURE

Ehrendorfer. *Arch. f. Gynäk.*, Berl., 1906, 80:32.

Holzapfel. *Ztschr. f. Geburtsh. u. Gynäk.*, Stuttg., 1909, 63:512.

Loennberg. *Monatschr. f. Geburtsh. u. Gynäk.*, Berl., 1903, 17:175.

Nebesky. *Ibidem*, 1913, 38:655.

Schwartz. *Ztschr. f. Kinderh.*, Berl., 1921, 29:102.

## CHAPTER II

### CEPHALHEMATOMA

Pathology—Etiology—Symptomatology—Diagnosis—Prognosis—Treatment.

**Pathology.**—In post mortem examinations of newborn infants, even after a spontaneous and normal labor, small hemorrhages are commonly found in the connective tissue underneath the galea and especially between the flat skull bones and their periost, as a rule, in the proximity of a suture. Only if the extravasation of blood is large and a definite hematoma is formed by a wider detachment of the periost do we speak of a cephalhematoma.

The covering skin usually is intact, though it may exhibit small punctate hemorrhages if, as often is the case, the cephalhematoma is located underneath a caput succedaneum. The periost is lifted up by dark fluid blood the quantity of which has been estimated to be between 2 and 4 cubic centimeters, rarely up to 100 or even 200 cubic centimeters. In a hematoma several days old the inner surface of the sac, including the denuded bone, is found covered with a yellow-reddish gelatinous substance, an exudate in which osteoblasts begin to form bone platelets (Meyer-Ruegg). This is particularly noticeable in the corners between bone and detached periost, and this accounts for the hard ring all around the hematoma which at the end of the first week has reached a height of from 2 to 4 millimeters. Careful search will often reveal small fissures or a distinct fracture in the underlying skull bone (Meyer-Ruegg), a point of great clinical importance.

In 127 cases of extracranial hematoma collected by Henning (quoted by Birnbaum), its situation was the following: right parietal, 57; left parietal, 37; over both parietals, 21; occipital, 7; frontal, 3; and temporal, 2. We thus find among the 127 cases analyzed by Henning 21 with more than one hematoma. Occasionally also a triple cephalhematoma has been described, e.g., in a case of Audebert.

A most characteristic feature in the anatomy of the supra-osteal

hematoma is its strict limitation within the expanse of the one flat skull bone over which it has formed. The explanation of this phenomenon has been clearly set forth by Merkel. The skull bones develop within the various strata forming the primordial cranium. In the newborn, therefore, the soft tissues of the sutures and fontanelles represent the direct continuation of both the pericranium on the outside and the dura mater on the inside. These two layers are too firmly attached to each other to permit their separation even under the pressure of the blood contained in a cephalhematoma. There is apparently only one case known in literature in which the hematoma extended across the sagittal suture from one parietal bone to the other (observation of Olshausen mentioned by Stumpf). In this instance, however, the sagittal suture at birth was completely ossified. Meyer-Ruegg calls attention to the fact that in some rare instances such extracranial traumatic hemorrhages may be situated not underneath the periosteum, as in the true cephalhematoma, but under the aponeurosis and then obviously may spread across the suture line.

**Etiology.**—In the second stage of labor, when the head has entered the pelvic canal, with each uterine contraction and subsequent relaxation, the skin of the skull is pulled forth and back. This recurrent displacement of the soft tissues against the underlying harder bone results in stretching and eventually in tearing of some of the blood vessels underneath the periosteum. Lorand thinks that this explanation of the origin of the cephalhematoma finds strong support in the fact that it is not often found in cases of pronounced pelvic malformation. The molded head is too firmly held in the contracted pelvis to allow any recession in the interval between contractions. A tangential force is required to effect this loosening of the skin, and it is supplied by abnormal friction. Therefore, not only slight pelvic anomalies and the greater rigidity of the soft parts (of cervix, vagina, and the vulvar ring) in the primipara, but also unusual hair growth on the fetal scalp seem to play an important rôle in the causation of the extracranial hematoma of the newborn. Bioche figured that in 79 per cent of the cases the mothers were primiparous. Pinard found that in 25 per cent of the cases the child's hair was from 3 to 5 centimeters long.

A cephalhematoma is rarely seen after a breech labor, and after a forceps extraction never just at the point where the forceps blades were pressing against the skin.

The fact that a cephalhematoma forms only about once in every 200 deliveries (according to Hofmohl, 0.6 per cent) suggests that the blood vessels underneath the periosteum are ruptured only under certain conditions. Prematurity of the child probably is of some etiologic significance in view of the acknowledged greater fragility of the blood vessels in the prematurely born infant. Meyer-Ruegg points to the frequency of the association of a cephalhematoma with hemorrhages in other organs. We shall have occasion, in a subsequent chapter, to dwell in detail on a possible relation of intracranial hemorrhages to cephalhematoma and other extravasations of blood on the basis of a hemorrhagic diathesis, so far not mentioned in literature as of possible etiologic significance for the formation of the cephalhematoma.

Retarded ossification is supposed to favor the formation of a cephalhematoma by allowing an abnormal degree of overriding in the sutures during molding of the head. Féré (quoted by Meyer-Ruegg) claimed that a cephalhematoma may be produced by the rupture, in labor, of vessels that run across a gap in a skull bone, the result of deficient ossification.

Mention must be made, in this connection of another form of subperiosteal hematoma which is not a cephalhematoma within the usual application of the term. It is formed by the blood escaping from the vessels of a cranial bone injured in labor. Obviously such a hematoma is of great diagnostic importance for the recognition of traumatic birth lesions of the cranium and in its relation to intracranial hemorrhages.

**Symptomatology.**—As a rule not until the second or third day of life is the cephalhematoma noticed, as a flat, soft tumor, most commonly lying over one of the parietal bones near its posterior and upper angle, only rarely over occipital, frontal or temporal bone. Within the next few days, as the result of continued extravasation of blood, the swelling increases. When at the end of the first week all around its well-marked contour a hard ring begins to form, the center of the tumor feels comparatively much softer, more like a dense cyst. From then on the swelling gradually recedes. Smaller hematomas are completely resorbed within three or four weeks; larger ones may require from six to eight weeks. The bony ridge at times becomes permanent and occasionally the entire hematoma transforms into a flat, rounded exostosis.

In exceptional cases a cephalhematoma has been noticed imme-

diately at birth and in one instance, reported by Winckel, its formation was ascertained in the course of labor.

The cephalhematoma itself does not affect the newborn's general condition. It apparently does not cause pain and proves insensitive to pressure. The blood loss into the hematoma is rarely great enough to cause anemia. This is more likely to occur in a case of multiple hematomas.

**Diagnosis.**—The true cephalhematoma is readily diagnosed. DeLee offers the following comprehensive outline of the symptoms which differentiate the caput succedaneum from the extracranial hematoma:

CAPUT SUCCEDANEUM	CEPHALHEMATOMA
Is present at birth.	Usually appears only on second day or later.
Is soft and boggy and pits on pressure.	Is soft, elastic, and does not pit.
Is diffuse.	Is sharply circumscribed with a distinct, sometimes hard, edge.
Runs over sutures.	Is limited by sutures to individual bone.
Covered by a dark red, mottled, sometimes ecchymotic skin.	Skin normal, except when cephalhematoma lies underneath a caput succedaneum.
Is movable on skull.	Fixed to site where it originated.
Is largest at birth and gradually gets smaller, disappearing very quickly.	Rarely present at birth, grows larger for a time; disappears only after weeks or months.

Of by far greater importance is the differential diagnosis between a cephalhematoma and the protrusion of cranial contents through a congenital or traumatic opening in a suture or a flat skull bone. Only the rarer occipital cephalhematoma alters the shape of the head so as to simulate in appearance a meningocele or encephalocele. More careful examination will readily clear up any doubts. The hernia cerebri, as a rule, shows a marked impulse when the child cries. It is compressible

and its emptying by pressure is prone to lead to characteristic meningeal symptoms. All these phenomena are missing in cephalhematoma.

Greater difficulty will be experienced in the most essential differentiation of the typical cephalhematoma from a subperiosteal hematoma in which the source of the effused blood is represented by injured blood vessels lying within or underneath cranial bones. In these cases the hematoma is but a symptom of the decidedly more important primary injury of the underlying skull bone. A hematoma of this sort may for some time successfully hide even a deep bone indentation or a guttered fracture. We shall leave a further discussion of this fact for the next chapter, but must emphasize in this connection the necessity of a most careful examination in every case in which an extracranial hematoma is seen in association with cerebral symptoms, especially if the latter become manifest or are aggravated by pressure on the external swelling. These symptoms, together with pulsation synchronously with the child's heart, and an increase of the hematoma on crying prove conclusively the free communication of an external hematoma with an intracranial hemorrhage.

**Prognosis.**—As already pointed out, the true cephalhematoma is not likely to affect the newborn. Occasionally the hematoma becomes infected, clearly indicated by a rise of temperature. There are a few cases on record in which such a suppuration led to bone necrosis and finally to a fatal issue as the result of a meningitis or septicemia.

**Treatment.**—In view of their clinical insignificance, cephalhematomas are best let alone. Some pressure may be exerted by means of a soft bandage not too tightly applied, for the purpose both of retarding further effusion of blood and of hastening its resorption. Aspiration of the blood by means of a puncture is a procedure without distinct advantage but involving the risk of infection. A bandage must be applied snugly whenever a hematoma is emptied to prevent its refilling. Cases of serious hemorrhage have been reported subsequent to puncture, apparently caused by hemophilia or a hemorrhagic tendency.

Incision followed by proper surgical treatment is required in every case in which sensitiveness, a rise in temperature, or local signs indicate the infection of a cephalhematoma.

## LITERATURE

- Audebert. Abst. in J. Obst. & Gynec. Brit. Emp., Lond., 1911, 19:352.
- Birnbaum. Malformations and Congenital Diseases of the Foetus.  
P. Blakiston's Son & Co., 1912.
- Bondy. Monatschr. f. Geburtsh. u. Gynäk., Berl., 1912, 35:499.
- Merkel. Handb. d. topog. Anat., 1:30.
- Meyer-Ruegg. Winckel's Handb. d. Geburtsh., 3:3, p. 152.
- Reuss. Krankheiten des Neugeborenen, Springer, Berlin, 1914.
- Stumpf. Winckel's Handb. d. Geburtsh., 3:3, p. 489.



## CHAPTER III

### INJURIES OF THE SCALP

Forceps injuries—Injuries caused by unskilled examinations or manipulations—Congenital skin defects.

After protracted labors, an oblong, reddish mark may be seen on the head of the newborn, most often situated over one of the parietal bones. It is produced by the pressure most frequently of the promontory, less often of an abnormal ridge on the inner surface of the symphyseal junction, or of an exostosis.

In cases of forceps extractions, required by a contraction of the pelvis, these typical marks are likely to be found associated with narrower and deeper impressions caused by the tip or edge of the forceps blade. If the forceps have to be applied in an oblique direction or along the anterior-posterior diameters, or if the forceps have to be used to effect a rotation of the head, either the tip or the lower long edge of one of the blades may actually cut through the skin and, as will be shown later, injure the underlying bone. With a slipping forceps the skin may be lacerated. Only an unskilled operator will fail to recognize that the forceps are gliding. In this manner the several observations, recorded in literature, are explained in which one ear of the newborn was found partially, and at least in one case completely, torn off. Stumpf mentions a severe hemorrhage from the supraorbital artery torn by a slipping forceps blade.

Outside of these rarer instances of serious lacerations, birth injuries of the skin will prove harmful to the newborn only when the pressure was severe or long enough to result in necrosis or gangrene, which terminate in fatal sepsis or a disfiguring scar. Wyder observed one case in which ultimately the entire scalp from neck over the occiput up to the large fontanel had become gangrenous.

There is still another group of cases in which the blame for the injury rests clearly with the attending obstetrician. Lacerations

and contusions of the scalp have been seen after careless and rough internal examinations. Stumpf mentions 2 cases in which deep incisions, in 1 of them 6 centimeters long, had been made across the large caput succedaneum in the attempt to open the presumably unruptured membranes. Both ended fatally. Skin perforations with safety pins and sharp-pointed instruments are on record. Some excuse may be found for serious damage done in the identical manner in one case in which a meningocele, and in several other cases in which a hydrocephalus had been mistaken for the protruding bag of waters.

Mention may be made in this connection of the observations recorded by Dziewonski, Jaeger, and others, of spontaneous rupture of a hydrocephalic head during labor, the scalp rent in Dziewonski's case being 35 centimeters long.

These traumatic birth injuries must not be confounded with peculiar skin defects occasionally seen on the head of the newborn, the result of severed amniotic adhesions. They are sharp edged, usually round ulcerlike wounds, varying in size and at times already partially closed by distinct scar formation. Their most characteristic feature is the common association of such defects on the head with others of the same character located on the trunk or extremities, often on the heels. Finkelstein refers to the forensic importance of a clear differentiation of these congenital from the traumatic skin lesions of the newborn, as evidenced by several law suits instituted against obstetricians.

## LITERATURE

- Dziewonski. Abst. in Gynäk. Rundschau, Vienna, 1914, 8:408.  
Finkelstein. Berl. Klinik, 1902, No. 168.  
Jaeger. Kor.-Bl. f. Schweizer Aerzte, 1918.  
Stumpf. Winckel's Handb. d. Geburtsh., 3:3, p. 490.  
Wyder. Ibidem, 3:1, p. 565.

## CHAPTER IV

### INJURIES OF THE CRANIAL BONES

Pathology and etiology of indentations, fissures and fractures—Frequency of injuries of the cranial bones—Symptomatology—Diagnosis—Differential diagnosis from injuries inflicted in criminal intent—Cranial injuries in precipitate labor—Responsibility of obstetrician—Prognosis—Prophylaxis—Treatment.

The elasticity of the flat cranial bones and their mobility against each other within the extent of the sutures afford to them unusual protection against serious traumatic injury, even if a definite disproportion between pelvis and head necessitates an excessive degree of molding. Therefore, the more serious traumatic lesions, as a rule, are seen only after labors terminated by forcible means. Cranial bone injuries usually are divided into indentations, fissures, and actual fractures. In the individual case such exact differentiation may be impossible. Deeper indentations, probably more often than suspected, are associated with fissures or fractures, recognizable only during a subsequent operation or at autopsy. Fissures really are only special types of fractures.

**Pathology and Etiology**—**INDENTATIONS**.—Indentations are in the main of two types. In the one, the impression is round or oval-shaped and shallow, more spoonlike; in the other it is an oblong groove with fairly steep edges, resembling a gutter. Most commonly they are produced by the protruding promontory of a contracted pelvis as the head forcibly is pulled over it during a breech or forceps extraction. For this reason it usually is the posterior parietal bone that is damaged in this manner between its tuber and the coronary suture, less often a frontal bone, and but rarely a temporal bone. Deep indentations of the frontal bone occasionally are the result of the direct pressure of a forceps blade. That the pelvic deformity rather than the operation is primarily responsible for the injury is proved by observations of the identical type of traumatism on every child of the same mother. An ankylosed coccyx or an unusually large sciatic spine may also leave its mark on a passing flat skull bone.

**FISSURES.**—Fissures are linear cracks in parietal or frontal bones running along and parallel to the radii of ossification. There may be but one fissure, often at the bottom of an indentation, or more of them, then spreading starlike. Etiologically they seem in many instances to be closely connected with imperfect ossification. Such defective areas are surrounded by abnormally thin bone, which is easily damaged by sudden or strong pressure. Traumatic fissures, however, must be clearly differentiated from fairly common defects of ossification which are represented in broader clefts, also running from the edge of a flat cranial bone in a radial direction towards its center.

**FRACTURES.**—The characteristic differential feature of a fracture from a mere fissure is that in the case of the fracture the trauma has caused cracks to form which, at least in part, run across the radii of ossification, and thus result in the separation of already united portions of the bone. In the case of a fracture a part of the bone edge, or an entire piece of bone, may become dislocated, and, therefore, is more prone to injure the underlying intracranial structures.

In the great majority of instances deep indentations and depressed fractures are the result of an obviously severe trauma, sustained in a difficult forceps or breech extraction, usually in the case of a contracted pelvis. Gfroerer observed in the Wuerzburg clinic 26 cases of this sort. In all instances the mother had a narrow pelvis. Only one labor had terminated spontaneously. All the other injured infants had been born in a breech presentation, mostly after a version.

Both slight and extensive cranial fractures, however, have been recorded also in spontaneous and entirely normal labors (probably for the first time by Hirt in 1815). This is a fact of great forensic importance. Zinsser described a fracture of both parietal bones seen in a small baby born spontaneously of a woman who had a roomy pelvis. He ascribed the injury to the sudden impact of the small and thin-boned head against the pelvic floor in the moment of the rupture of the membranes. Another case, observed by Hartmann, seems worthy of citation: A normal-sized child was spontaneously expelled by a multipara just while Hartmann was getting ready to apply the forceps. The newborn infant was deeply asphyxiated and could not be resuscitated. It showed a depressed fracture in the middle of the left frontal bone, undoubtedly produced by the promontory and also a pressure mark, 3 centimeters long,

on the occiput. Post mortem examination furthermore revealed a fracture between the condylic and squamous part of the occipital bone. It is evident that if the extraction would have been actually made with the forceps, responsibility for this extensive injury would have been placed on the operation. Noteworthy in this case is the assertion of the mother that all her preceding four children had been born with cranial injuries. One child, six years old, still showed a deep spoon-shaped depression over the left parietal bone. Goodman also described three cases of cranial injuries subsequent to short labors without instrumentation. Frank (quoted by Stumpf) placed a case on record in which five successive babies of the same mother exhibited the same cranial injury. We shall end these quotations with the reference to a case of Veit (mentioned by Reuss) in which he blamed a parietal fracture on the rigid contraction of the cervix following the injudicious administration of a large dose of ergot. Large doses of pituitrin, which at times also cause the cervix to contract, and always tend to increase greatly the expelling force, might play a greater rôle in the causation of traumatic injuries of the cranial bones than so far is suspected or proved.

In that rare type of parturitional fracture which involves the cranial base, most often the condylic portion of the occipital bone becomes detached from the squamous part. The mechanism of this separation presumably is the following: In forceps extraction the squama may be torn from the condyles if the rest of the head does not readily follow the traction exerted over the occiput, and if simultaneously the head is compressed in a fronto-occipital direction whereby the squama is pushed underneath and loosened from the condyles. The mechanism is practically the same, though acting in a reverse direction, in the case of difficult breech extraction (Kuestner).

**Frequency.**—Very little information is available concerning the frequency of parturitional traumatization of the flat skull bones. Stumpf asserts that studies made by various investigators on large series of cases have rather uniformly shown a frequency of approximately one per cent. Kuestner emphasizes that with the improvement of obstetric diagnosis and especially of obstetric technic these injuries undeniably have become less common.

**Symptomatology.**—It is impossible to outline a specific symptomatology of cranial injuries. In most of the cases in which the damage to the bone is slight, noticeable symptoms are entirely miss-

ing. There cannot be any doubt that slight indentations, immediately correcting themselves, mere fissures and the not depressed fractures in many cases for this reason remain unrecognized. As a matter of fact, even deep indentations often do not give rise to any visible anomalies in the behavior of the newborn infant.

Marked and serious symptoms will appear only from a coincident secondary traumatism of intracranial tissues. Since their character is entirely dependent upon the nature and extent of the resulting intracephalic lesion, they will be described in the next chapter dealing with these particular injuries.

**Diagnosis.**—Neither permanent indentations nor depressed fractures will offer any diagnostic difficulties, except when they are at least temporarily hidden beneath a large caput succedaneum or a hematoma. Such a hematoma may not be a true cephalhematoma but may be caused by an accumulation underneath the scalp of blood escaped from the injured bone.

Cranial injuries of the newborn play an important part in legal actions against mothers accused of infanticide. It, therefore, will be necessary to discuss here the essential points in the differentiation of cranial birth lesions from injuries of the newborn inflicted with criminal intent.

From this consideration, obviously then, are excluded all cases of cranial traumatism produced by, or occurring in the presence of, an attending physician. The typical and only possible defense of a mother accused of having killed her child with blows against its head is that the baby was born spontaneously in the injured condition in which it is found, or that the child sustained the injury by falling head down to the ground as the result of its unexpected expulsion in a precipitate labor.

Certain reliable observations, quoted in the foregoing pages, beyond any doubt establish the fact that indentations and fractures may, though rarely, develop in the course of normal, spontaneous labors. Under these conditions the skin covering the lesion will fail to show any marked change save a possible reddening. In every case of parturitional indentation or guttered fracture, a definite bony protrusion must be discoverable somewhere in the mother's pelvis. This, however, is not necessary if the fracture is not depressed. Careful study of the cranium is likely to reveal defects of ossification in a spontaneously injured skull bone.

In regard to a claim of the mother that she was surprised by

a precipitate labor, it must be admitted that cases are on record in which an infant's head was injured by falling to a hard floor while the mother was standing up, or by dropping into a deep open (unsanitary) toilet with the mother in a sitting position. Regarding cases of unexpected expulsion of the child with the parturient in the upright position, it must be emphasized that experience shows that both the friction caused by the mother's thighs and clothes, and particularly the resistance of the umbilical cord prevent the head from striking the floor hard, and therefore, in most instances, actually prevent an injury of the skull. Practically without exception the cord is too short to permit the baby's fall without being torn. In precipitate birth on an open toilet, the infant's fall also is retarded by the necessary rupture of the cord, and serious injury is usually precluded by the drop of the baby into soft matter. Fritsch claimed that fissures and fractures caused by fall against the ground rather characteristically run in a radial direction from the edge of the bone towards its center, but this later has been denied by Dittrich. In a series of 216 precipitate births, collected by Winckel, not one child was seriously hurt.

The unusual adaptability of the elastic head of the newborn to external trauma is demonstrated in an observation of Osler, quoted by Stumpf: The child was born in the toilet of a fast-moving train, fell to the tracks, and showed only slight contusions. Exceptional conditions, on the other hand, may lead to serious traumatism. Stumpf mentions the following observation: The child was precipitately born in the moment in which the mother, hurrying to her room, had reached the top of the staircase. Tumbling down all the steps, the baby sustained a fatal fracture of the cranial base.

It will not be amiss to emphasize the obvious fact that in all cases of the mother's claim that the injury was caused by a precipitate labor, the umbilical cord would have to be abnormally long or would be found either torn out of the infant's navel or torn somewhere else, its forcible laceration being unmistakably proved by the characteristic ragged ends. Pelvimetry may furnish a clue, since a marked disproportion between a contracted pelvis and a normal head or a normal pelvis and a large head will make it most unlikely that the labor was precipitate.

Cranial injuries inflicted by the mother or some one else in the attempt to kill the newborn most probably, though not necessarily, will exhibit some of the following characteristics: The overlying

skin is injured; shape, size, and character of the injury strongly suggest the object most commonly employed for its infliction, viz., a hatchet, hammer, heel of a shoe; the injuries usually are multiple; their location irrefutably proves that they cannot be the result either of birth or of a fall head down. A deeply depressed fracture which runs from one flat skull bone across a suture into the adjoining bone is never caused by the traumatism of a spontaneous labor, and in a precipitate labor, could occur as a result of a fall only under specific conditions which would have to be verified by inspection of the locality where the birth is supposed to have taken place. A large hematoma in the region of the cranial injury proves that the infant was alive when it sustained the parturitional or criminal trauma.

In some instances, infanticide is attempted by hitting the newborn's head against a wall, a stove, or a piece of furniture. For this purpose the baby, as a rule, is firmly grasped around its waistline. This fact occasionally is evidenced by concomitant contusions and saggillations of the skin in this region, by fracture of ribs and also at times by severe internal abdominal lesions, such as rupture of liver or spleen.

Even experts in forensic medicine, however, agree that insuperable difficulties will occasionally be encountered in determining positively whether a certain cranial injury occurred spontaneously in a normal or precipitate birth or was deliberately inflicted by the mother or another accomplice in the crime.

In malpractice suits the medical expert may be called upon to say whether in his opinion a certain cranial injury can be justly charged to lack of proper skill on the part of the attending obstetrician. There can be no doubt that not infrequently serious cranial lesions are due to the failure of the physician to recognize pelvic contractions or deformities which preclude the successful performance of forceps extraction, to unnecessary haste in breech extractions, to faulty application of the forceps and incorrect manipulation during extraction, etc. The medicolegal expert, however, will have to keep in mind the fact that under certain conditions even the greatest skill cannot prevent injury to the child, and that injuries even of a serious nature may occur in spontaneous labors. It must be emphasized in this connection that certain defects in ossification may closely simulate fissures and fractures; e.g., a detached tip of the squama of the occipital bone (Meyer). As a matter of fact, a point



properly emphasized by Goodman, as a rule it is difficult, if not impossible, to convince a mother that any injury of the newborn is not due to rough handling or to carelessness on the part of the attending physician.

**Prognosis.**—Ample clinical evidence justifies the assertion of some writers that indentations and infractions of skull bones, which cause no immediate symptoms, are most unlikely to affect the child later in life. Obviously such a statement presupposes that the search for immediate symptoms is made in a more thorough manner than at present is customary with the average practitioner and even specialist in obstetrics.

Many of the slighter depressions disappear spontaneously; the deeper ones are prone to become permanent, and in certain locations will prove more or less disfiguring.

Serious cranial injuries, especially extensive guttered fractures, in most instances cause sufficient traumatization of the brain and meninges to result in the death of the child, either immediately or within a few days. This is particularly true for the basal fractures in which often the transverse sinus is lacerated or the medulla guillotined in the occipital foramen narrowed by the slipping condyles.

In the light of our present knowledge concerning intracranial traumatic birth lesions (discussed in detail in the next chapter), we must recognize our inability of excluding with any degree of certainty a possible intracephalic damage, even if the extracranial and cranial injury is slight and seemingly unimportant. In a non-fatal case of cranial injury, therefore, it is impossible to prognosticate whether the child will be permanently well or might later in life develop physical or mental defects, and especially epilepsy, which might be ascribed to an unrecognized intracranial trauma sustained at birth. More will be said concerning this important point in the next chapter.

As a direct result of a perforating bone lesion, occasionally a meningocele spuria traumatica forms, a condition most thoroughly studied by Schindler. In these cases the dura had been torn. Cerebrospinal fluid escaped underneath the galea. The interposition of soft tissues kept the fractured edges of the bone apart and prevented their union. A subsequent hernialike protrusion of the brain actually causes the edges to separate further, and finally, with the growth of the brain, the resulting tumor may attain the size of a fist or more.

**Prophylaxis.**—The most common cause of a serious cranial injury undeniably is the forcible extraction of the head, usually of the aftercoming head, through a narrowed pelvic canal. Therefore, such injuries most successfully can be prevented by resorting to symphysiotomy, pubiotomy or cesarean section in all cases of marked disproportion between head and pelvis. This presupposes a correct obstetrical diagnosis, and most of all, the avoidance or actual elimination of all vaginal examinations in cases in which certain findings or the character of the present or preceding labors suggest the possibility of a mechanical obstruction.

It seems superfluous to urge in this connection accuracy and care in the application of the forceps and in the execution of the operation. The greater vogue of version and extraction in these past few years possibly has taught the valuable lesson that undue haste in the delivery of the aftercoming head is dangerous and unnecessary, especially if as a preliminary step the vagina and perineum have been stretched.

**Treatment.**—Not all noticeable cranial birth lesions require therapeutic interference. This fact necessarily brings the question of exact determination of the indications for interference into the foreground of the problem of treatment. Gfroerer and Hofmeier, in 1913, studied the later fate of 25 cases of depressions and infarctions of the parietal bone observed in the Wuerzburg clinic since 1895. From the viewpoint of etiology it is interesting to note that only in one instance the child had been born spontaneously in a vertex presentation, the remaining 24 children by means of breech extraction, mostly after versions. Of these, 7 had died immediately or within a few days, and as it seems worthy of emphasis, in some of these the depression had corrected itself spontaneously before death, which in every case was due to the coincident intracranial trauma. The surviving 18 infants seemed well and normal at the time of their discharge from the hospital, all between eight and ten days old. Concerning 17 of these the investigators were able to obtain reliable information. Five had died from various causes, none of them exhibiting before death any signs of physical or mental defect, or other conditions ascribable to the evident traumatization at birth. The other 12 children personally examined by the investigators were found healthy and normal in every respect. None showed an objectionable cosmetic effect. Hofmeier draws the perfectly logical conclusion that his own observations do not permit him to be fear-

ful of delayed consequences. He adds as his opinion that if there should be coincident intracranial injuries sufficiently serious to cause immediate symptoms or liable to lead to later sequelae, the mere correction of the depression could hardly be expected to do any good. Therefore, the symptomless case is best let alone, but if there are signs of an intracerebral disturbance, the treatment should be surgical and not merely corrective as accomplished with massage of the surrounding bone or the corkscrew elevation. Goodman also advises most earnestly to let the case severely alone if there are no definite and urgent indications for an operation.

One of the simplest corrective procedures for a symptomless indentation of a flat skull bone is the manual redressment achieved by squeezing and massaging the bone surrounding the depression. This method seems to have been first suggested by Murray and Kerr (1901), and proves fairly effective in a limited number of the cases. We find this method still recommended by Hoffmann, one of the most recent writers dealing with this question. It must be understood, as already pointed out by Hofmeier, that its actual effect can hardly be more than cosmetic.

Heister (as quoted by Soli) attempted to lift the depressed bone by attaching over it a piece of adhesive. The first rational attempt to correct the indentation by means of an instrument, fastened into the bone, must be credited to Tapret (1877). Then followed the various methods, known in literature by the names of Vicarelli, Mangiagalli and Baumm, in which after a preliminary incision of the skin a hold is secured of the bone by means of a corkscrewlike instrument. This principle of correction of the deformity was greatly popularized by Hauch's contention and proof that such a screw device can be safely inserted directly into the bone through the properly disinfected skin without any incision.

However, in spite of various safety devices suggested as modifications of the corkscrew, occasionally the sharp-pointed end of the instrument will penetrate the skull and injure the underlying dura and in other instances will fail to secure a hold firm enough to permit the pulling up of the depressed bone. This led to the attempts of lifting the infraction by means of a spatula (Boissard) or a short right-angled hook (Kosmak) inserted between dura and skull bone. Villard and later Boni, when publishing their very encouraging results with the spatula maneuver, emphasized the advan-

tage of any procedure that establishes an opening through the bone and thus secures drainage.

Even before the problem of intracranial birth hemorrhages was thoroughly understood, the fact had been known that at times a subdural hematoma develops underneath a lesion of the cranial bone. The spatula method of correction, therefore, actually includes the principle later deliberately developed in the trephining operation (Viannay).

Baumm, Hauch, Herff, Kuestner, Oeri, Scheffzek, and others have recorded many successful corrections with the corkscrew method. If we, however, analyze their cases, we can easily discover a few in which to-day the mere correction of the depression would be considered entirely insufficient as a therapeutic measure. In a case of Herff the depressed bone under manual redressment "fairly snapped into position." The infant died fourteen hours later, as shown at autopsy, from an intracranial hemorrhage due to a tentorial tear. Kuestner employed Baumm's instrument—so constructed that an injury to the dura is impossible. The child died four days later. The dura had been actually perforated by the tip of the screw. A large hematoma responsible for the death, however, was not found at the hole through the dura, but underneath the tentorium. Oeri in one of his cases successfully corrected the spoon-shaped depression in the parietal bone, but discovered at the post mortem examination a spinal hemorrhage due to a fractured vertebra.

In some of these cases the writers specifically mention that the child was spastic or in a stupor. With the information acquired within the past decade concerning the pathology and symptomatology of birth injuries of the head, the modern obstetrician, in the presence of such symptoms, cannot be satisfied with the mere mechanical restoration of the depression.

Thus better understanding of the intracerebral consequences of severe traumatization of the head at birth has led from mechanical correction of a depression and trephining at the site of the indentation to the osteoplastic flap operation, deliberately planned to reach a definite region of the brain. Further consideration of this operation is better left for the subsequent chapter.

From the viewpoint of therapy, parturitional depressions of skull bones, with or without actual break in the continuity of the bone, have to be divided into two groups: (1) Those cases, in

which most careful examination and observations fail to reveal any symptoms ascribable to an intracranial damage, and (2) those with definite or suggestive signs of an intracephalic disturbance of some sort.

In the first group absolute conservatism will be the method of choice if the indentation is not excessively deep and positively does not represent a depressed fracture. Correction of the depression in this group of cases by manual redressment or by the corkscrew, spatula or similar methods, is not free of danger, is not likely to relieve to any noteworthy degree possible intracranial pressure and, for this reason, can have solely a cosmetic effect.

Such advocacy of conservatism for the symptomless case, however, in no way implies that the depression is considered harmless and not worthy of further observation. Serious symptoms, calling for prompt therapeutic interference, not so rarely will manifest themselves within the next few days or later.

In the case of the deep depression, the bone might safely be assumed to be fractured. Careful operative elevation will be desirable for these cases, even in the absence of immediate symptoms, as a prophylactic measure against such possible delayed consequences as epilepsy of the Jacksonian type.

In all cases in which respiratory anomalies, spasticity, convulsions, stupor or other symptoms indicate or suggest an intracranial traumatization, manual redressment will prove disastrous, and mere elevation of the fractured bone an obviously inadequate procedure. These cases must be managed in accord with a therapy by experience found suitable for the various types of intracranial injuries, and, therefore, is discussed in detail in the next chapter.

## LITERATURE

- Baumm. *Zentralbl. f. Gynäk.*, Leipz., 1903, 27:569.  
Boni. *La Ginecologia*, 1910, 7:705.  
Brindeau. *Arch. mens. d'obst. et de gynéc.*, Par., 1918, 7:103.  
Finkelstein. *Lehrbuch der Säuglingskrankheiten* (quoted by Gfrörer).  
Fritsch. *Müller's Handb. d. Geburtsh.*, 3:637.  
Gfroerer. *Ztschr. f. Geburtsh. u. Gynäk.*, Stuttg., 1914, 75:101.  
Goodman. *Arch. Pediat.*, 1915, 32:587.

- Hartmann. Vierteljahrsschr. für gerichtl. Med., 1911, 41:21.
- Hauch. Obstétrique, 1911, abstr. in J. Obst. & Gynec. Brit. Emp., Lond., 1911, 20:84.
- Herff. Zentralbl. f. Gynäk., Leipz., 1912, 36:1265.
- Hoffmann. Med. Klin., Berl., 1911, 7:1772.
- Hofmeier. Zentralbl. f. Gynäk., Leipz., 1913, 37:1510.
- Kosmak. Am. J. Obst., N. Y., 1913, 67:264.
- Kuestner. Zentralbl. f. Gynäk., Leipz., 1908, 32:830.
- Meyer. Arch. f. Gynäk., Berl., 1912, 96:280.
- Oeri. Hegar's Beitr. z. Geburtsh. and Gynäk., Leipz., 1909, 13:309.
- Reuss. Krankheiten des Neugeborenen, Berlin, 1914.
- Scheffzek. Zentralbl. f. Gynäk., Leipz., 1908, 32:831.
- Schindler. Ztschr. f. Kinderh., Berl., 1912, Ref. 3:1.
- Soli. Arch. f. Gynäk., Berl., 1912, 97:283.
- Stumpf. Winckel's Handb. d. Geburtsh., 3:3, pp. 490, 628.
- Viannay. Revue pratique d'obstétrique et de pédiatrie, abstr. in Zentralbl. f. Gynäk., Leipz., 1912, 36:1160.
- Villard. Ann. de gynéc., et d'obst., Par., 1902, 57:223.
- Zinsser. Charité Ann., Berl., 1911, 35:492.

## CHAPTER V

### INTRACRANIAL INJURIES

Historical facts—General pathology—Classification of intracranial traumatic lesions—Cephalhematoma internum—Subarachnoidal hemorrhages—Dural hematoma—Brain hemorrhages in ventricles, or in brain substance—Ischemic necrosis—Intracranial injuries without hemorrhage—Etiology of intracranial lesions—Immediate causes—Mechanical causes of subdural hematomas—Tentorial lacerations and ventricular hemorrhages—Predisposing causes of intracephalic hemorrhages—Hemorrhagic diathesis and manipulations during resuscitation as contributory causes—Frequency of intracranial injuries—Symptoms of intracranial hypertension—Convulsions—Paralysis—Localizing symptoms of supratentorial, infratentorial and ventricular hemorrhages—Diagnosis of intracranial injuries—Changes in fetal heartsounds—Delayed coagulation time of the infant's blood—External evidences of traumatization—Ophthalmoscopical examination of the newborn—Spinal puncture—Problem of differential diagnosis—Prognosis of life—Prognosis of future health in regard to possible physical and mental defects—Prophylaxis of cranial injuries in forceps extractions, in the management of the aftercoming head, during premature labor, during protection of the perineum and during resuscitation—Dangers of twilight sleep—Treatment of intracranial hemorrhages by means of injection of blood or serum, by spinal puncture—Symptomatic and surgical therapy.

Of all cephalic injuries caused by the trauma of birth those affecting the contents of the skull, brain and meninges are most important. This fact is duly appreciated in older medical literature. It always seemed obvious that all severer injuries of the cranial bones, such as deep indentations and guttered fractures, as a rule, will also damage intracranial structures. It is more surprising that it has been known for a very long time that serious intracranial lesions, especially meningeal hemorrhages, not so rarely are encountered in autopsies of newborn infants even in the absence of any evidence of traumatism. As early as 1832 Cruveilhier offered excellent illustrations of intracephalic hemorrhages of the newborn in his pathologic-anatomic atlas. This condition has been discussed thoroughly from the anatomic point of view by Weber and also by Virchow in 1852. The description of the pathologic anatomy of these subdural hematomas furnished by Kundrat in 1890 up to the present time has been changed merely in certain details.

In considering this topic historically it seems striking that it was not the obstetrician but the neurologist who first manifested interest in the clinical aspect of the problem.

Little of London, in 1843, in his first paper dealing with cerebral spastic paralysis in young children asserted that the condition is due to a lack of development of cerebral tissues and also to a meningitis, and mentions rather casually that this type of paralysis seems to follow difficult and prolonged labor with or without the use of instruments. He expresses the opinion that palsy subsequent to an abnormal labor is due to an intracranial hemorrhage. In a second paper, published in 1862, he asserts that cerebral spastic palsy in children is the result of an intracranial birth hemorrhage in about three-fourths of all instances.

There will be opportunity to quote later in detail the views of other neurologists concerning the possible or probable relation of brain trauma at birth to the later physical and mental development of the child. It, therefore, will suffice to state in this connection merely that the neurologist has manifested a keen interest in this problem early and has maintained it ever since. In chronological order, probably the pediatrician has to be mentioned next as concerning himself with the question. A short monograph of Finkelstein, published in 1902, reveals a keen appreciation of the outstanding importance of birth trauma in the health and physical development of the infant.

The last to enter the field was the obstetrician. It can be truthfully asserted that his researches have finally led to a better understanding of the etiology and pathology of intracranial birth lesions and are directly responsible for the general interest of late visibly evidenced in the numerous scientific contributions to this subject.

The impetus for this renewed and keener interest, it seems, has been furnished by the classic paper of Seitz (published in 1907) dealing in the main with the symptomatology of brain hemorrhages in the newborn, and the paper of Beneke (1910) on lacerations of the tentorium. It is a fact, historically interesting, that solely the new method of opening the infant's skull at autopsy (described by Beneke) has permitted us to form a definite and clear conception of the exact pathology and actual frequency of intracranial injuries caused by the birth trauma. The student of the extensive literature devoted to these lesions must keep in mind the important fact that exact and reliable knowledge concerning the intracranial hemor-



rhages of the newborn infant has been gained only within the last ten years, and that many of the older contributions, still extensively and rather indiscriminately quoted by recent writers, often express nothing but mere surmise or theory. It is a deplorable fact that modern textbooks of obstetrics in this question of the cranial birth trauma express entirely erroneous views and in elaborate statistics of neonatal mortality practically never mention or classify the one important and common cause; viz., intracranial traumatic lesions.

**Pathology.**—It seems appropriate to begin the discussion of the pathology with a description of those methods of autopsy which alone permit exact determination of the existing intracranial conditions.

Seitz suggested for all cases of suspected intracranial lesions, whenever possible, severing the head immediately after death and hardening it in a freezing mixture before opening the skull. The cadaver, however, must not be permitted to lie any length of time in the usual dorsal position because gravitation invariably will cause some of the blood to flow backwards and, as has been pointed out, by Henschen, to actually reach the spinal canal after death. This method furthermore is not entirely satisfactory because it does not preclude the disappearance of flattening of convolutions under increased intracranial pressure. It, therefore, seems preferable to open the skull immediately in accord with the method devised by Beneke. The customarily employed technic destroys the tentorium and for this reason heretofore has not permitted a proper appreciation of the frequency and character of tentorium injuries. Beneke opens the skull in the sagittal suture and carefully pulls the parietal bones down on either side. Rough manipulation may result in injury to the underlying dura mater, and, indeed, might cause a tear in the tentorium. However, in the case of such an artefact, no hemorrhage will be found associated with the visible injury. Both hemispheres of the brain then are ablated and the tentorium thus laid free for inspection. In a later paper Beneke and Zausch suggested a slight modification of this technic for certain cases. In the execution of the older method the vena magna Galeni is severed, and thus the anatomic evidence of the source of the hematoma might be destroyed, e.g., in a ventricular hemorrhage. In such cases the typical Beneke method must be changed so that each hemisphere is removed separately under careful preservation of the corpora quadrigemina and of the large ganglia. Next the cover of the third

ventricle is lifted. Only in this manner the location of a tear in the vena magna Galeni can be rendered visible.

In order to avoid an injury to the longitudinal sinus, it has been suggested that incisions be made on either side of and parallel to the sagittal suture through the edges of the parietal bones by means of sharp scissors before the parietals are carefully bent down on either side.

## CLASSIFICATION AND PATHOLOGY OF INTRACRANIAL TRAUMATIC LESIONS

For practical purposes intracranial birth lesions most conveniently are divided into those characterized by a large hemorrhage and those in which but little or no blood is extravasated. Further subdividing these two groups, we arrive at the following classification:

### *A. Hemorrhages*

- (1) Cephalhematoma internum
- (2) Subarachnoidal hemorrhages
- (3) Dural hematoma
  - (a) Supratentorial
  - (b) Infratentorial
  - (c) Mixed type
- (4) Brain hemorrhages
  - (a) Ventricular
  - (b) Diffuse (Couvelaire) or circumscribed (Kruska)

### *B. Lesions without Hemorrhage*

- (1) Contusio cerebri
- (2) Ischemic areas (Kruska)
- (3) Slight tentorial lacerations.

**Hemorrhages**—**CEPHALHEMATOMA INTERNUM.**—In dealing with the cephalhematoma externum we had occasion to point out certain features in the embryonic development of the cranial bones which explain the fact that some of the vessels of the cranial periost pass directly through the skull bones into the firmly attached dura mater. Therefore, even in the absence of definite bone lesions external traumatism in the newborn may cause the formation of an epidural hematoma. It seems likely that in many instances an

external cephalhematoma thus will be accompanied by an internal cephalhematoma. The fact is occasionally mentioned in clinical reports (e.g., by Sidbury). The opportunity for more exact study of this type of intracranial hemorrhage is but rarely offered since the firm attachment of the dura to the skull bones apparently prevents the formation of an epidural hematoma large enough to cause the infant's death.

Leaving out of consideration in this connection epidural hemorrhages incident to injury of a cranial bone, we can state that the typical cephalhematoma internum, as a rule, represents only an accidental finding at autopsy, and so far as known, is void of any clinical significance also in the later life of the child.

**SUBARACHNOIDAL HEMORRHAGES.**—Hematomas in the subarachnoidal space, so-called leptomeningeal hemorrhages, lie on the convex surface of the brain hemisphere, more frequently near the midline. Occasionally they are bilateral, when they will be more pronounced on one side. There is ample pathologic evidence available for the conclusion that after breech and forceps extractions minute subarachnoidal hemorrhages are not by any means rare. Ziehen thought they may occasionally be responsible for convulsions of the Jacksonian type. In accordance with their more common distribution spasmodic contractions released by them would be more pronounced in, or may be limited to, the lower extremities. At necroscopy of a premature infant, born after an easy spontaneous labor and dying thirty-six hours later, Beneke and Zausch found a large subpial hematoma in the posterior portion of the brain covering the cerebellum on both sides.

In the opinion of Reus, however, in this type of intracranial hemorrhage in the overwhelming majority of instances definite clinical symptoms fail to appear.

**DURAL HEMATOMAS.**—Though much rarer than the subarachnoidal hematomas, subdural hemorrhages are of decidedly greater clinical significance. Indeed, they represent from a practical point of view the most important group of all intracephalic parturitional hemorrhages. When writers without further qualification still speak of cranial birth hemorrhages, they are actually referring solely to the supra- and infratentorial subdural hemorrhages of the newborn.

In marked contrast to conditions in the adult, in the newborn infant, dural hemorrhages with but rare exceptions are of venous origin, and arise from the median meningeal artery only in some cases of guttered skull fracture after forcible delivery. Statistical

proof of the prevalence of the subdural type among fatal intracranial hemorrhages can be found in the study of Kowitz, based on six thousand autopsies of newborn or very young children. In accordance with their respective frequency in the cadaver he arranges these hemorrhages as follows: subdural, subarachnoidal, ventricular, and strictly cerebral.

Seitz's first investigations seemed to confirm the older view that subdural hemorrhages in the newborn lie most commonly over the convex surface of the brain hemispheres underneath the parietal bones. The error of this conception is evident in the light of our more recent information concerning tentorial tears, which had been entirely overlooked and were practically unknown up to the first studies of Beneke. To-day authorities seem to agree that intracranial hemorrhages of clinical importance in the newborn most often are situated just above or below the tentorium.

The tentorium, as pointed out by Beneke, consists of an upper and a lower blade. The upper blade is the one more commonly torn, usually at its free edge. The tear varies in extent and shape, but in general indicates a tearing of the fibers perpendicularly across their direction. In the depth of the opening the thinner and transparent lower blade is seen intact, covering the cerebellum.

Pott differentiates three types of typical lacerations of the tentorium. The first and worst form is a tear through the free edge, usually lying about in the middle, but often it is bilateral and through both blades of the tentorium. The torn edges are ragged, fringed, covered with thick coagula of the blood which has escaped from the injured veins running along the free margin. The blood escapes downward and upward, as a rule, apparently more of it spreading above the tentorium forward into the temporal fossa, or rising laterally over the surface of the occipital lobe. Less frequently blood flows downward and covers the cerebellum and medulla, finally also passing into the spinal canal.

In the second, milder type, so accurately described by Beneke, the laceration involves only the upper blade. The resulting hemorrhage is likely to be less severe.

In the third and least serious type, in all probability the most common type, the hemorrhage often seems limited to a small hematoma between the blades of the tentorium or at the end of the falx. This type apparently in most instances is clinically without importance and is likely to be overlooked even in a careful autopsy.

Beneke emphasizes the striking fact that this pathology of the tentorium was known to Cruveilhier almost ninety years ago and was the subject of a paper published by Virchow in 1850. It has been forgotten, apparently on account of the faulty post mortem technic generally employed in which the tentorium is severed from the pars petrosa of the temporal bone before it can be inspected.

In hemorrhages of the frankly *infratentorial type*, especially in the cases in which the large veins emptying into the sinus transversus are torn, the cerebellum and the adjoining portions of the medulla are found more or less completely covered by extravasated blood. Usually some of the blood flows down into the upper portion of the spinal canal.

In some of the cases of tentorial lacerations a *mixed form* of hemorrhage is produced by some of the blood escaping upward above the tentorium. Infratentorial hemorrhages, arising as a rule from tentorial tears, but occasionally also from lacerations of the intervertebral joint capsules of the upper vertebrae (Henschen), in general are less extensive than the supratentorial hemorrhages.

Seitz, who has given us the most elaborate study of the *subdural hemispheric hematoma*, gives credit to Kundrat (1890) for the first information concerning this type of intracranial hemorrhage. He had found them often in autopsies of young infants with small heads, born after short normal labors.

This hematoma between dura and arachnoidea, as a rule, lies only over one hemisphere. It varies considerably in size. If extensive, it may, in caplike fashion, cover the entire hemisphere. The quantity of extravasated blood has been estimated by various authorities between 40 and 90 cubic centimeters. Of great clinical importance is the fact that a large hematoma of one side distinctly compresses also the other hemisphere.

To avoid repetition, it will prove more convenient to mention anatomic details in reference to the source of the extravasated blood later in the chapter while discussing the mechanical causes held responsible for the injury of certain vessels.

**BRAIN HEMORRHAGES—Hemorrhages in the Lateral Ventricles.**—Hemorrhages in the lateral ventricles apparently do not occur often. As a rule, the hemorrhage is excessive, and the blood flows through all the ventricles towards the medulla and into the spinal canal.

A case of this sort is graphically described by Beneke and Zausch and may serve as illustration. Dura and pia on the convexity of the brain were found normal in appearance over both hemispheres. The left

lateral ventricle was filled with blood. The clot continued through the interventricular foramen (of Monro) into the third ventricle and from there extended through the aqueduct (of Sylvius) and the foramen of Magendie into the fourth ventricle. A thick layer of blood lay under the cerebellum, surrounded the medulla oblongata and extended along the cervical cord down to the middle of the thoracic cord. Injuries to the brain tissue itself could nowhere be discovered. The extent of the hemorrhage left no doubt that it was the result of the rupture of a very large vein, situated so that the blood escaped first into the lateral ventricle. Since there was no tentorial tear, the rupture, in the belief of the writers, must have occurred in the vena magna Galeni, or in one of the main trunks of the vena cerebri interna, though this latter explanation seemed less acceptable.

The very method of autopsy of the head devised by one of the two authors (Beneke), made it impossible to establish in this instance the actual source of this profuse hemorrhage. It is in this connection that Beneke suggests the modification of his post-mortem technic already described in this chapter.

It will be shown later that other investigators incline to the belief that at least in some cases the tela choriodea must be regarded as the source of a ventricular hematoma.

*Hemorrhages in the Brain Substance.*—True intracerebral hemorrhages are decidedly rare in the newborn. In a series of autopsies, recorded by Couvelaire, the five hemorrhages discovered within the brain tissue were all found in premature fetu, weighing less than 3,000 grams. He pointed to the interesting fact that in six other premature infants, weighing, however, more than 3,000 grams, diffuse tissue hemorrhages were situated in the medulla and spinal cord. These observations of Couvelaire permit the deduction that intracerebral hemorrhages are more likely to occur in the soft brain of premature infants. Leclercq and Paput emphasized that in the traumatic hemorrhages of the cerebro-spinal system of the newborn, quite unlike the adult, the meningeal hemorrhages are so decidedly more frequent than hemorrhages in the substance of the brain or cord. Of 14 intracranial hemorrhages found by them in 30 autopsies of newborn infants, but one was discovered in the substance of the cerebellum.

An *ischemic necrosis* of the brain tissue as the immediate cause of a secondary hemorrhage into the degenerated tissue is strongly suggested in a case described by Beneke and Zausch. The infant died twelve days after version and extraction. At autopsy they found in the right tem-

poral lobe of the brain a large blood coagulum. After its removal an irregular cavity remained. Both macroscopically and microscopically the clot proved to be fresh, certainly less than twelve days old. By exclusion the authors arrived at the final deduction that the hemorrhage occurred in a primarily softened area of brain tissue. The problem of ischemic necrosis has been thoroughly investigated by Kruska. He described 20 instances of localized brain lesions, discovered at autopsy, which were not caused by hemorrhage. In his opinion, they represent ischemic foci produced by the spastic contraction of blood vessels. Necrotic areas of this sort vary in size from very small ones to extensive destruction of brain tissue. They were found either solitary or multiple. As the only possible explanation of such a vessel spasm, Kruska could suggest a reflex from an intense mechanical skin irritation. Beneke and Zausch accepted this explanation of a reflex origin of these lesions, but suggested that possibly also the sudden change of temperature might represent the required intense external irritation. From this point of view, the ischemic necrosis of the brain cannot be properly classed a traumatic birth injury of the newborn. It represents, however, a type of those intracranial lesions developing in connection with birth, which are characterized by the absence at least of an immediate hemorrhage.

**Intracranial Lesions without Hemorrhage.**—In the majority of cases of ischemic necrosis of the brain a hemorrhage fails to occur. In 3 of 20 cases described by Kruska the softened area had become infected and changed into a typical abscess. It is a mooted question whether such an ischemic necrosis might, at least in some instances, account for a porencephaly.

A *contusion of the brain* during labor has been described by Seitz. The mobility of the skull bones against each other, this faculty of the skull to be molded, as a matter of fact, actually protects the skull contents against the external trauma incident to labor. While the skull is being compressed a corresponding amount of cerebrospinal fluid is pressed into the spinal canal. Thus the skull cavity is actually, though not greatly, reduced without any compression of the brain itself. Under exceptional conditions, Seitz assumed, the brain in this manner may be contused or concussed. The newborn then exhibits symptoms, including convulsions, characteristic of intracranial hypertension, which, however, quickly disappear. Seitz acknowledged that such fleeting signs of an intracranial lesion might, however, also be produced by a slight and transient edema, or, as is claimed by other authors, even by small hemor-

rhages. It is worthy of note that Seitz's suggestion of a possible concussion of the brain during labor was offered at a time when knowledge concerning intracranial lesions was practically limited to the subdural hematoma and there was nothing known concerning the frequent injuries of the tentorium.

The fact is now firmly established that in a considerable number of lacerations of the tentorium discovered at autopsy no hemorrhage at all or but bare traces of extravasated blood are found. Such tears represent definite parturitional traumatic lesions which, though insufficient to cause the death of the infant, may give rise to only temporary symptoms immediately at birth.

**Etiology.**—The extensive literature which in the past decade has accumulated on the question of the causation of intracranial birth injuries shows a bewildering confusion of ideas. Their frequent combination with external traumatic lesions, their anatomic characteristics, and their location brought *mechanical factors* as the immediate cause of their production into the foreground. The normal physiological reduction of intracranial space during molding, but especially forced and exaggerated compression in the cases of mechanical dystocia or of operative delivery, always seemed most obviously to represent the mechanical factors responsible for the intracephalic injuries. Further studies, however, revealed the fact, quite evident now, that extensive intracranial destructions often are discovered within the skulls of infants that were born spontaneously after easy and quick labors and even in children delivered in cesarean section. An explanation of the intracranial traumatism on the basis of mechanical influences at least for such cases seemed unsatisfactory. Thus writers added as causative factors, prematurity, syphilis, asphyxiation, and more recently hemorrhagic diathesis. Probably all these elements enter into the etiology of the various intracranial birth lesions and especially of hemorrhages, but on more careful study one can easily see that in the individual case they vary greatly among each other in significance.

Mechanical factors, probably almost always, are the immediate and direct causes of the traumatic injury. It seems plausible, however, that the effect of the trauma is likely to be severer if the brain is unusually soft or the vessel walls unusually fragile. Prematurity or lues then will represent definite etiological factors which *predispose* the intracranial tissues to more extensive damage. Again, if the infant's blood is lacking in its normal ability to coagulate, or if an infant with but a slight intracranial injury is roughly handled during resuscitation (swing-



ing after the method of Schultze!), then hemorrhagic diathesis and violent efforts at resuscitation obviously *contribute* in the origin of serious and fatal intracranial birth injuries. Asphyxiation of the newborn, to which many writers assign a most important etiological rôle, probably acts both as a *predisposing* and a *contributory* factor, because an engorged sinus or vein not only is more prone to rupture, but also to extravasate more blood.

In accord with his own theory, each writer gives particular prominence to the one or the other of the many factors which undoubtedly enter into the etiology of these lesions. In my belief they are more appropriately grouped as *immediate*, *predisposing*, and *contributory* causes.

**IMMEDIATE CAUSES.**—Immediate causes of intracranial lesions in the main are undeniably of a mechanical nature. The mechanical origin of an intracranial injury is obvious in the presence of extensive or deep depressions and fractures of skull bones, the result of severe pressure, either exerted directly by a forceps blade, or produced indirectly by forcible traction of the head along protruding portions of the rigid pelvic canal (promontory, symphysis, exostosis, etc.). These injuries have been adequately discussed in a preceding chapter.

Less apparent is the mechanical cause of the traumatization of certain structures within the skull in the absence of any evident mechanical obstruction to the passage of the fetal head.

A definite compression with reduction of the volume of the skull represents an integral element of every normal labor. Experience proves that in general this compression is free of any noteworthy harmful effect on the newborn child. This forces the conclusion that under normal conditions a process is at work which precludes a pathologic increase of intracranial pressure. As now understood, this protection of the skull contents, and, most important, of the brain tissue, against compression is procured by the escape of a small amount of cerebrospinal fluid toward the spinal canal, and furthermore by a reduction of the volume of the blood within the brain. This automatic adjustment of skull contents to the changed skull volume during the second stage of labor is so very satisfactorily accomplished in the overwhelming majority of instances because the actual diminution of the intracephalic space is comparatively insignificant. During the process of molding, as a matter of fact, the reduction in the length of certain diameters, chiefly of the lateral, is accompanied by a corresponding increase of others, chiefly the longitudinal. It will be shown later how important a rôle is

played by this lengthening of the anterior-posterior diameters in the causation of injuries to the tentorium. Here it will suffice to emphasize that experience shows that the elastic brain easily adapts itself to the altered configuration of the head.

There still remains the question to be answered, why these mechanical factors, at play in all labors, only at times should become responsible for serious intracerebral injuries.

The explanation formerly given, and accepted as entirely satisfactory, was to the effect that serious damage is done only if either molding is excessive or is accomplished too quickly by an artificial or operative delivery in those cases in which the dystocia is due to a disproportion between the fetal head and the pelvis. Thus developed the conception, still all too prevalent, that the infant's brain is seriously damaged only in a difficult labor or by an instrumental delivery. A thorough modification of this view is required. The fact is now firmly established, that also the physiological traumatism of a normal labor is sufficient to lead to serious consequences if other coincident conditions supply those predisposing and contributory elements which will cause the normal physiological effects of the birth trauma to attain pathological importance.

Following approximately the classification adopted in preceding pages for the discussion of the pathology of intracranial traumatic lesions, we shall, in a similar manner, group direct mechanical factors responsible for the pathology into those causing hemispheric hemorrhages, tentorial tears and ventricular hemorrhages.

*Mechanical Causes of Subdural Hematomas.*—When the head is fully molded the effected change in the relative position of adjoining cranial bones is most pronounced in the sagittal suture. As the result of this overriding of the edges of the parietals the subjacent dura is both folded and stretched. Only if the dura is abnormally fragile and the overlapping excessive, or very suddenly accomplished, does the dura break and the longitudinal sinus itself tear open. More commonly, as demonstrated by Kundrat and Seitz, under these conditions only the veins of one side are torn, exactly at the site of their entrance into the sinus. They give way to the combined effect of traction and distention, the latter being either due to local congestion from partial compression of the vessels, or, in cases of asphyxiation, being the local expression of a general venous congestion.

Exaggerated overlapping of the parietals over the squama of

the occipital bone, in the belief of many writers, in an identical manner is prone to cause rupture of the veins emptying into the transverse sinus, or laceration of the sinus itself, especially in breech extractions.

Subdural hematomas have been discovered at autopsy in babies born in a state of beginning or advanced maceration. In them the hemorrhage clearly antedates birth. In cases of evident congenital syphilis the explanation seems admissible that the hemorrhage has been caused by the destruction of vessels by a syphilitic process. In other instances a definite history of a severe trauma of the pregnant uterus by a blow or fall suggests strongly the traumatic origin of the lesion, but in neither case can we speak of a parturitional injury in the stricter sense.

*Mechanical Causes of Tentorial Lacerations.*—As the result of a very thorough study of the problem, Beneke, as the first, was able to offer a most acceptable explanation of the exact mechanism of laceration of the tentorium. It is generally believed that certain histological structural details of such tissues as bones or ligaments permit reliable conclusions in regard to the functional purpose of these textures. The longitudinal direction of the fibers in the falx and their lateral extension on either side into the tentorium then would indicate that it is the chief mechanical task of the falx to prevent an abnormal extension of the long diameters of the cranium, i. e., to counteract during molding the effect of lateral compression. Sudden or severe lateral compression through the compensating elongation of the cranium thus might exert on the falx a strain severe enough to cause it to tear. Presumably the tear would occur at the weakest point, and this is where the falx fibers diverge to form the upper blades of the tentorium. Beneke's conclusion that lateral compression represents the primary mechanical factor in the causation of tentorial injuries has been fully confirmed by the observations of Bauereisen, Herff, Pott, and many others. Experimental investigations (Moreno, L. Meyer and Hauch, etc.) have demonstrated furthermore that a comparatively slight pressure is sufficient to injure the tentorium in the newborn. Therefore, some of these investigators feel justified in asserting that in some of the cases seen at autopsy the tentorial tear might well have been caused by unskillful manipulation during resuscitation, as, e. g., by too firm a fixation of the infant's head between both wrists, an almost unavoidable procedure during swinging.

This apparent vulnerability of the tentorium, even of the normal full term infant, has been convincingly demonstrated in L. Meyer's and Hauch's experiments. They discovered that also compression of the head lengthwise readily causes the tentorium to tear. Further enlightenment concerning the mechanism of the origin of these tears is offered by Benthin. He observed that after a left-occipito-anterior delivery in case of a unilateral tear the injury was on the right side, and in the case of a bilateral tear the damage was more extensive on the right side. The stronger pressure against the left parietal and the resulting flattening of the bone in this presentation causes a sharper dent in the right parietal with corresponding increased tension on the falx fibers running over to the right. Pressure in a lateral direction, he reasons, increases the vertical diameters of the cranium, and the falx thus is pulled upward, as easily demonstrated on a specimen. Traction is exerted on the fibers running transversely in the tentorium. This traction in the L. O. A. position is stronger on the right side and, therefore, is more prone to be harmful on this side.

All these observations and experimental studies tend to show that compression of the head, especially in a lateral direction, plays no less important a rôle as a mechanical factor in the causation of tentorial lacerations than we have shown it to play in the origin of lacerations of the veins emptying into the longitudinal sinus. However, there is the one important difference, that the tentorium apparently proves decidedly more vulnerable than the dura. It undeniably can be severely traumatized by a quick compression of so short a duration that the infant's head even immediately after birth will fail to exhibit such characteristics of prolonged compression as persisting overlapping of the parietals in the sagittal suture, or a large caput succedaneum. In view of these facts it becomes plausible that the tentorium occasionally is torn simply as the result of the forced passage of the fetal head through an incompletely dilated cervix or a rigid vulvar ring.

During the second stage of labor, in a general way, lateral compression prevails, but near its end, when the occiput of the fully rotated head is pressed against the symphysis during deflexion, a definite pressure is exerted in an antero-posterior direction. Benthin, and also Seitz, base on this fact the claim that most likely tentorial lacerations are produced by improper efforts to protect the perineum against injury. This seems particularly applicable to the

attempt of delivering the head in the interval between uterine contractions by exerting strong pressure against the forehead of the fetus either over the perineum or by means of a finger introduced into the rectum. In this effort the infant's occiput is forcibly pushed against the pubic arch. During such manipulations an asymmetric lateral pressure exerted with greater strength over the one frontal or parietal bone must prove particularly dangerous to the falx.

It is worthy of special emphasis that observations and experiments prove that the soft portions of the birth canal, an incompletely dilated cervix, or the rigid perineum of the primigravida, represent definite mechanical obstacles which may lead to serious intracranial traumatism. It is a matter of conjecture whether or not pituitrin from this point of view plays a direct rôle in the causation of parturitional injuries of the newborn. As a matter of fact, by a larger dose of pituitrin, the fetal head is often quickly forced through a not fully effaced cervix. In studying the numerous detailed records of severer brain injuries in literature, one cannot fail to notice the frequency with which the administration of pituitrin is mentioned in these histories. This might solely express the widespread use of pituitrin as an oxytocic, but in at least some of the cases a close relation of pituitrin to the brain injury might well be surmised, e.g., in a case of Lippman: A premature child weighing five and a half pounds developed typical convulsions 48 hours after delivery. One half ampule of pituitrin had been injected every thirty minutes during the entire labor. We find Sidbury, Neff, and Porter, among others, expressing their belief that pituitrin was undeniably responsible for many fatal hemorrhages that they had seen in newborn infants.

A study of these recorded cases of fatal intracephalic injuries, the majority to be found in German literature, also reveals the frequent mention of twilight sleep. Again this might be only the incidental result of the greater popularity of twilight sleep a decade ago among some of the German obstetricians. The fact, however, cannot be overlooked that this method of pain relief lengthens the second stage of labor and in a large number of cases requires termination of labor by forceps. In the opinion at least of Neustaedter, twilight sleep may impair the life or future health of the child in that it supplies two definite factors which are commonly held responsible for intracranial injuries.

Kuestner, Demelin, Herff, Meyer and Hauch, Fischer, and others

have placed on record observations of serious intracranial hemorrhages seen in infants delivered in cesarean section. For some of these cases it seems plausible that the injury occurred before delivery as the result of the efforts of the uterus to press the head into the narrowed pelvis. But this explanation certainly does not apply to a case like the one reported by Kuestner, in which the indication for cesarean delivery was furnished by an advanced cervical carcinoma. For some of the cases, therefore, it is evident that the head was severely traumatized solely by its forced extraction through a uterine incision of insufficient length.

Still another mechanical explanation for the causation of tentorial tears has been advanced by Wilke and Seitz. If in a breech delivery the occiput of the aftercoming head is sharply pressed against the symphysis, the force with which the cerebellum thus is pushed upward against the overlying tentorium, may be sufficient to cause its laceration. It is approximately in the same manner that Bauereisen, Mayer, Meyer, and Hauch, etc., account for the particularly injurious effect on the tentorium of the Veit-Smellie maneuver. We may quote here as an instructive illustration of the vulnerability of the tentorium of an aftercoming head a case described by Mayer: Eighth child delivered in breech presentation without the slightest difficulty. First symptoms of an intracranial injury noticeable on third day. Autopsy shows an extensive laceration of the tentorium and the sinus rectus with excessive hemorrhage both above and underneath the tentorium.

*Causes of Ventricular Hemorrhage.*—Most writers seem to assume that the ventricular hemorrhage is the result of a congestion in the vessels of the plexus chorioideus and, therefore, as a rule, is seen only in connection with deep asphyxiation of the newborn. Beneke and Zausch, and others are unwilling to accept this explanation for all cases. The usual profuseness of the hemorrhage in these cases would rather suggest the rupture of veins larger than those of the plexus. Beneke feels that in their rupture mechanical factors would seem to play the same important rôle now vindicated for all other meningeal hemorrhages of the newborn. He offers the following explanation: with the heightening of the vertical diameters of the head as the result of its lateral compression, the falx is pulled upward, and with it also the sinus rectus. Under exceptional conditions in this way not the tentorium, as is more commonly the case, but the vena cerebri magna is torn where it connects with the sinus.

**PREDISPOSING CAUSES—*Syphilis*.**—Little need be said in regard to the actual relation of syphilis to intracranial birth traumatism. Lues is mentioned and stressed as a causative factor chiefly in the older writings. Weyhe's figure of 18 per cent is still carelessly copied by those who overlook the important fact that Weyhe's paper was published in 1880, 18 years before the discovery of the Wassermann reaction. All recent contributors, who base the diagnosis of syphilis on a positive Wassermann reaction, practically concur in the opinion that this disease has significance in the etiology of birth trauma, if not entirely so, chiefly only in so far as it is responsible for prematurity of the newborn.

***Other Diseases of the Mother.***—There are sporadic observations on record which seem to suggest an increased susceptibility of the brain for traumatization in cases in which the mother during pregnancy was suffering from an acute infectious disease, plumbism, alcoholism, advanced arteriosclerosis, nephritis, etc. However, ever since it has been shown that even in cases of eclampsia the convulsions of the newborn more likely are the sequela of an intracranial lesion, caused by the forced delivery, than due to the maternal toxemia, writers have placed but little importance on maternal diseases as direct or even only as predisposing factors in the causation of intracephalic birth traumatism.

***Prematurity.***—All modern statistics of cephalic birth injuries point clearly to the etiologic significance of prematurity. The percentage of premature infants is high in all the various types of intracranial parturitional lesions, being the highest in the hemorrhages occurring within the substance of the brain or cerebellum. This fact is generally ascribed to an abnormal fragility of the vessel walls, of the dura and of the brain tissue itself, the result of incomplete development. The premature infant proves remarkably vulnerable to the traumatism of birth, and an analysis of the birth histories shows that in a large percentage of the fatal intracranial hemorrhages of prematurely born infants the labor was definitely recorded as normal and spontaneous. It was thought that such intracranial damage was due to the lessened protection offered by soft skull bones. But, indeed, investigations carried out by Kretz, forced him to the conclusion that there actually is more danger to the child from a far advanced ossification with corresponding narrowing of the open sutures. In these cases the molding of the head leads to a wider overlapping of the parietals with increased tension and pressure on the underlying dura and the veins emptying into the sinus.

An evident susceptibility to traumatization of the skull content-

in the premature child fully justifies the assertion of various writers that serious intracephalic damage can result under these conditions even if the compression is not exaggerated and only of short duration, as might be caused alone by rigid soft portions of the birth canal. Thus Meyer and Hauch found an extensive bilateral laceration of the tentorium in a child, weighing but 2150 grams, delivered by cesarean section. Beneke and also Pott described tentorial lacerations discovered in small feti of four and five months.

*Quick Release of the Molded Head.*—Abels assumes that in the second stage of labor, with the well-molded head deep in the pelvis, every uterine contraction temporarily raises the fetal blood pressure. A coincident rise of pressure also in the intracranial blood vessels is precluded by the equalizing counterpressure of pelvic and uterine walls and, in part, of the cervix. Within the area of the dilated cervix, where the compensatory outer pressure is wanting, the caput succedaneum forms. In this same region, in normally flexed occipital presentations, the sagittal suture and underneath it the longitudinal sinus is exposed to an abnormal internal pressure and becomes engorged. Sudden release of all counterpressure, by a precipitate passage of the head through the vulva, thus may cause the rupture of the engorged sinus. It is his belief that this theory satisfactorily explains the comparative frequency of intracranial hemorrhages after easy and precipitate labors, especially in small and premature babies (pituitrin!). On the basis of Abels' theory, which in general is identical with the accepted explanation of the so-called caisson disease, various writers have emphasized specific but commonly disregarded dangers of a quick forceps extraction or possibly even of the vaginal cesarean section. However, it seems that Abels' theory has not met with much favor. At least some of the observations, cited by him in its support, in the light of newer information permit of a different and more acceptable interpretation. Quick release is likely to prove more often detrimental because it causes a sudden change in the configuration of the skull. The quick release in a precipitate labor was preceded by a quick compression which is actually responsible for the intracephalic damage.

*Asphyxiation.*—To asphyxiation as a direct cause of brain injuries of the newborn a decidedly more important rôle is assigned in the writings of older, than of more recent, authors. These older writers were acquainted only with the hemispherical subdural and strictly cerebral hemorrhages in which a congested condition of the injured sinus



or veins offered a most obvious explanation for their rupture. A change of views had to come inevitably when autopsies performed after Beneke's method began to establish the great frequency of tentorial lacerations, and this new knowledge led to a proper appreciation of mechanical factors in the direct causation of intracranial birth traumatism, many of which are not associated with noteworthy hemorrhage.

To-day we are well fortified in the assertion that in very many of the seemingly asphyxiated newborn infants the asphyxia is rather the result than the cause of the intracephalic lesion, and we shall show later how efforts to overcome this presumptive asphyxiation tend to aggravate the consequences of the existing lesion.

One of the most important arguments in favor of the etiologic relation of asphyxia to intracranial hemorrhages is the irrefutable fact that in many infants, evidently the victims of a cerebral hemorrhage, small petechial hemorrhages are also found in the various serosas of the pleural and the abdominal cavity, generally held as characteristic of the death from suffocation. These coincident ecchymoses as will be shown presently are now more acceptably explained, presumably in the majority of cases, as caused by a hemorrhagic diathesis.

The etiological significance of asphyxia cannot be proved any longer by reference to statistics. Even elaborate tabulations of the immediate causes of infant mortality within the first few days of life, practically without any exceptions, still fail to mention as a cause a parturitional intracephalic trauma. The number of these deaths, as now is well known, is not small. In these statistical tabulations of *clinical* diagnosis the deaths from intracephalic lesions in part are included in the mortality figures for the various obstetrical operations, but presumably to the larger extent the traumatic deaths, especially subsequent to spontaneous labors, are counted as asphyxias. Both morbidity and mortality figures for asphyxiation of the newborn are misleading and grossly incorrect. If writers employ these figures of a clinical diagnosis of asphyxiation together with the correct figures concerning injuries as established at post mortem in calculating a percentage relation between asphyxia and intracranial birth lesions, their method is faulty and their deductions therefore unacceptable. Anatomic facts lead to the conception that parturitional traumatic injuries of almost every type are primarily due to definite mechanical conditions.

There is no positive scientific evidence available even for the contention that congestion alone does cause rhexis of the vessels in the chorioidal plexus. Asphyxiation positively cannot have any direct bearing on the lacerations of the tentorium which, as definitely established, represent the most common type of all intracranial birth injuries.

Etiologic relation of asphyxiation to intracerebral parturitional lesions, therefore, in the light of our present information, is practically limited to the plausible assumption that a congested sinus or vein is more likely than an empty one to rupture both under pressure or strain. Asphyxiation at best can be regarded only as a predisposing cause, and under certain conditions, as a contributory factor in so far as the congestion is likely to result in the extravasation of a large amount of blood.

CONTRIBUTORY CAUSES—*Hemorrhagic Diathesis*.—Robert M. Green, apparently as the first, in 1914 expressed the thought that intracerebral birth hemorrhages might be solely the local manifestation of a hemorrhagic tendency which so often can be noticed in the newborn. Further investigations, all so far carried on only in this country, have furnished ample evidence for the validity of this interpretation of the hemorrhage in a number of instances. Foote dwells on the fact often mentioned in literature that in autopsies of newborn infants with intracranial hemorrhages smaller and larger hemorrhages commonly are also seen in the mucosas and especially in the serosas of various organs all through the body. As already mentioned, these petechial hemorrhages used to be considered indicative of the asphyxia responsible for the cranial lesion. More likely all the hemorrhages in such a case are due to a common cause, a hemorrhagic diathesis. Recently Rodda published his extensive studies of coagulation and bleeding time for the newborn. He found that there exists normally a delay in coagulation, the time being for the apparently normal healthy infant immediately after birth between five and nine minutes, with an average of seven minutes. The bleeding time, ascertained with Duke's method, he established as normal in a range between two and five minutes, with the average at about three and a half minutes. Infants with evidences of abnormal hemorrhages showed a coagulation time around eleven to twelve up to thirty-two minutes. The conclusion thus seems inevitable that a congenital anomaly of blood coagulability plays a significant rôle in serious intracranial hemorrhages, at least as a contribu-

tory factor. This anomaly probably accounts for the presence of smaller petechial hemorrhages and ecchymoses also in other parts of the body where the direct traumatic cause for their development evidently could have been only of limited importance.

Rodda cites a most illuminating observation. In a case of twins the first child was delivered by means of forceps. The second smaller child followed easily in a breech presentation and died, as shown at necropsy, from an intracranial hemorrhage. The coagulation time of the surviving first child was found to be fifteen minutes, of the second child thirty-two minutes. In the second child the presumably smaller trauma incident to birth resulted in a fatal hemorrhage evidently on account of the hemorrhagic diathesis which permitted a slight, under normal conditions insignificant, intracranial lesion to extravasate a large amount of blood.

Warwick in a very recent report comments on the fact that of 53 cases of intracranial hemorrhage discovered in 136 consecutive necropsies only 12 were stillborn babies, while 41 lived for some time. In many cases the histories showed that the infants were seemingly normal at the time of birth, pathologic symptoms often not appearing for several days. In 20 of the cases the hemorrhage over the brain was associated with hemorrhages in other organs, strongly suggesting the syndrome now generally known as hemorrhagic disease of the newborn. "This at once brings up the question of the decreased coagulability of the blood being intimately associated with the etiology of the cerebral hemorrhages. One is justified in such an assumption when one finds that nearly one half of the cases of intracranial hemorrhage are associated with hemorrhages in other organs."

It is surprising that this literature does not take into consideration certain information available in surgical literature concerning the evident relation of chronic asphyxiation to a decreased blood coagulability. Investigations along these lines in regard to asphyxia of the newborn would seem most promising. They may lead to a better understanding of the hemorrhagic disease of the newborn and thus of the significance of asphyxiation as a predisposing factor in the causation of intracranial hemorrhages.

In a later chapter dealing with the treatment of these lesions we shall have opportunity to demonstrate that the therapeutic effect of blood and serum injections has furnished important clinical sup-

port for the contention that a decreased coagulability of the blood predisposes the formation of intracranial hematomas.

A delayed clotting time necessarily proves particularly serious in combination with the contributory factor to be considered next.

*Manipulations During Resuscitation.*—In many of the customary procedures applied during resuscitation of the asphyxiated newborn, most typically in the swinging after the method of Schultze, the infant's shoulders are grasped on either side and the head fixed between the balls of the thumbs or between the wrists. These swings have been denounced by many authors as objectionable for various reasons. The fact, however, seems almost generally overlooked that still another danger lies in this inevitable lateral compression of the head, which in the excitement of the occasion easily might be exerted too sharply. Bauereisen's experiments leave no doubt that such compression by itself may cause a serious tentorial tear. Since the infant manipulated in this manner is asphyxiated and its blood vessels engorged, even a small tentorium injury produced in this state is prone to cause a noteworthy extravasation of blood. Theoretically, at least, it can be assumed that a concomitant abnormal coagulability of the blood under these conditions will result in a fatal hemorrhage. A combination of conditions of this sort, probably not uncommon, seems to explain plausibly a clinical picture which is almost typical. The infant is slightly asphyxiated. It seems to be normal immediately after resuscitation, but gradually develops the characteristic signs of an intracranial hemorrhage, to which it succumbs within the next few days.

All vigorous manipulations during resuscitation, especially those in which the baby is swung or held suspended with its head downward, necessarily will favor the escape of blood even from very small ruptured vessels. In this connection reference to the fact cannot be omitted that many of the infants born after twilight sleep require efforts to overcome their seeming asphyxiation.

**Summary of Etiology of Intracranial Birth Injuries.**—In a large number of the cases the very obvious cause for the intracranial lesion is an external cranial trauma, evidenced by a deep indentation or a guttered fracture. Almost without exception these infants succumb immediately to the injury. Of much greater practical importance are internal injuries not so clearly established by external evidence. For the majority of them the responsible mechanical cause is recognized in definite anomalies of labor, breech labors, extraction of the

aftercoming head after version, difficult forceps extractions, especially in the cases of mechanical dystocia, etc. There still remains, then, a fairly large group of cases in which labor was easy, spontaneous, often precipitate, the child small, frequently premature. Also in these cases the immediate cause of the injury is a mechanical one, the compression of the head on its passage through the birth canal, both through its bony and its soft portions. Asphyxiation and, especially, prematurity render the infant abnormally susceptible to the trauma even of normal labor, and thus predispose it to serious injury. In the presence of only slight intracephalic traumatic lesions, a decreased coagulability of the blood and inappropriate manipulations during resuscitation necessarily tend to hasten or to prolong the escape of blood from injured vessels, though small, and thus represent important contributory factors in the causation of cerebral hemorrhages.

Certain mechanical factors cause intracranial tissues to be crushed or torn in the course of labor. The immediate consequences of these injuries in the main depend upon the injury to blood vessels. Blood vessels, not fully developed, diseased or engorged, are more likely to be traumatized. The escape of blood is determined by the size of the injured vessel, but is exaggerated by congestion and unduly prolonged by a hemorrhagic tendency.

**Frequency.**—The consideration of the question of frequency of intracephalic birth lesions, contrary to more common practice, here is placed after the thorough discussion of the pathology and etiology of these injuries. It is assumed that familiarity with the anatomy and causation of these injuries is requisite for the appropriate appreciation of the relevance of certain facts here presented.

Since accurate information concerning the incidence of intracranial lesions of all types, and especially of tentorial lacerations, became available only after the general adoption of Beneke's special technic of post mortem examination of the skull, older statistics have become practically valueless. I, therefore, shall limit myself to quotations from the more noteworthy publications beginning with the year 1911.

Pott (1911): When Pott became interested in this problem he discovered 14 tentorial tears in 101 autopsies. With a more careful technic and with minuter study of the findings he found the evidences of tentorial injuries in 6 out of the last 15 autopsies. He states in his conclusions: the fact that some residue of a dura hemor-

rhage can be discovered in a considerable number of the children coming to post mortem up to the age of six months, proves that the percentage of tentorial tears with some hemorrhage must be very great.

Bauereisen (1912): In 667 consecutive deliveries, some of them premature, 47 infants were stillborn or died soon after birth. In all these cases, the head was severed from the body and hardened before being opened. All hemorrhages thus were fixed in their exact anatomic relations. Tentorial lesions were found in 11 instances, at times associated with other intracranial lesions. Of these 11 babies, all were born asphyxiated, in 9 the attempts at resuscitation had failed, 2 had lived two days. All were operative cases, forceps, and breech extractions.

Benthin (1912): In a series of 1239 consecutive labors (including premature) 107 infants were stillborn or died soon. Of these, 73 came to autopsy, showing 8 tentorial tears evidently responsible for death (11 per cent of all autopsies).

Meyer and Hauch (1913): In 64 necropsies of newborn infants tentorial injuries were found in 28 instances, but only in 12 of them the tear itself was responsible for the death. Since these 12 severe lacerations were observed in a series of 1200 labors, the authors calculate for fatal tentorial tears an incidence of 1 in every 100 labors, with both slight and serious tears in over 2 per cent of all labors.

Leclerq and Paput (1913): Of 30 newborn infants coming to autopsy, in 14 an intracranial hemorrhage was found.

Henschel (1913): Of 1277 infants examined post mortem, 29 (2.27 per cent) had died from intracephalic birth hemorrhages, which represents a death rate of 0.2 per cent from such hemorrhages in all labors.

Kowitz (1914): In an exhaustive study dealing with the etiology of hemorrhagic pachymeningitis, based on 6000 autopsies of young children, the author states that fatal intracranial hemorrhages, both after spontaneous and artificial deliveries, arranged in the order of their relative frequency, involve most often the dura and least frequently the cerebral tissue itself, with hemorrhages of the arachnoidea and into the ventricles ranging between them.

Moreno (1915): In 40 necropsies of newborn babies the tentorium was found torn in 10 cases, 5 times the tear involving also the falx. This is an incidence of tentorial tears of 25 per cent in all

autopsies and of 1 per cent in all deliveries. However, not in all instances had the tentorial injuries caused the death.

Hedrén (1918): In a total of 700 necropsies, excluding all macerated infants, 65 (9.3 per cent) cases showed intracranial hemorrhages. In 50 of these 65 cases, the reliable obstetrical records showed that the labor was spontaneous, in most instances normal in every respect, only 23 of them occurring at full term, being premature in the remaining 27 cases. Hedrén gives the details of each case and presents the most exact study of the problem extant. Of these 50 intracranial hemorrhages, 42 were meningeal and only 5 of the strictly cerebral type. It seems also noteworthy that out of 27 subdural hemorrhages over the convexity of the brain he found some hemorrhages also on the other side in 21 cases. There were 6 purely ventricular hemorrhages and 2 ventricular hemorrhages combined with cerebellar hemorrhages, all 8 occurring in spontaneous labors.

Hedrén considers syphilis a predisposing factor of some importance, but lays particular stress on the striking frequency of coincident ecchymoses in serous membranes. They prove to him the significance of asphyxiation in the etiology of the intracranial hemorrhages.

Vischer (1919): In 186 autopsies of newborn infants and infants up to the age of one month, performed exactly in accord with the method of Beneke, 74 brain hemorrhages were found, among them 51 tentorial tears. This means almost 40 per cent of hemorrhages and over 27 per cent of tentorial injuries of all autopsies. In this group of 51 tentorial tears, the hemorrhage was considerable in 27 cases; it was slight or nearly absent in the other cases. Only in 23 cases of the whole group of 74 hemorrhages, the tentorium was found intact.

Warwick (1919): In 36 routine autopsies of stillborn infants and those dying in early infancy 18 (50 per cent) showed definite evidence of a hemorrhage either somewhere over the brain surface or in ventricles. Of these 18 cases only 2 represented long labors. Both were twin labors, and in both instances it was the second born of the twins who succumbed to an intracephalic hemorrhage. There was in this group only 1 case of forceps extraction, performed on a six months' fetus. Of the 18 babies, 2 were stillborn, 4 were asphyxiated at birth and the remaining 12 seemed at first perfectly normal, born after spontaneous labors. In 8 of the 18, at post mortem, macroscopic hemorrhages were discovered in other organs, forcing

the conclusion that the intracranial hemorrhages were only incidental to that syndrome of symptoms usually grouped as hemorrhagic disease of the newborn. Five of these 8 vomited blood before death and exhibited a marked delay in the coagulation time of their blood.

Bailey (1920): In 100 autopsies performed in the Manhattan Maternity Hospital in New York, intracranial hemorrhages were found in 40 cases, of which 30 were stillborn infants, the others having died within four days after birth, which in 17 instances was spontaneous and normal.

Rodda (1920): Statistics from post-mortem examinations made in the Newborn Clinic of the University of Minnesota reveal the noteworthy fact that more than 50 per cent of all infants that die intrapartum or during the first days of life, even after easy delivery, but frequently after breech and premature labors, have succumbed to intracranial hemorrhages.

Schaefer (1921): In 680 autopsies were found 140 definite hemorrhages, 114 being supratentorial, 9 infratentorial, the remaining of a mixed type. Of 43 infants born after difficult forceps extractions, 39 had serious tentorial tears. In a large number of the fatal cases the labor was spontaneous. In about one-half of the spontaneous cases, in which the infants were premature and small, the tentorium was found injured.

This wealth of statistical material, representing but a selection from the more important contributions of the past decade, does not permit accurate calculations in regard either to the total frequency of intracranial birth injuries or the relative frequency of the various types. The reasons are obvious. In some instances percentage figures are given on the basis of all post mortems including young infants, in others the stillborn or macerated feti are excluded. Again, most writers employ the term "cerebral" or "brain" hemorrhages for all intracranial hemorrhages, while but a few distinguish clearly between the various types.

However, the references quoted here are entirely sufficient to permit the following conclusions: intracranial lesions as the result of the birth trauma are very common. They are discovered with ever-increasing frequency. The chronological arrangement of the foregoing quotations from literature clearly demonstrates this fact. The percentage figures for intracranial birth injuries as cause of infantile death are growing, not because these injuries are becoming



more frequent, but because more investigators are looking for them. Among all the intracranial birth lesions hemorrhages rank first as causing death, while tentorial lacerations must be assigned the first place in regard to mere frequency. Serious and fatal lesions occur frequently in the course of spontaneous and easy labors, especially in premature infants. In a considerable percentage of the autopsies definite intracranial lesions, especially subarachnoidal hemorrhages, and tentorial tears, without any or with but slight hemorrhage, represent only accidental findings. This proves that many infants necessarily survive though they have sustained definite intracerebral lesions during birth. The important bearing of this evident fact on the later physical and mental development of the child will be discussed later in this chapter.

**Symptomatology.**—The fact now firmly established by anatomic investigations, that intracranial birth traumatism occurs with great frequency, places on the clinician the pressing obligation to provide a clearer picture of their clinical symptomatology than that available at this time. Most of the recent writers still are quoting Seitz, the recognized pioneer in the study of the pathology and symptomatology of subdural birth hemorrhages, but, as a rule, fail to appreciate that our information in regard to lesions other than subdural hematoma, and especially concerning tentorial tears, is of more recent date, and that, therefore, some of the phenomena and symptoms so precisely determined and so well described by him, call for a different interpretation.

There are various reasons which prompted me to offer in the subsequent pages so detailed a discussion of the symptomatology of cranial birth lesions. As at present, so probably also in the future, the overwhelming majority of all confinements will be managed by general practitioners. Very few newborn infants at birth or even soon afterward are seen by expert neurologists. Effective therapy, often life saving, however, is plainly dependent upon early diagnosis. Therefore, the most important of all efforts to alleviate the effects of cranial birth traumatism, must be to familiarize the practitioner with the symptomatology of these lesions, so that he will be able actually to diagnose or at least to suspect such injuries.

In the attempt to render this detailed description of the symptomatology particularly useful to the practitioner, a classification of symptoms has been adopted which is based rather on the require-

ments for a prompt clinical diagnosis, than on the anatomic-pathologic conditions which, indeed, determine the specific symptoms.

GENERAL SYMPTOMS—*Intracranial Hypertension*.—A marked dissimilarity between the symptomatology of intracranial lesions and, in special, of hemorrhages of the newborn, and those commonly seen in the adult, is plainly the result of anatomic differences in their respective skulls. Within the rigid adult skull, with the exception of a deep depressed fracture, hypertension is produced by an increase in the contents of the skull capsule. In the case of the fetus, we have to take into consideration an actual reduction of the skull volume from molding during labor, and after delivery an ability of accommodation to an increase of skull contents through yielding of sutures and fontanel.

The compression of the head during labor, if not excessive, or abnormal in other respects, apparently causes no signs of hypertension. The reduction of the skull capacity from molding probably is small, and adequately compensated by the escape of some of the cerebrospinal fluid into the spinal canal, presumably also by a hastened absorption of the fluid into lymph vessels, and possibly by a reduction of the blood volume within the cranium. This escape of cerebrospinal fluid into the spinal canal has been definitely proved by manometric measurement of the spinal fluid pressure in cases of breech labors. Spinal puncture for the purpose of reducing the size of the head and facilitating its passage in breech presentations has been advocated and successfully practiced by various obstetricians.

Temporary anemization of the brain during molding apparently does not cause any noticeable symptoms, at least not under normal conditions. It has been emphasized by various investigators that the bradycardia which could be expected as a sign of an intracerebral hypertension is uniformly absent; however, it has been pointed out that bradycardia may fail to manifest itself only because in the newborn the vagus always is markedly hyposensitive.

Excessive ischemia of limited areas is assumed, as already pointed out, to cause in some cases a circumscribed necrosis with an occasional secondary hemorrhage into this area (Kruska). Some writers explain on the basis of this type of secondary hemorrhages the cases in which the child, apparently healthy and normal at birth, develops the unmistakable symptoms of a serious intracranial hemorrhage only several days later. This peculiar and not uncommon feature in the symptom-complex of intracranial hemorrhages for

some of the cases, however, at present is more plausibly explained by the evident fact that a hemorrhagic tendency may cause a larger hematoma to form gradually and only a few days after the injury has been sustained. Delay in the appearance of the symptoms of intracerebral hypertension and of hemorrhage may also be caused by a compensatory expansion of the skull cavity made possible by the stretching of sutures and the bulging of fontanels.

If hemorrhages are small, or an edema only limited in its extent and, therefore, quickly absorbed, physical and clinical signs may be so insignificant that they likely are overlooked or possibly cannot be ascertained even by careful observation. It is in this respect that the methods of examination at present available prove rather inefficient and are in urgent need of further refinement. In the present state of our information the possibility of an existing cerebral hemorrhage must be taken into consideration in every case in which melena or hemorrhages from other mucous membranes suggest a hemorrhagic diathesis, or the anomaly of coagulability is established by a test of the blood clotting time. It also seems quite evident that a more general, or preferably routine, use of ophthalmoscopic examination of the newborn will prove of decided value in the diagnosis of intracranial lesions which fail to cause the typical clinical manifestations.

The classic symptom of an increased intracranial pressure is the abnormal tension or actual bulging of the large fontanel. The latter indicates a comparatively large hemispheric or ventricular hemorrhage, or a pronounced cerebral edema which often develops as a direct sequela of a hemorrhage. It is important to remember that the large fontanel may remain normal if the hemorrhage is small or if it is primarily and entirely subtentorial. Tension or bulging of the fontanel may be noticed almost immediately or, more commonly, will become manifest only gradually. This latter phenomenon is a most valuable sign of a progressive process. In a similar manner the observant obstetrician may be able to ascertain that only gradually also certain sutures become widened. These are extremely important points both in the diagnosis of continuation of the effusion of blood from a broken vessel and in the localization of the primary focus.

In general, newborn infants with intracranial hemorrhages at first are very restless and cry almost incessantly, a phenomenon ascribed to the pain caused by the stretching of the dura mater. If the hemorrhage is not excessive, these infants refuse to nurse. This

is due to the absence of the normal sucking reflex, easily established by gentle rubbing of the lips with a finger. In the normal newborn this irritation without exception prompts sucking attempts of the infant.

Various writers attribute the striking paleness of these children to an abnormal irritation of the vasomotor center rather than to the actual blood loss. They find a confirmation for this theory in the frequency of pronounced dermographism in these cases.

Respiratory symptoms, though hardly missing in any case, in themselves are not characteristic because in the main they are determined by the location of the effused blood. They are distinctly less pronounced in hemispheric hemorrhages than, e.g., in strictly infratentorial bleeding, when the resulting hematoma more directly affects the respiratory centers in the medulla.

With only slight intracranial hypertension respiration usually is slow and deep, at times irregular, but always interrupted by that rather persistent cry. The slow, stertorous breathing, so characteristic in the adult, as a rule is absent in the newborn. Therefore, in hemispheric hemorrhages with only slight hypertension, in the absence of convulsions, the infant rarely is cyanotic, which, on the other hand, is the common condition in infratentorial hemorrhages.

*Convulsions.*—Convulsions represent the most obvious of all symptoms of an abnormal increase in intracranial tension. They are characteristically of the cortex type. Tonic and clonic, they involve chiefly the flexors of the extremities, but often include the muscles of the respiratory group, and usually are associated with rolling of the eyeballs, twitching of the face muscles, opisthotonos, with definite rigidity of the neck especially in infratentorial hemorrhages, occasionally with trismus, especially in ventricular hemorrhages.

The convulsions vary in frequency, duration, and interval, and are easily brought on by all manipulations of the infants, almost without fail by pressure against the bulging fontanel. A loud yelling cry during inspiration at the height of the spasm is followed by complete relaxation and a period of more normal breathing. If convulsions follow each other in close succession, the child, as a rule, becomes unconscious and remains in a comatose state.

On careful observation, a state of distinctly increased reflex irritability or spasticity can be noticed preceding the first convulsion. Touch or a slight scratch will cause sharp contraction of an extrem-

ity, or, indeed, may release the first convulsive seizure. Even between spasms the flexors persevere in a spastic and rigid state.

It is necessary in this connection to emphasize the well-known fact that all reflex phenomena are notoriously uncertain in the newborn. Though information available in this respect is somewhat contradictory, Reuss presents the following summary: the patellar reflex usually is very distinct within the first few days of life and may be found to differ in intensity on both sides. The achilles reflex has been found positive in 60 per cent of the cases by Furmann, in not quite 15 per cent by Bychowski. The cremaster reflex, according to some authorities, is present in the majority of infants within the first few weeks of life, which, however, is denied by others. The abdominal wall reflex, according to Farago, can be ascertained practically in every newborn infant by a slight scratch with a needle just above the mons veneris. This has not been found to be the case in the investigations of Furmann and Bychowski. Pediatricians for a long time have been acquainted with the fact that the Babinski reflex, as a rule, is positive in the newborn. But it has been claimed recently by some writers that instead of the typical Babinski in cases of intracranial hypertension one, not rarely, obtains a dorsal flexion of the foot associated with a plantar flexion of the toes.

It seems obvious that further progress in the problem of symptomatology and diagnosis of intracranial birth lesions to a large extent will depend upon better information concerning the actual status of the reflex irritability of the normal newborn.

Rather uniformly, writers emphasize some characteristics of the convulsions which occur in the cases of ventricular hemorrhages. These hemorrhages usually are profuse. All the symptoms of hypertension appear promptly. The convulsions often are of a decidedly tonic type. This is particularly pronounced in the extremities which seem rigid. Most characteristic is a trismus, a fact which accentuates the close resemblance of the symptomatology of ventricular hemorrhages in the adult and in the newborn, but also adds to the difficulties in the differential diagnosis from tetanus. Seitz emphasizes that in cases of ventricular hemorrhages careful observation might reveal that the upper extremities, at least at first, are more extensively involved than the lower, explained by him by the anatomic fact that a larger quantity of the blood escaping through the fourth ventricle is accumulated in the upper section of the spinal canal.

If a great deal of blood, either in infratentorial or in ventricular

hemorrhages, enters the spinal canal, some of it may reach the lumbar cord and the cauda equina, and then give rise to such symptoms as erection of the penis or contraction of the scrotal skin.

*Paralysis.*—As the intracranial hypertension increases, chiefly as the result of further extravasation of blood, the primary excitation and spastic condition gradually yields to a paralytic state, especially of the muscles of the extremities. Increased local pressure has finally paralyzed certain motor centers. Obviously in many cases death supervenes before this secondary paretic state is reached. In the infants who survive the primary excitation phase, the sequence in which one muscle group after another passes into flaccid paralysis offers a valuable aid in the localization of the primary source of the hemorrhage and in recognizing the direction of the gradual expansion of the hematoma. This point shall be discussed later in detail. A paresis involving all extremities practically manifests the terminal stage. A most confusing clinical picture of a spastic condition in certain muscle groups with simultaneous paralysis of others, in some instances, is brought about by a combination of an irritative cortex hematoma with hemorrhages within the brain substance (chiefly ventricular) which paralyzes certain centers.

*LOCALIZING SYMPTOMS.*—Definite localization of an intracranial hemorrhage in the newborn is of greatest importance for appropriate therapeutic, especially operative, interference. From this point of view, it is essential first to determine whether the hematoma lies above or underneath the tentorium. In the case of the supratentorial hematoma, an effort must be made to localize it either immediately above the tentorium at the base of the brain, or over the convexity of a hemisphere, with a final endeavor to determine its location either over the cortex surface or within the brain itself, on one or the other side.

At the outset it might be stated that in the newborn such an exact localization, however desirable, is extremely difficult and but rarely possible. Conditions in the newborn as compared with the adult patient differ in the following essential points: Hemorrhages in the newborn are likely to be profuse. Even a distinctly localized hematoma is less prone to exhibit definite local symptoms because the pressure is more readily diffused by the flexibility and expansibility of the skull. Perfectly normal newborn infants often show definite spasticity of certain muscle groups especially in the extremities. This spasticity frequently is followed by a striking flaccidity.

While the central nervous system at birth as a whole notoriously is in a state of imperfect development, the motor centers of the cortex are functioning fairly well. Therefore, the symptoms of cortical irritation will always tend to dominate the clinical picture, even if the cortex itself is not directly affected by the hemorrhage, and, e.g., only pressed upward by a hematoma lying beneath the hemisphere.

It will be shown in the following pages that the essential feature of more exact localization consists in the careful observation of the sequence of certain symptoms which, in many instances, precede the first convulsion. It is this indisputable fact which so clearly places the duty of painstaking observation on the physician who manages the labor and thus alone has the opportunity to see the child immediately after birth.

*Supratentorial Hemorrhages.*—Hemispheric subdural hemorrhages, as a rule, are unilateral. The question as to which side is involved, is easily and reliably settled in the presence of definite unilaterality of symptoms. Every large hematoma, and usually also a secondary edema, however, unfortunately often will affect through compression the other hemisphere and thus the valuable symptom of unilaterality is quickly eliminated. In the opinion of some observers the typical bilateral convulsion always is preceded by a unilateral affection, which may be of very short duration, but is hardly ever absent. Seitz asserts that very careful observation during the first seizures often enables one to determine that the extremities of one side contract more violently than those of the other side. In this manner he succeeded in 5 out of 7 cases to diagnose correctly the seat of the hemorrhage before death.

Immediate and continued observation occasionally permits one to ascertain the gradual and successive involvement of cortex motor areas exactly corresponding to the natural course of expansion of a blood clot from its original source towards the lateral or posterior portions of the brain. Thus there might be recognized first the distinct affection of the motor center of a lower extremity in the upper third of the central convolutions of the opposite side. When the blood reaches the middle third of these convolutions, the upper extremity begins to be affected. With the escaping blood finally pressing against the lower third of the anterior central convolutions, symptoms of irritation and later of paresis will appear in the muscles innervated by the *facialis* and *hypoglossus*.

The final predominance of symptoms in the facial muscles and

the striking rapidity with which the paretic stage as a rule follows the primary and short stage of irritation, according to Seitz, also is easily explained by certain mechanical factors. The extravasated blood gravitates downwards so that the upper portion of the coagulum will be comparatively thin and the lower portion thicker, thus pressing more firmly against the cortical centers of the facial nerve. It is for this reason, according to Seitz, that an aggravation of the facialis symptoms in form of paralysis often is accompanied by a coincident recession of the symptoms in the extremities if the hemorrhage has ceased. It will be well to remember in this connection that Seitz considered only hemispheric hemorrhages. This same prevalence of the facialis and hypoglossus symptoms over certain cortex symptoms in the early symptomatology of intracranial birth lesions is quite obvious for tentorial lacerations in which the escaping blood under the base of the brain directly affects the intracranial portions of these nerves. Thus the detailed symptomatology, so carefully worked out by Seitz, as it affects the various muscles innervated by facialis and hypoglossus is of eminent value in the diagnosis and more exact localization of all supratentorial hemorrhages, whether primarily hemispheric or basal.

**NERVUS FACIALIS.**—In contrast to the more common sequence in other muscle groups, especially of the extremities, of overstimulation followed later by paralysis, in the facialis group most frequently overstimulation is of very short duration, limited to some twitching, e.g., in the *M. orbicularis oculi*, or is entirely absent, so that in most instances the first noticed anomaly is a paralysis. In this intracranial type of facialis affection, as a rule, all branches are involved, and a circumscribed paralysis, such as a lagophthalmos alone, is the exception.

Unilateral spasticity or, as more frequently is seen, a unilateral paralysis of the entire facialis group definitely establishes the contralateral location of the hemorrhage, if it can be proved that the paretic condition is of central and not of peripheral origin. However, unilateral facial paralysis of intracranial origin is but rarely observed, because in a large hemispheric hematoma, indirectly through the increased pressure, the facialis of the other side, and, in cases of basal supratentorial hemorrhages directly, both faciales usually are affected.

By far the more common type is the extracranial peripheral facialis lesion, usually caused by the direct pressure of a forceps



blade or of a protruding portion of the pelvic wall in a malformed pelvis. The peripheral nature of the trauma often is evidenced by a visible mark or depression. In these instances the paralysis always can be noticed immediately, and, as a rule, the symptoms disappear promptly within a few hours, or at least show definite improvement from day to day. In contradistinction the symptoms of an intracephalic injury of the facialis appear only from one to three days, and occasionally even later after birth, and are likely to become more marked from day to day.

All signs of anomaly in the muscles innervated by the facialis in the newborn demand most careful study and interpretation. Whenever the condition seems persistent or the symptoms are distinctly progressing, an intracranial origin should be considered. Almost conclusive for the intracephalic and truly central origin are tonic or clonic contractions in the affected muscle groups, especially if other muscles, e.g., those supplied by the oculomotorius also are involved, or if other definite signs of an intracranial hypertension are present.

**NERVUS HYPOGLOSSUS.**—This nerve usually is affected in the same sense as the facialis. In the paretic state the tip of the tongue is deviated toward the paralyzed side.

**NERVUS ACCESSORIUS.**—Affection of the eleventh nerve by a hemorrhage, most likely due to a tentorial laceration, will manifest itself chiefly in the condition of the sternomastoid muscle. The head will be turned toward the paralyzed side. Great care must be taken in the proper interpretation of this symptom; first, because of the possibility of a congenital torticollis, and secondly, because during the convulsive state the head might be held fixed in this attitude, but then suddenly thrown into the opposite direction. Various observers emphasize the absence of all neck rigidity in the cases of hemorrhages in the upper cranium as compared with its frequency in infratentorial hemorrhages.

**NERVUS OCULOMOTORIUS.**—The condition of the *M. levator palpebrarum superior* proves most useful for the purpose of more exact localization. Since the larger portion of oculomotor fibers remain uncrossed, twitching or paresis of this muscle will occur on the same side on which the hematoma lies. This same rule also holds for the contracted pupil. However, in accord with the general experience of neurologists and surgeons in the adult, in the newborn as well the interpretation of a unilateral miosis or mydriasis for the purpose of localization is very unreliable. Extreme contraction of one pupil,

together with strabismus and nystagmus, can be seen in many cases of cranial hemorrhage in the newborn. Some of the effused blood, especially in hemispheric hemorrhages, is likely to reach and to irritate the cortical center of coördinate ocular movements in the gyrus angularis at the end of the temporal sulcus. For this reason a reactionless, widely dilated pupil renders the prognosis particularly bad, though the infant may continue to live for a few more days.

*Infratentorial Hemorrhages.*—All severer hemorrhages of the infratentorial type are prone to impair the function of the important centers in the medulla oblongata to such extent that death will ensue quickly. On the other hand, it is generally conceded that slighter peribulbar hemorrhages, especially from minor injuries of the tentorium, may give rise to symptoms hardly distinct enough to permit their recognition.

In the foreground of all symptoms due to infratentorial hemorrhages stands impairment of respiration. The infant usually is cyanotic and very markedly so during a convulsion. However, in an infratentorial hemorrhage, like the signs of an abnormal intracranial tension, respiratory embarrassment may make only a delayed appearance.

If a considerable amount of the extravasated blood escapes down the spinal canal, at first convulsive contractions may be more marked in the arms than in the legs; later the signs of an irritation of the lumbar cord might be noticed in the form of contraction of the scrotal skin or an erection of the penis.

Some of the most notable differences in the typical symptomatology of hemispheric and of infratentorial hemorrhages of the newborn can be tabulated as follows:

HEMISPHERIC	INFRATENTORIAL
Infant cries a great deal during first few days.	Infant usually is very quiet, apparently is sleeping or is comatose.
Breathing center becomes affected comparatively late. Infant is pale.	Breathing center, as a rule, is affected early. Infant is cyanotic, especially during convulsion.
Death may be delayed.	Death usually occurs early.
Fontanel becomes tense within comparatively short time.	Fontanel at birth is practically normal. Increased tension, if

## HEMISPHERIC

## INFRATENTORIAL

at all, becomes noticeable only later.

Prompt appearance of symptoms of intracranial hypertension.

Symptoms of hypertension appear late.

Rigidity of neck and opisthotonos hardly noticed.

Rigidity of neck and opisthotonos usually marked.

Symptoms from affection of facial and oculomotor nerves at first may be unilateral.

Symptoms from affection of facial and oculomotor nerves, as a rule, immediately are bilateral.

In spite of such clear differences in certain classical signs, the clinical picture in the individual case is likely to exhibit a mixture of symptoms for the very obvious reason that in the majority of instances, as now generally conceded, birth hemorrhages originate from tentorial lacerations. In them blood may diffuse both above and below the injured tentorium. Even in the more common basal supratentorial hemorrhages gravity often causes some of the blood to flow downwards and affect the cerebellum and medulla. Later, as already pointed out, the relatively more advanced development of the cortical motor centers usually will cause symptoms of indirect irritation of the cortex to predominate in the clinical picture of all forms of intracranial hemorrhage. It seems justifiable to emphasize in this connection once more the importance of a most careful observation of the early symptoms and of their gradual changes.

*Ventricular Hemorrhages.*—Ventricular hemorrhages, in most cases, are profuse and lead quickly to the death of the infant. If this is not the case, convulsions are prone to be of markedly tonic type. Most writers mention a trismus as a very common and often misleading symptom.

Hemorrhages within the brain substance are comparatively rare. They are not likely to offer symptoms definite enough to permit their localization. This, in part, is due to the general underdevelopment of the central nervous system of the newborn infant.

**Diagnosis.**—The diagnosis of an existing intracranial birth lesion and its more definite localization necessarily depends upon the exact observation and correct interpretation of the various symptoms which have been described in detail in the foregoing pages. It would cause unnecessary repetition to outline here the different

symptom-complexes which permit the diagnosis of each of the various types of lesions. It seems preferable rather to point out certain general facts which are of unquestionable importance in the recognition of an intracranial birth traumatism.

At the outset we shall emphasize that the history of the labor, whether it was spontaneous, delayed, or required artificial termination, is of decidedly less value than customarily accorded it.

In most of the severer cranial lesions, the child is stillborn or dies soon after birth. In the latter group the infant at birth seemingly is in a state of deep asphyxiation. Attempts at resuscitation obviously will fail, though regular contractions of the heart may continue for a short time. An intracranial traumatism as the cause of death can be suspected when a difficult forceps extraction has caused distinct external cranial injuries, or when after a very protracted labor marked overlapping in some sutures indicates long-continued and excessive compression, especially if the child is premature. A cephalhematoma is a symptom of value only in the presence of other signs suggesting an intracranial injury. In the majority of cases of parturitional or neonatal death, the diagnosis of an intracranial birth trauma can be established only by an autopsy performed according to the method of Beneke. In this connection the fact should be borne in mind that serious and fatal injuries not infrequently occur in the course of spontaneous and quick labors.

On the other hand, no undue importance in the diagnosis of internal lesions must be placed on external evidence of traumatism, such as depression or laceration of the skin or even the indentation of a skull bone. It has been definitely established that those children who exhibit no noticeable symptoms in spite of definite external cranial lesions at birth, later in life, as a rule, remain physically and mentally normal.

Asphyxiation always should suggest the possibility of an intracranial lesion, and especially if the child immediately after the expulsion seems fresh and normal, and only later exhibits the signs of impaired respiration.

A suspicion of an intracranial trauma should be aroused whenever the newborn is either crying almost incessantly or is strikingly quiet, especially when he does not respond with sucking motions to a slight irritation of his lips with a finger. The absence of these symptoms immediately at birth and their progressive appearance soon afterwards must be regarded strongly suggestive of a gradually

developing intracranial hypertension. Particularly valuable for the purpose of diagnosis proves in this respect the observation of a gradual involvement of certain cranial nerves, chiefly facialis and oculomotorius, especially if the sequence in which new symptoms successively manifest themselves corresponds to the natural extension of a hematoma downwards and backwards as determined by gravitation.

Certain special methods of examination have proved particularly valuable in the diagnosis of existing and even of only impending intracephalic lesions.

FETAL HEART SOUNDS.—Too recently to have permitted either confirmation or repudiation, at least two authors have offered interesting information concerning the possible value of variations in the pulse rate of the fetus in the diagnosis of imminent danger to his life.

From a large series of careful observations on parturient women combined with an autopsy study of all stillborn infants, Baumm concludes that a fetal *tachycardia*, appearing without a preliminary *bradycardia* in the course of a protracted labor of an afebrile woman, is pathognomonic for a threatened intracranial hemorrhage. In a group of labors in which this phenomenon had been definitely ascertained by practically continuous observation of the fetal heart sounds, 11 babies died of brain hemorrhages, while 4 others who survived showed very definite symptoms of cerebral irritation of some sort. Baumm believes that the timely discovery of this symptom in some cases might enable the physician to save the child by quick delivery.

Baumm furthermore concludes from his observations that a *tachycardia preceded by a bradycardia* is rather suggestive of the fact that the hemorrhage already has occurred. No hope to save the child is held out by prompt delivery.

The reversed sequence of a *tachycardia followed by a bradycardia*, in his opinion, probably is the expression of a beginning mechanical asphyxiation.

However, he found fetal death may occur suddenly during labor, recognized by the sudden cessation of all heart sounds without any preliminary changes in their rate. Of 7 stillborn babies, in whom the heart sounds had remained normal up to the moment of birth, 5 showed extensive intracranial hemorrhages. Similar hemorrhages were found in 3 other infants in whom the heart had been observed to stop suddenly during labor.

E. Sachs, on the other hand, maintains that danger to the fetus is clearly denoted by a slowing of the fetal pulse below 100, or by repeated changes of the rate within wide limits even if it never slows below 100. He specifically asserts that this ominous bradycardia below 100 is characterized by the fact that it is not preceded by a tachycardia. He doubts that a retardation of the fetal pulse rate is an expression of abnormal intracranial tension, and in general is unwilling to accept all of Baum's deductions. Sachs emphatically states that a fetal pulse rate up to 160 must not be regarded as an indication for interference and positively does not justify a difficult forceps extraction which in itself implies great danger to the fetus. The only acceptable indication for immediate interference can be found in a bradycardia below 100 persisting during the intervals between a few consecutive uterine contractions.

DELAYED COAGULATION TIME.—The etiologic relation of hemorrhagic diathesis as a contributory factor in the causation of serious intracranial hemorrhages has been adequately discussed in the foregoing pages. The importance of establishing or excluding an anomaly of the coagulability of the blood of the newborn, in every case suggestive of a cerebral hemorrhage, seems self-evident. A comparatively simple and for all practical purposes sufficiently accurate method, found useful by Rodda, may be briefly described as follows: The heel of the infant is cleansed with ether. A puncture is made with a sterile lance blade, deep enough to produce a free flow without requiring any squeezing of the skin. A clean watch glass containing a No. 6 shot receives the second drop of blood. Another watch glass is inverted over the first. The glasses, held together, are gently tilted every thirty seconds until the shot no longer rolls, but is fixed in the clot. In the end the shot is so firmly imbedded that the glass may be inverted without dislodgment of the shot.

The greatest source of possible error lies with the blood flow. Blood obtained by pressure from too small a puncture will clot very quickly. For the sake of comparison it is preferable always to use the second drop; it coagulates faster than later drops.

This method does not pretend to ascertain the absolute clotting time, but the result is a clear-cut relative time for comparative work. Its object is to discover merely gross variations from the normal range. The average coagulation time in the newborn, ascertained by this method, is seven minutes, with a normal range between five to nine and up to eleven minutes. The newborn normally exhibits

a tendency to prolongation of clotting time during the first days of life. Hemorrhagic conditions show a distinct exaggeration of this tendency or a markedly prolonged coagulation time up to thirty and forty and even ninety minutes. In Rodda's belief routine determinations in the newborn will allow the recognition of at least certain hemorrhagic conditions before the onset of symptoms, and thus will give the opportunity for prompt or even prophylactic administration of blood or serum.

Markedly delayed clotting time or such evidences of a hemorrhagic diathesis as hematemesis, melena, hematuria, or petechial hemorrhages in visible mucous membranes, in the presence of other clinical signs will always strongly suggest or actually confirm the diagnosis of an intracranial hemorrhage.

**PROLONGED BLEEDING TIME.**—According to Rodda the bleeding time, ascertained by the method of Duke, in general shows the same type of curve as the clotting time. The bleeding time in normal newborn infants averages three and a half minutes, with a normal range between two and five minutes. Hemorrhagic disease in most instances shows a corresponding marked prolongation of the bleeding time up to fifteen and twenty minutes.

**EXTERNAL TRAUMATISM.**—With the exception of evidently perforating injuries, fractures of skull bones and very deep indentations, other external traumatic birth injuries of the head do not permit reliable conclusions concerning intracephalic conditions.

A very large caput succedaneum indicates a long-continued molding, but not that sudden and excessive compression of the head which is of such great importance in the causation of intracranial traumatization.

A cephalhematoma one finds frequently mentioned in the necropsy findings of infants with severe internal injuries. However, there is no direct relation of the true cephalhematoma to the serious and fatal cerebral damage. It has been mentioned in a preceding chapter, that anatomic conditions, the passing of blood vessels from the pericranium directly through the skull bones into the underlying dura, may cause small flat epidural hematomas to form close to the inner surface of the cranial bones. This epidural hematoma, however, represents only an accidental finding at autopsy, and, so far as is known, is of no particular clinical significance.

In cases of perforating cranial injuries some of the blood of a subdural hematoma may escape through the opening from the

cranial cavity underneath the scalp. The resulting hematoma on superficial examination may resemble a typical cephalhematoma. Its true origin and with it an intracerebral hemorrhage will be revealed by pulsation, and still more clearly, if pressure against this hematoma releases the typical symptoms of intracranial hypertension.

A very marked overriding of skull bones in certain sutures, observed immediately after birth, especially after a forced labor (quick forceps extraction, pituitrin, etc.), must be regarded as suggestive of a possible internal traumatism. On the other hand, this symptom may disappear very rapidly, indeed, possibly as the result of a rapid increase of intracranial tension from a hemorrhage.

**OPHTHALMIC EXAMINATION.**—Though employed and advocated for many years as a most valuable aid in the diagnosis of cranial birth injuries, the ophthalmoscope still is but rarely used in obstetric clinics. This lack of interest presumably is due to definite technical difficulties in its use on the newborn.

The first examinations of this kind date back to Jaeger in 1861. An elaborate paper by Stumpf and Sicherer presents an interesting history of the various efforts made in this direction. These two investigators, from the study of a very large material, arrive, among others, at the following conclusions: The corpus vitreum was always found normal. The eyeground of the newborn in general resembles that of the adult, though, as a rule, all the colors are lighter. If the examination is made very soon after birth a distinct venous stasis is common. The papilla usually appears normal, but in some cases (as also emphasized by Jaeger and Koenigstein) is from a dark gray to a bluish gray. The usually coexisting reddish discoloration at the scleral edge with fine, red, radiary hemorrhagic lines, permits the interpretation of the darker and grayish hue of the papilla as due to a hemorrhage into the subvagal space of the dural sheath. Within a few days the blood is resorbed and the color becomes normal.

Most striking in the ophthalmoscopic examination are the relatively common hemorrhages in various portions of the eye. The marked difference in the percentage figures of their frequency as given by the various authors, probably is due to the difference in the time when the examinations were made. Smaller hemorrhages are speedily resorbed and quickly become invisible. Sicherer's figures are based on observations made within twenty-four hours after birth.



In 400 newborn infants he discovered 65 hemorrhages, of which 23 were bilateral, 14 in the right, and 5 in the left eye.

All investigators agree that these hemorrhages are fresh and could not possibly have developed during intrauterine life. Sicherer agrees with Schleich that the hemorrhages from the finer retinal vessels are the result of a congestion, and that, therefore, etiologically they stand in some relation to the minute and larger cerebral hemorrhages.

The suggestion has been made that the congestion in the eye develops only secondarily to an intracranial hypertension. Compression of the sinus cavernosus interferes with the normal emptying of the ophthalmic vein in spite of the partial relief offered through the existing anastomoses with veins of the face. More unfavorable under such conditions, however, would prove the situation for the vena centralis retinae, which, as a rule, without any anastomosis with the vena ophthalmica, empties directly into the sinus cavernosus. The more common occurrence of such hemorrhages in the eyes of premature infants is generally ascribed to the particular fragility of the retinal vessels as the result of underdevelopment.

A further support of Schleich's theory that the congestion is the effect of the compression of the cavernous sinus is seen by Sicherer in the fact that in the more common left anterior occipital presentation the hemorrhage is more likely to occur in the right eye, or if bilateral, is more pronounced in the right eye, just the opposite findings being made in the right anterior presentation. This would seem to prove that the eye lesion is more frequently found on the side on which the sinus is exposed to greater pressure.

Most of the writers agree that small peripheral hemorrhages are of no importance for vision later in life, while large hemorrhages, especially when situated near the macula, may permanently affect central vision. Such hemorrhages may cause permanent structural changes or interfere with further normal structural development.

It seems interesting to note in this connection that in some of the reports concerning the eye findings mention is made of a complicating melena which is supposed to have been caused by the intracranial hypertension. There still are investigations wanting concerning a possible etiologic relation also of the ocular hemorrhages to a hemorrhagic diathesis.

Paul gives the following figures as expressing the relation of retinal hemorrhage to certain obstetrical anomalies: Narrow pelvis,

50 per cent; prematurity, 40 per cent; complicated and protracted labors, 40 per cent; normal labors with child of normal size, 20 per cent. He lays particular stress on asphyxiation as the cause of the ocular hemorrhage.

In the opinion of Kearney the ophthalmoscopic study of the eyeground of the newborn offers one of the earliest and most reliable means of diagnosing intracranial hypertension. He described the characteristic ocular findings as follows: "There may be seen a mild edematous blurring of the upper and lower margins in the first few days of life, later an edematous blurring of the entire surface of the nerve head and all its margins. When the intracranial hemorrhage is severe and the tension greatly increased, gross edematous changes may appear quite early, the edema being confined to the nasal half of the disc, or affecting the entire disc, a typical papilledema."

In Kearney's belief such eyeground findings together with an increased pressure of the cerebrospinal fluid, as ascertained by lumbar puncture, in selected cases permit the definite diagnosis of a pathologic intracranial hypertension and justify a simple decompression operation even in the absence of convulsions.

**SPINAL PUNCTURE.**—Cerebrospinal fluid is withdrawn by means of a spinal puncture in the main for two purposes: (1) to obtain fluid for morphological, cultural or chemical study, and (2) to relieve an abnormal tension. Spinal puncture serves the double purpose of diagnosis and therapy also in its employment on the newborn for which of late it has been recommended by serious workers and not mere enthusiasts "for every case of suspected intracranial hemorrhage."

Obviously here we are concerned only with spinal puncture as an aid in diagnosis.

Blood reaches the spinal canal directly in all infratentorial and peribulbar hemorrhages and indirectly, by gradual diffusion, from supratentorial basal, and even from hemispheric, hemorrhages if they are copious.

Most commonly the puncture is done in the lumbar section where an accidental injury of the cord by the needle is the least likely to occur. A fluid strongly mixed with red cells or representing almost clear blood will be obtained especially in the cases of free subtentorial hemorrhages but, as must be kept in mind, also in epidural spinal hemorrhages, i.e., in cases of traumatization of the spinal column during birth. If the fluid is only of a yellowish color, presum-

ably containing only a small amount of blood, it must be examined microscopically without delay. Morphologically altered, degenerated erythrocytes, detritus, hematin pigment, etc., prove an older hemorrhage, while perfectly normal red cells in small numbers do not exclude the possibility of an accidental contamination of the spinal fluid with blood from injuries of small vessels by the puncture needle. It is obvious that the absence of blood in the withdrawn spinal fluid does not necessarily exclude a supratentorial and especially hemispheric hematoma.

Statements concerning the diagnostic value of the pressure of the spinal fluid, estimated by the speed of the escaping fluid or actually measured with a manometer, are most contradictory. Most writers are inclined to deny any reliable diagnostic significance to a seemingly increased pressure.

A perusal of the numerous detailed case reports in literature, however, leaves the definite impression that withdrawal of spinal fluid or blood (quantities of 20 to 30 cubic centimeters are repeatedly mentioned) in a strikingly large number at least of the recorded cases, is followed by a prompt temporary or permanent cessation of those symptoms which commonly are ascribed to cerebral hypertension or localized cortical pressure.

Impressed by the great diagnostic value of spinal puncture, in spite of its evident short-comings and dangers, some authors, e.g., Henschen, have advocated making the puncture as near as possible to the primary source of the hemorrhage. A puncture between the second and third cervical vertebra thus will promptly establish the existence of a peribulbar extravasation of blood in its incipient stage. The increased danger of a cervical puncture, in the belief of its various enthusiastic advocates, is more than outweighed by its beneficial effect in relieving the local pressure on the important centers in the medulla oblongata which necessarily will prove fatal if permitted to continue or to become excessive.

Henschen, Doazen, Frazier, Robt. M. Green, go a step further and practice in selected cases an "easy and harmless" cranial puncture which fails to yield the desired result only if the blood had time to clot firmly. However, most authorities agree that blood effused in intracranial hemorrhages as a rule clots very slowly and often is found still in a fluid state even at post mortem. In the special method of cranial puncture described by Henschen, a comparatively thick puncture needle is pushed, on one or if necessary on both sides, into

the subdural space from the lateral corner of the large fontanel. This puncture can also be made under the same precautions from the posterior fontanel.

Some of the writers, e.g., Ball, Doazen, etc., from this point of view, consider it logical and practical to search for the blood clot through a small trephine opening whenever puncturing with the needle does not supply the desired information.

Without sufficient personal experience one cannot undertake to pass judgment on such procedures as cervical or cranial puncture. It does not seem justifiable to recommend even the decidedly less dangerous lumbar puncture as a routine procedure to the practitioner for all suspected cases of hemorrhage. Nevertheless the fact deserves serious consideration and investigation, that diagnostic spinal punctures are employed and urgently recommended by such recognized authorities as Cushing, Frazier, Doazen, Henschen and many others. Robt. M. Green, Foote, Brady, Sidbury, Hutinel and several other careful investigators feel no doubt that "in every case of suspected hemorrhage" (Sidbury), "in every newborn showing within twelve to twenty-four hours after birth respiratory distress and cyanosis, with or without muscular rigidity and twitching" (Foote), "in every baby that within a few days of birth becomes pale, refuses the breast and shows a peculiar though slight edema of the face" (R. M. Green), a lumbar puncture should be done promptly. According to Sidbury such a routine will help in three ways: (1) letting off of the spinal fluid will relieve the intracranial pressure and stop the convulsions; (2) it may cure the patient; and (3) it will aid in the diagnosis.

However, it must be remembered that the relief of the intracerebral pressure may foster a continuation of the hemorrhage, especially in the cases of a decreased coagulability of the blood. Therefore, it seems desirable, in my opinion, to combine a routine of spinal puncture in suspected cases with a routine of ascertaining the blood clotting time, and to precede the puncture with blood or serum injections, whenever there seems to be a delay in clotting.

The chief value of the puncture as a diagnostic aid undeniably lies in the readiness with which it can be applied, and in the promptness with which in some cases it will definitely establish an existing intracranial hemorrhage. Only little time, as a rule, is available for therapeutic and especially operative interference to prove useful or life saving.

We shall presently see that spinal puncture also is a procedure of no mean importance for the purpose of differential diagnosis from conditions closely resembling intracranial parturitional traumatism.

**Differential Diagnosis.**—The difficulties of early and accurate diagnosis of a traumatic intracranial hemorrhage at birth are augmented to a notable degree by the evident fact that the general clinical picture of such an accident or at least some of the outstanding symptoms of such lesions are closely simulated by certain other conditions not uncommonly affecting the newborn infant. This applies particularly to convulsions.

In an exhaustive study of this question, Esch clearly points out the essential differences of the convulsions of the first few days of life from those appearing later. Early convulsions are not rare. D'Espine found in a total of slightly over 15,000 newborns in a large maternity service the incidence of early convulsions to be approximately two in a thousand, with a mortality of 62.5 per cent. A large part of these cases represents instances merely of a cortical or sub-cortical irritation as the immediate result of a birth trauma, while in many other cases definite cerebral lesions were discovered to account for the convulsions. Esch, therefore, concludes that it is advisable to differentiate, as far as possible, the general convulsions or limited spasms of certain muscle groups of the newborn into a functional and an organic group.

**FUNCTIONAL CONVULSIONS.**—*Myotonia neonatorum* (of Hochsinger).—This condition manifests itself in a slight rigidity of the flexors of all extremities at birth and persists for a short time even in perfectly normal infants. This myotonia may increase the tendency to tonic spasms but never becomes their actual cause.

**Reflex Irritability of the Brain.**—This is often increased at birth for a short time and may actually become responsible for convulsions. Despite the comparatively incomplete development and function of the central nervous system of the newborn, the reflex mechanism obviously is functioning. In no other manner could be explained the clearly reflectory manifestations of crying or sneezing.

**Eclampsia Neonatorum.**—This is a term often rather loosely employed, especially by pediatricians, to denote a convulsion of a newborn infant. The obstetrician more commonly applies the term solely to the convulsions of infants born of eclamptic mothers. Presumably in these cases certain toxic substances, prone to cause convulsions, had been transmitted by way of placental circulation from the ma-

ternal into the fetal blood. Though this assumption is not based on any definitely known biologic facts, such a transition of soluble toxins, theoretically at least, seems possible. In the light of recent knowledge, however, it seems more plausible that in many of these cases the convulsions are due to a cranial trauma incident to a labor which, in view of the condition of the mother, often is terminated by means of a difficult forceps or a version with immediate extraction.

*Tetany.*—In the opinion of Reuss, tetany should be specially grouped within the wider term of spasmophilic diathesis or tendency. He claims that typical tetany with its characteristic diagnostic symptomatology, including laryngospasm, has never been observed in the newborn. The precise diagnosis of true tetany within the first few weeks of life, however, really is impossible because the necessary proof of electric hypersusceptibility cannot be furnished. In the newborn the electric sensitiveness of all peripheral nerves is low.

Typical tetanoid symptoms, closely resembling the classical trias of Erb's, Trousseau's and Chvostek's phenomena, but never associated with laryngospasm, as pointed out by Reuss, have been observed by Kehrer (*Arch. f. Gynäkologie*, 1913, 90:372). Reuss saw a very similar symptom-complex in a case of subdural hemorrhage. How far such tetanoid symptoms in the newborn might be related to a traumatic birth injury of the parathyroid glands is a question I shall discuss later in its proper connection.

ORGANIC CONVULSIONS.—In the majority of instances general convulsions or spasms limited to isolated muscle groups are due to organic lesions. Among them the traumatic intracranial lesions, considered in detail in the preceding pages, stand in the foreground. For practical and especially therapeutic purposes it becomes indispensable to differentiate them diagnostically from certain other chiefly infectious processes which affect the child late in intra-uterine life or soon after birth and tend to cause spastic seizures.

Before entering into a detailed discussion of the differential diagnosis from this last group, it will prove advantageous to point out briefly a few essential facts. In marked contrast to the functional group, the cases in which convulsions or circumscribed spasms are caused by definite organic lesions are likely to exhibit one or more of the following symptoms: severe external injuries, slight stupor or coma between seizures, rigidity of neck, anomalies of fontanels or sutures, deformities of the head (hydrocephalus), a unilaterality

of symptoms, or a progression of symptoms which in their sequence express the anatomic extension of an enlarging hematoma.

In the differentiation between the traumatic lesions, chiefly hemorrhages, and the other processes, chiefly infections, a consideration of the mother's condition and of the history of labor is of a decidedly limited value. Fever in the newborn is practically always absent in the earlier stages of traumatic lesions. The most useful aid in the differential diagnosis is spinal and cranial puncture.

*Tetanus.*—According to a report of Mirons (published in 1904) in Roumania out of a total of 23,000 infants dying within the first month of life 10,000, almost one-half, had succumbed to tetanus. In some of the tropical countries the infant mortality from tetanus alone has been found to be between 10 and 25 per cent. It, therefore, cannot be surprising to meet in medical literature with the term "tetanus neonatorum," but obviously it should be, and probably is not, strictly limited to denote the specific infection with tetanus. This disease now is but rarely observed in the newborn in civilized countries, and hardly ever seen in a maternity since the modern technic of the aseptic management of the umbilical cord stump has been generally adopted. This very scarcity of tetanus renders the problem of a differential diagnosis proportionately more important.

The period of incubation in tetanus presumably varies from a day or two up to several weeks. The first symptoms, therefore, are not likely to manifest themselves within the first few days of life, and most commonly have been recorded as appearing at the end of the first or during the second week of life. However, Heubner claims to have seen a true case of tetanus develop on the day of birth.

The first symptom is a tonic contraction of the masseter muscles. The spasms then extend to other muscles of the face and neck, and later of extremities and trunk. They are of a markedly tonic type and cause an almost characteristic rigidity of the entire body. The temperature may remain normal but usually there is a high fever. Death may ensue quickly or is delayed for a day or two. Mortality figures vary widely, and are given by Fronz as 42 per cent, by Shukowski as 98 per cent (quoted from Reuss). In some rare cases the infection is of a chronic type and the child then is more likely to recover.

Convulsions of a pronouncedly tonic type are comparatively less frequent in intracerebral birth hemorrhage, though Waldstein,

Abels, and others emphasize a trismus as rarely absent in ventricular hemorrhages. Seitz points out the fact, important in differential diagnosis, that, with the exception of the musculus orbicularis oculi, eye muscles do not participate in tetanic convulsions. But Abels observes that unfortunately the contractions of the orbicularis actually prevent a satisfactory examination of the behavior of the other eye muscles during the seizure.

A careful and critical analysis leaves no doubt that neither the presence nor the absence of the various clinical phenomena, enumerated above, either singly or in groups, will allow a definite differentiation of tetanus from a parturitional cerebral lesion in all cases.

There are only a very few cases on record in which the tetanus bacillus actually has been discovered in the newborn. If we consider the general uncertainty of the exact diagnosis of tetanus in the light of the fact that our more accurate information concerning the frequency and semeiology of cerebral birth lesions is of comparatively recent date, the deduction is permissible that even today probably in some cases the diagnosis of tetanus is made incorrectly and thus the infant deprived of proper therapeutic attention. Modern literature contains the records of several cases in which infantile death was ascribed to tetanus while the post mortem examination revealed as its actual cause a cerebral hemorrhage. Therefore, the demand can be made to attempt a differentiation in every suspected case of tetanus by means of a spinal puncture. If red blood cells in sufficient quantity or in characteristically altered forms are found in the spinal fluid, tetanus can be excluded promptly.

*Encephalitis.*—Comparatively little is known concerning a septic encephalitis which occasionally is found in post mortem examinations of newborn infants. In some instances the process evidently is of metastatic origin, in the majority of cases, however, the etiology remains obscure. Mention has been made of the possible connection of septic encephalitis and porencephaly to cerebral hemorrhages, and to the formation of ischemic foci as the direct result of birth traumatism. Kruska in 26 cases of ischemic brain lesions found that in three the resulting necrotic area had become infected and thus been transformed into a typical brain abscess.

*Meningitis.*—Certain congenital deformities especially of the cortex and congenital hydrocephalus, in the belief of various writers, prove the possibility of an intra-uterine meningitis. Seitz (*Arch. f. Gynä-*



kologie, 1907, 83:701) advanced suggestive reasons why there might exist a connection between severe traumatism of the abdominal wall during pregnancy and intracranial lesions with subsequent congenital encephalitis.

In regard to hemorrhagic pachymeningitis the opinion seems to prevail that it is rather of hereditosyphilitic origin.

A purulent meningitis not rarely is observed very early in life and even in the newborn. In most instances it is due to an otitis, which in the opinion of Aschoff, is caused by the entrance of infected amniotic fluid into the middle ear. A long-drawn-out labor, especially after the membranes have ruptured, and asphyxiation, favoring the aspiration of presumably infected amniotic fluid, thus possibly may play a rôle in the causation of purulent meningitis of the newborn.

In regard to the problem of differentiation of meningitis from meningeal hemorrhages, the following points are of importance: meningitis, as a rule, is accompanied by fever which, however, may be absent in fulminant cases ending fatally very quickly, or may not appear until the terminal stage. On the other hand, some cases of meningitis in the newborn exhibit such characteristic meningeal symptoms as convulsions, rigidity of neck, protrusion of the fontanel or distention of sutures, or almost typical tetanic symptoms (De Bruin).

Again the question suggests itself how often the diagnosis meningitis is made incorrectly. In the doubtful case a spinal puncture might solve the problem, as is well illustrated in a case mentioned by Bonhoff and Esch: A newborn baby exhibited the typical symptoms of a supratentorial hemorrhage. On the sixth day a spinal puncture was made. The spinal fluid was found turbid, containing many polynuclear leucocytes. On the ninth day convulsions, fever, with exitus five days later. Autopsy showed a purulent meningitis from a right otitis media.

However, as pointed out by Brady, blood in the spinal fluid in such instances does not necessarily prove an intracranial hemorrhage because the fluid will be bloody also in the rare instances of hemorrhagic spinal meningitis.

*Acute Congenital Hydrocephalus.*—In Seitz's opinion a serous ventricular meningitis may offer unsurmountable difficulties in the differential diagnosis from ventricular hemorrhage, though, in the case of a congenital hydrocephalus, the protrusion of the fontanels and distention

of sutures is likely to be excessive. In such a case a spinal puncture may well settle the diagnosis.

...**Prognosis**—**PROGNOSIS OF LIFE.**—The immediate effect of a traumatic intracranial birth lesion is dependent upon the extent and the location of the resulting hemorrhage. Traumatic lesions not associated with hemorrhage, e.g., many of the tentorial lacerations, apparently leave the infant unharmed. The fact also is well established that small hemorrhages, like a cephalhematoma internum or especially the small subarachnoidal hematomas, as a rule, fail to cause any noticeable symptoms. It is, on the other hand, known that a newborn may reveal signs strongly suggesting intracranial traumatization which, however, are so indefinite or disappear so quickly that it is impossible to determine whether they are due to small hemorrhages or, as suggested by Seitz, to a mere "concussion of the brain" or a fleeting edema. As a matter of fact they do not clearly indicate any immediate harm done to the infant. Whether such slight, and at first practically symptomless, intracranial lesions may be responsible for deficiencies in physical or mental development later in life, is a question discussed later in this chapter.

Whenever the effusion of blood is excessive and the clot interferes with the function of certain vital centers, especially those in the medulla oblongata, death ensues either immediately, during, or within a few days after birth. The pathology and etiology of these hemorrhages have been described sufficiently to make it obvious that in many of the cases the child may appear perfectly normal at birth and only a few days later begin to exhibit the signs of a hemorrhage which gradually or suddenly leads to a fatal issue. It is this group of cases in particular in which early diagnosis and prompt therapeutic interference will greatly enhance the prognosis both in regard to life and restitution of perfect health.

Smaller intracephalic hematomas are known to have indirectly caused a baby's death through their infection or by favoring a pneumonia as a result of impeded respiratory function.

The prognosis always is decidedly unfavorable in cases in which intracranial hypertension has led to convulsions, especially if these follow each other in short intervals. The change from the convulsive to the paralytic state practically indicates the approaching end.

Of greater practical importance and interest in the problem of prognosis are the possible delayed consequences of a traumatization of the brain in parturition.

PROGNOSIS OF HEALTH.—Up to a comparatively recent date obstetricians have shown but little interest or belief in the claim of neurologists that traumatization and asphyxiation of the infant at birth are responsible for certain physical and mental anomalies which manifest themselves only in the later life of the child. These views of the neurologists of late, however, have prompted obstetricians to advocate operative termination of labors for the purpose of avoiding such untoward sequelae. There cannot be any doubt that in the light of more recent information concerning the causation of intracranial parturitional lesions the teachings both of the neurologist and the obstetrician call for thorough revision.

In 1843, Little, in his first paper dealing with cerebral spastic paralysis of children, asserted that the condition is due to a lack of development of cerebral tissues and also to meningitis. Only incidentally he mentioned in this connection that this abnormal condition seems to follow difficult and prolonged labor terminated with or without the use of instruments, and, in his opinion, under these circumstances probably was due to an intracranial hemorrhage. In a second paper, published in 1862, he expressed it as his belief that cerebral spastic paralysis of children in three-fourths of all instances was caused by an intracephalic birth hemorrhage.

Authors quoting Little are in the habit of adding that Sarah McNutt (1885) and Kundrat (1890) by certain findings at autopsies have definitely established this relation of palsies of central origin to such intracranial birth hemorrhages.

Congenital spastic paralysis in children quite frequently is found associated with mental defects. It is unnecessary for our purpose to show here by further quotations from literature how this fact gradually led writers to the deduction, now widely accepted, that asphyxiation, forceps extractions, or protracted labor play a most important rôle in the etiology of imbecility, idiocy and other mental deficiencies, of epilepsy, hydrocephalus, strabismus, deafness, speech defects, cortical aphasia (Ziehen) and so on.

What are the chief arguments commonly given in support of such an assumption?

*Anatomic Findings.*—The still prevailing custom of writers to mention McNutt and Kundrat in connection with this particular aspect of the sequelae of parturitional injuries is not justified. That intracranial hemorrhages of the newborn may cause spastic conditions and convulsions and finally death, which represents the problem

investigated by them, is an acknowledged fact. We are here, however, concerned only with those children who have survived the assumed brain injury. Anatomic findings to be acceptable as proofs for the contention that birth trauma bears a close etiologic relation to various deficiencies of the older child, therefore, necessarily would have to comply with the following essential requirements: If discovered at the post-mortem examination of an older child, their character or location must leave no doubt that they are due to a trauma sustained at birth. Of the known types of traumatic lesions of the newborn's brain and meninges only those can be taken into consideration which do not cause death, i.e., those which in routine necropsies of all newborn babies represent solely accidental findings, not directly responsible for the infant's death.

Pachymeningitis hemorrhagica has been ascribed especially by older writers (Doehle, Weyhe), to a birth trauma and has been looked upon as the anatomic lesion which accounts for nervous and mental defects in some cases.

Kowitz recently has published the results of his exhaustive studies concerning the relation of intracranial hemorrhages to pachymeningitis chronica interna. From the findings in 6000 autopsies, performed on children up to the age of two years, he concluded that subdural birth hemorrhages in a considerable number of cases give rise to a hemorrhagic pachymeningitis which is found in 3.9 per cent of all children dying between the age of eight days to two years. It seemed to him that all children thus affected gradually succumb in early childhood partly as the direct result of meningitis, partly from intercurrent diseases to which they apparently are rendered less resistant by the meningitis. He became convinced that a hemorrhagic pachymeningitis which manifests itself only later in life has no causal connection to this traumatic type seen in the newborn.

Finkelstein, Reuss, and various others of modern pediatricians apparently are of the opinion that the hemorrhagic type of meningitis is seen practically only in hereditosyphilitic infants.

Anatomic findings have been recorded which are supposed to prove a direct connection between birth trauma and internal hydrocephalus. Two cases of Fischer may be briefly cited as illustrations: (1) A child delivered by means of an abdominal cesarean section showed unmistakable signs of a brain injury within twenty-four hours. Gradually a hydrocephalus developed. Death at age of

four months. At post mortem evidence of an old hemorrhage was discovered in the middle and posterior fossa. Fischer thinks that pressure exerted by the hematoma interfered with the escape of the liquor and also caused a venous stasis which led to the development of the hydrocephalus. (2) A child born under twilight sleep appeared perfectly normal at birth. Hydrocephalus became noticeable about three weeks later, followed in another week by exitus. A very large hematoma was found. Its size excluded any possibility that it could have existed at birth without causing any symptoms. The hemorrhage must have started later and progressed slowly. Also in this case Fischer felt quite certain that the hydrocephalus developed secondarily to the hemorrhage.

The ischemic foci described by Kruska may be regarded as suggestive, but not by any means conclusive, anatomic findings in support of the assumption that certain birth lesions stand in an etiologic relation to later physical and mental deficiency at least in some cases.

Plausible and entirely acceptable seems the assumption of Kahl-den, Richter, Monakow and others, that porencephaly may be the sequela of a parturitional cerebral hematoma. The ischemic areas with subsequent necrosis, described by Kruska, may then be looked upon as the more definite anatomic findings in support of such a theory.

From a careful perusal of literature the definite deduction can be drawn that convincing anatomic evidence for an important or frequent causative connection of intracranial birth lesions to mental deficiency still is wanting.

*History of Difficult Labor.*—The term "difficult labor," especially in the writings of neurologists, is rather loosely applied to labors terminated artificially, to abnormal labors, especially if the child was born asphyxiated, and again to merely long labors. It is necessary to emphasize that the diagnosis "difficult labor" practically without exception in this particular literature is based solely on the assertion of the mother that her labor was difficult. This holds true for all the statistical investigations made in asylums for the insane or feebleminded which form the main basis for the view now rather prevalent that difficult labors are of considerable importance in the causation of mental deficiency. These statistics seem to prove also that a long labor is more prone to interfere with mental development later in life than a forceps extraction. It does not seem to have

occurred either to these investigators, or the obstetricians who willingly accepted the deductions drawn from these statistical figures, that an investigation carried out in this manner implies at least two great errors which practically nullify its scientific value: (1) It is claimed on theoretical grounds that the fetal brain suffers from the prolonged compression during a protracted second stage of labor. The mere claim of a mother that her labor was long may convince a neurologist, but should not prove to an obstetrician that the fetal head was exposed to undue compression. As a matter of fact, in primigravidae labor most often is abnormally prolonged only by a slow first stage. (2) A study of this problem limited to children found in asylums obviously excludes all stillborn infants and those who died early in life. It is evident that this prevents the inclusion into calculation of a considerable number of children extracted by means of forceps. It seems fair to assume that these two great deficiencies of these statistical investigations account for the fact that they seemingly prove "difficult" labor more harmful than forceps for the later physical and mental development of the child.

The often quoted paper of Arthur Stein and its discussion before the New York Academy of Medicine express well the present situation. The obstetrician rather naïvely has accepted the verdict of the neurologist, based on superficial and truly unscientific statistical investigations, that a long-continued compression of the head must be avoided by prompt application of the forceps. The existing situation is well illustrated when we find in DeLee's splendid book the following quotations: Beach found in 810 idiots a history of hard forceps in 4 per cent, and in 26.6 per cent of spontaneous but difficult labor. Porter states that 17 per cent of the epileptics in the Indiana School for the Feebleminded had a history of difficult labor.

A more promising route of approach for the possible solution of this important question from the standpoint of the obstetrician, will be discussed later in this chapter, but in this connection emphasis will be laid only on the fact that there is no acceptable proof extant for the assertion that forceps extractions are less dangerous to the infant than a long labor. Present information concerning the mechanical factors involved in the causation of intracranial birth lesions leaves no doubt that traumatic injuries are most prone to occur when the compression of the head is extreme, asymmetric, and most of all, if it is accomplished quickly. Any of these conditions is more

likely to prevail if the passage of the head is hastened by means of oxytocics, manipulations or instruments.

Obstetricians who advocate a more liberal use of the forceps in the interest of the child also neglect to take into account the very important fact that the neurologic investigations, by which they are so greatly influenced, were made at a time when difficult and instrumental labors still rather generally were considered the most indispensable causative elements in all intracranial injuries at birth. It seems inconsistent to advocate hastening of the expulsion of the child in face of the established fact that in from 20 to 25 per cent of cases in which an intracephalic traumatic birth lesion of some kind is discovered at autopsy the exact obstetric history shows a spontaneous, often quick, but otherwise normal labor.

*Primiparity.*—References to a striking frequency of firstborn among the inmates of institutions for the insane, epileptics, or feeble-minded are commonly cited in literature, and incidentally it may be mentioned that some of these date back to the middle of the past century. The high figure for primogenity among the inmates of these institutions was, and still is, considered a valid proof for the etiologic significance of a first, which means, of a difficult, labor for nervous and mental deficiency. If we once more recall the fact that complications of labor have lost much of their former dignity in our conception of the origin of intracranial lesions, primiparity obviously is deprived proportionately of its value as argument in this discussion. It, furthermore, is incorrect to draw any conclusions from the number of firstborn among defective children without including into the calculation of such a percentage figure also the percentage of firstborn among children of this particular age. So far as one can see this has not been done by any of the investigators though it seems essential for any statistical calculation worth serious consideration.

*Clinical Observations.*—It would be futile to quote here various writers, chiefly neurologists, who simply express it as their personal belief that parturitional traumatization in many instances is the sole cause of retarded physical or mental development. Without any difficulty, as many authors could be cited who hold opposite views.

At times such opinions are based, at least partly, on certain clinical observations. M. Allen Starr states that post-mortem examinations of idiots reveal signs of an old hemorrhage in 20 per cent of the cases, and concludes that obstetricians are not to any noteworthy extent

responsible for idiocies and palsies. In a study of epilepsy later in life in relation to convulsions of infancy Morse finds that epilepsy is far more likely to develop when the cause of the infantile spasms seemed to have been a brain injury than when they more probably were due to disturbances in the gastrointestinal tract. If the small infant has but one or even repeated convulsions, it is practically impossible to determine whether they are epileptic or not. Most authors agree that early epilepsy probable is extremely rare. Emmet Holt and also Koplik emphasize the fact that the overwhelming majority of children who have convulsions during infancy fail to develop epilepsy.

The connection of a certain anomaly of development with the normal or abnormal trauma of birth can be definitely established only in two ways: (1) by directly linking up the later defect with a definite intracranial lesion observed or accurately recorded at birth and (2) by following up the life histories of a large number of children whose birth histories are known in every detail. This latter method undeniably is the more reliable one and actually has been followed by several obstetricians on a large material. Investigations so far made along these two lines have furnished much valuable information.

Volland studied the histories of entire families and thus seemed to have been able to demonstrate a probable injurious influence of a birth trauma in some instances. In the same families normally born children remained well, whereas some of those born after abnormal labors became epileptic.

A possible relation of birth traumatism to the development of hydrocephalus is at least suggested by a recent observation of Oden. A mother of four children has a narrow pelvis. Both parents are free of all stigmata of disease. First child, born prematurely, is healthy; the second, born at full term by instrumental delivery, developed a hydrocephalus; third, born at full term after difficult labor, died with marked cephalic enlargement; fourth, delivered at full term with cesarean section, is well at age of four months.

Koenig acknowledges that in 25.7 per cent of the cerebral palsy cases seen by him, difficult labors or asphyxia at birth are recorded in the history, but that in all cases, with the exception of only 2, many other predisposing factors, such as syphilis, bad heredity, alcoholism of parent, etc., were discovered. Of these two exceptions one was a hydrocephalic, the other a microcephalic child.



Vogt in an analysis of the probably causative factors of epilepsy found that in the large majority of cases the history reveals a decidedly more plausible explanation than asphyxiation at birth. Out of a total of 471 cases of congenital or early cerebral defects only in 7 an injurious parturitional factor, by exclusion of all other so far known causes, remained as the only possible etiological factor to which the deficiency could be charged.

Zappert (in Pfaundler-Schlossmann's *Handbuch der Kinderheilkunde*) states that but four conditions can be justifiably brought into any relation to intermeningeal hemorrhages:

1. General rigidity with only slight or without dementia, usually also without convulsions (Little's symptom-complex).
2. Paraplegic rigidity without dementia and without convulsions.
3. Simple hemiplegia with feeble-mindedness and with convulsions.
4. Bilateral hemiplegia with feeble-mindedness and with convulsions developing into a pseudobulbar paralysis.

Group one and two presume bilateral lesions. One could think in connection with them of the more diffuse subarachnoidal hemorrhages which in the newborn may not cause any pronounced symptoms and still may later interfere with the normal development of the cortical motor and psychic centers. In the etiology of these two conditions also small hemorrhages within the cortex itself or severe contusions may be of importance.

In the etiology of groups three and four intermeningeal subdural hemorrhages undeniably play a definite rôle.

It must, however, be pointed out in this connection that tissue defects in the brain of the newborn may occur independent from any parturitional trauma. Thus Osler found a cavity in the brain of a fetus removed from the uterus of a mother who died from typhoid in the sixth month of pregnancy.

There are several cases on record in which an injury of the fetal brain apparently was solely due to a severe traumatism of the pregnant uterus. Cotard, e.g., recorded the following case: A pregnant woman sustained a severe blow against her abdomen. The child born three months later held all four extremities in forced flexion. Post-mortem examination revealed a necrotic defect in the brain. Reuss mentions similar observations of Seitz and Zappert.

Summarizing all this information we can say that in some, though rare, instances a physical or mental deficiency manifesting

itself only later in life can be directly ascribed to a definite cerebral lesion noticed at birth. But even this fact does not offer us any clue concerning the assumed relation of slight and symptomless intracranial birth lesions to later defects. That such slight lesions do occur probably in many cases cannot be denied. Do they exhibit delayed sequelae? It became apparent to Hannes among the first that an answer to this question could be supplied by the obstetrician who, in possession of a detailed record of all phases of the birth and of the immediate condition of the newborn, will study the physical and mental condition of a large series of these children in afterlife.

In consideration of the more general claim that asphyxiation and instrumental delivery are the prominent factors in the interference with normal development of the child, Hannes selected from his birth records two groups of 150 cases each, the one representing infants asphyxiated at birth, including a considerable number of deeply asphyxiated, resuscitated by swinging after the Schultze method, the other group comprising children born by means of forceps, after versions, extractions, in breech presentation, etc. Both for the purpose of control, and also in view of the established fact that intracranial injuries occur also in the course of normal labors, Hannes added a third group of 150 perfectly normal, spontaneous labors. This study, most carefully carried out, revealed that each group showed almost the identical number of apparently abnormal, backward and actually defective children. In the total number there were only 2 manifestly idiotic children, 1 in the group of abnormal labors, 1 in the normal labors, and none among the asphyxiated. He discovered one child suffering from a spastic palsy, in the opinion of an expert neurologist, due to porencephaly, which might have been the result of a birth traumatism. This child was born after version and extraction.

Following the example of Hannes identical investigations were made by Kwozek, who concluded that his most careful examinations of the physical and mental condition of children born asphyxiated, or born either in instrumental labors, or spontaneously after difficult labors have failed to reveal any difference from the children born after perfectly normal labors. In the very few instances of manifest deficiency, in every case without any exception, other conditions, generally recognized as etiological factors for deficiency, were easily revealed. The same deductions were drawn by Recht-

schaft from investigations made along the same lines. Hannes quite recently collected and analyzed all the material so far studied in the manner first suggested by him and presents the following instructive data: Group A, asphyxiated at birth, 157 children examined, 9 of them abnormal (5.7 per cent), in 3 distinct other hereditary factors, which leaves 6 (3.8 per cent) in whom asphyxiation might have to account for the later deficiency. Group B, artificially delivered but not asphyxiated, 242 children examined, 6 of them abnormal (including 1 case of brachial palsy but mentally normal); in 3 distinct hereditary factors, which leaves 3 (1.2 per cent) in whom the abnormal delivery may account for mental and physical defect. Group C, normal labor, fresh at birth, 206 children examined, 10 of them (4.9 per cent) abnormal; in 3 distinct hereditary factors, which leaves 7 (3.4 per cent) in whom the birth trauma of a perfectly normal labor in the absence of any symptoms might have to account for the deficiency. If Groups A and B are put together, which corresponds more closely to the classification of neurologists of "difficult labor," we have 399 reexamined children with 3.7 per cent deficiency, or with exclusion of those exhibiting recognized hereditary factors, 2.2 per cent in whom a birth trauma could be held responsible. In these combined groups were found 3 epileptics, 1 with marked heredity, that is, only 2 possibly injured during birth.

Hannes feels that all newer investigations only confirm the position he held ten years ago, that asphyxiation, abnormal labor or instrumental delivery do not differ from normal labor as a possible cause for abnormal mental development.

Researches have been made by obstetricians concerning the later effects, particularly of forceps extractions. Gans presents the following figures: out of a total of 180 forceps children, either examined by himself later in life or concerning whom reliable information had been obtained indirectly, there were discovered 2 backward children, and 9 with at least a history of convulsions of some sort. In the 1 backward child (five years old) the forceps extraction was found recorded as very easy, not followed by any symptoms suggesting an intracranial injury. In the other backward child (nine years old) the forceps extraction was not difficult. Of the 9 children with convulsions, 5 had died in convulsions of not determined character. Of the 4 surviving children (ages between four and nine

years) only 1 was a typical epileptic. All 9 were delivered by means of easy outlet forceps without any noticeable immediate injuries.

This last fact deserves strong emphasis in view of the common recommendation of the low forceps in the very interest of the child.

Out of a total of 448 forceps deliveries made between 1900 and 1914, Engelken was able in 1920 to obtain accurate information directly or indirectly concerning 232 children. Among them were 2 suffering from epilepsy, 2 from chorea, 1 idiot, 2 imbeciles, 4 feeble-minded and 2 who died in convulsions of some kind. In this entire group other factors than a presumable parturitional injury were manifestly responsible for the mental anomalies, with the possible exception of 1 of the epileptics and 1 of the imbeciles.

A paper of Wulff, published in 1892, still is often quoted by modern writers. He claimed that of children born asphyxiated, or after difficult labors only one-sixth begin to walk and only one-ninth begin to talk at the proper time. In order to test this striking claim, Hannes in his investigations made specific inquiries in regard to these two points. He found that of such children 52.6 per cent walked at one year, that 42.6 per cent began to talk in the first year and 50 per cent in the second year. Thus no noteworthy deviation from normal conditions could be ascertained.

Analyzing critically this somewhat contradictory information furnished on the one hand chiefly by neurologists, on the other, by obstetricians, concerning an etiologic relation of birth trauma to physical, nervous, and mental defects of later childhood, the following facts become apparent:

(1) The deductions of neurologists rather uniformly are based on investigations which from the viewpoint of reliability and scientific value are decidedly objectionable. (2) Neurologists have practically limited their researches to attempts of linking up the evident deficiency with some abnormality of labor. (3) Obstetricians, in contradistinction, endeavored to reveal later sequelae of the trauma both of normal and of abnormal labor and delivery. (4) Investigations more recently carried out by obstetricians tend to weaken to a considerable degree the older opinion of neurologists that asphyxiation and difficulties of labor subject the newborn to a great risk of later deficiency, so that they must be regarded etiological factors of great importance in the various forms of physical and mental defects, becoming apparent only later in life.

Klotz justly emphasizes that in the individual case a history

both of difficult labor and of bad heredity does not *a priori* permit the deduction that heredity alone explains the deficiency. We can see the same appreciation of the difficulty of a decision, but from the opposite point of view, when Goddard in his exhaustive study of the causes of feeble-mindedness, makes the following statement: "Since many normal children had been delivered by the use of instruments, with more or less temporary deformity of the head but without any effect on mentality, it is unreasonable to conclude in those cases where there is both hereditary feeble-mindedness and a history of instrumental delivery, that the latter is the cause of the mental deficiency." Some clinical evidence has been cited in the foregoing pages which might be adduced in confirmation of still another opinion, expressed by Wulff almost thirty years ago, that cranial trauma might prove specifically risky for the child with a faulty heredity.

If writers, as, e.g., Tucker, believe that epilepsy in every case is due to definite cerebral cell lesions and that an idiopathic type of epilepsy does not exist, they necessarily are forced to the conclusion that very many cases of epilepsy must be due to birth traumatism. It is not quite clear how these writers can harmonize their views with the prevalent opinion of the importance of faulty heredity in the etiology of epilepsy, except by accepting Wulff's hypothesis as an established fact.

The problem of prognosis concerning delayed sequelae has become more complex with the knowledge that undeniably also the physiological trauma of normal labor occasionally must account for them.

The following facts now seem irrefutably established: (1) Intracranial traumatic lesions, light and severe, develop in the course both of normal and abnormal labors. (2) Evidences of such injuries can be discovered at autopsy in approximately one-half of all infants, stillborn or dying within the first few days of life. But only in one-half of these cases the injury caused the death. In the other half such traumatic lesions, chiefly tentorial lacerations without or with only slight hemorrhage (Vischer), represent but incidental findings. (3) Post-mortem examinations of very young infants up to the age of two years reveal frequently the residuae of older hemorrhages which at least in part must have occurred at the time of birth. In some of these cases definite symptoms at birth had suggested or clearly indicated an intracranial traumatism, in many others even

most careful observation had failed to reveal any sign of an anomaly. (4) Changes in the eyeground of the newborn, presumably the expression of an intracranial parturitional trauma, can be discovered in a considerable number of cases (according to Paul, in 20 per cent of normal labors, and 50 per cent of contracted pelves). (5) Reliable observations, in some instances extending far into adult life, have established the fact that a perfectly normal physical and mental development is certainly not precluded by marked cranial and intracranial injuries evidenced by such unmistakable symptoms of the newborn as convulsions (Seitz).

The present situation concerning the problem of possible delayed effects of parturitional brain injuries may be briefly stated as follows: meninges and brain are injured very frequently in the course both of abnormal and also of seemingly normal labors. Presumably in the large majority of instances in which the trauma leads to a free hemorrhage, death ensues within a short time. In those instances in which the immediate symptoms of the intracranial lesion subside, later consequences might be expected though undoubtedly often they do not appear. Obviously no later consequences can be looked for if immediate symptoms either do not become manifest or are not recognized. Certain post-mortem findings prove that the lesions may be so slight that they can hardly cause noticeable symptoms. Whether such slight lesions can be responsible for delayed manifestations, especially in form of mental defects, remains an unsettled problem though theoretically the possibility cannot be denied. However, without fear of contradiction I make the assertion that in a large number of cases to-day definite symptoms of intracranial parturitional injuries are overlooked. The obstetrician of to-day still fails to appreciate his responsibility in this matter. Obstetric routine will have to include a careful examination of the newborn and observation of his behavior during the first few days of life. Such a routine, which some day may possibly include an ophthalmoscopic examination, a study of the coagulation time of the blood, and possibly a spinal puncture in all cases of suspected hemorrhage, will tend to reduce greatly the number of that still too large group of unrecognized cranial lesions. Such a routine may help clear up the question of the etiologic relation of birth trauma to later physical and mental defects, and incidentally may lessen the occurrence of such defects by affording an opportunity for prompt and effective therapeutic intervention.

**Prophylaxis.**—In the preceding discussion of the etiology of intracranial birth lesions, it has been shown that in the main they are the direct result of sudden or excessive compression of the head. It has furthermore been proved that in the presence of certain predisposing factors, especially of prematurity, the trauma of spontaneous normal labor may be sufficient to injure intracranial structures, and finally, that asphyxiation, brusque manipulation during resuscitation, and a hemorrhagic diathesis, as contributory causes, tend to exaggerate the effect even of minor lesions.

From this point of view it is entirely feasible to discuss an obstetrical prophylaxis of intracranial birth traumatization.

The newer knowledge, that severe traumatic lesions frequently occur also in spontaneous labors, has lessened but not eliminated that responsibility with which the obstetrician used to be charged for such injuries. Certain obstetric manipulations and procedures undeniably prove disastrous to the infant. Disregard of certain details in the proper execution of obstetrical operations renders these procedures needlessly dangerous.

**FORCEPS.**—The immediate infantile mortality in 285 cases of high forceps has been calculated by Baisch as between 43 and 50 per cent. Almost without exception autopsy on these children reveals a serious cranial traumatism. The danger of the high forceps is notorious and every obstetrician of experience tries to evade this operation.

Great risk to the child is not limited to high forceps. Gans investigated the immediate and delayed effects on the infant. Of a total of 562 forceps extractions performed in Winter's clinic, and this means a large series of operations performed only by more or less expert obstetricians, he found (excepting the macerated feti) an immediate mortality of 10.32 per cent, which is a rather low figure when compared with the statistical reports of other large maternity services which give a percentage of immediate mortality varying between 7 and 27.7 per cent.

Still more impressive are certain figures taken from a study made by E. Sachs. He took a special group of forceps extractions *made solely in the interest of the child*. In these 90 cases the infant was delivered in a normal and apparently unimpaired condition only 56 times. The child was asphyxiated slightly in 21, very deeply in 11, and stillborn in 2 cases. One of the deeply asphyxiated babies

died immediately. Of the 3 that died, at autopsy 1 showed a parietal fracture, the two others tentorial lacerations.

A list of traumatic lesions seen in connection with forceps extractions will include, outside of minor external injuries, the following serious accidents: intracranial hemorrhages, injuries of skull bones varying from indentations to deep gutter fractures, fractures of the occipital squama, lacerations of sutures, tentorial tears, facial paralysis, Erb's palsy, injuries to ears and eyes, fractures of the jaw, laceration of the sternomastoid muscle, accidental compression of the umbilical cord, and injuries of the vertebral column and spinal cord.

It will be well to remember that these represent injuries observed in obstetrical clinics where surely the majority of operations are in expert hands, and performed in general only under well-defined indications. A reduction in the number of these injuries, which in part are unavoidable, in the general practice of obstetrics, can be achieved only by limitation of the number of forceps applications.

Forceps must be applied only under definite indications. When the operation seems desirable *in the interest of the mother*, the possible mutilation of the infant should be taken into account more seriously than is the prevailing custom. This applies particularly to the obvious readiness of many practitioners to apply the forceps on account of assumed exhaustion of the patient, which more critical analysis in many cases would reveal to be rather impatience of the parturient or weariness of the attendant. With the fetal head on the pelvic floor, the popular low forceps, often to the great disadvantage of the child, could be replaced by a small dose of pituitrin, an episiotomy, if necessary, being added to eliminate abnormal resistance on the part of the pelvic floor.

In considering a forceps extraction *in the interest of the child*, one must keep in mind the fact that prolonged compression of the head no doubt is less harmful to the infant than a difficult extraction. It is in this type of case that the liberal incision advocated by DeLee for his prophylactic forceps may prove particularly advantageous to the child. Authorities do not agree as to the exact significance of changes in the fetal pulse rate in the recognition of approaching danger. The observations of Baumm have already been mentioned. He concluded that a bradycardia by itself is of no prognostic value. A tachycardia following a marked retardation of the fetal pulse, in his opinion, indicates that a brain hemorrhage already has oc-



curred. Even quick delivery cannot any longer save the child. Only if in an afebrile woman a fetal tachycardia is not preceded by a bradycardia does he advocate prompt application of the forceps for the purpose of saving the infant. Benthin, on the other hand, summarizes his experience in the statement that an absolute indication for immediate delivery exists if either the fetal heart beat remains below 100 at least during two consecutive uterine contractions, or the pulse rises above 180 with simultaneous passage of meconium, especially if the pulse rate fluctuates.

Careful observation of the fetal heart during labor in some cases will enable the attendant to recognize the imminent danger, but no hard and fast rules can be made concerning any typical changes in the pulse rate which would necessitate hurried delivery in the interest of the infant.

The decision of forceps application in the interest of the child, in the individual case, still is a matter of personal discrimination on the part of the operator. The one thoroughly familiar with the problem of birth injuries in the case in which the head is still higher up in the pelvis, will not fail to realize that any possible advantage to the child from a quick delivery is likely to be nullified by the greater risk of intracranial injury, and in such a situation will risk a forceps extraction only if the peril to the child is obvious and not merely suspected. Even in a low forceps he will eliminate any possible danger of sudden and severe compression of the head by an unprepared or abnormally rigid vulvar ring by making an episiotomy.

Serious traumatization of the head is proportionately reduced by greater skill of the attendant, not only by mere mechanical skill, but by his ability to recognize slighter degrees of disproportion between head and pelvis, minor and rarer anomalies of the pelvis, and all deviations from normal in the mechanism of the passage of the head through the pelvic canal. Both this diagnostic ability and the technical skill required for the correct performance of the operation, in the main, are the combined product of experience and judgment. There cannot be any doubt that as a whole the practitioner underestimates the difficulty of determining the necessity for a forceps extraction and of performing it properly with the least possible risk to the child.

In many instances serious or fatal injury can be ascribed to the following defects in the technic: the exact state of rotation of

the head is not recognized and the forceps applied in the transverse diameter of the pelvis, but not, as it should be, as closely as possible corresponding to the transverse diameter of the fetal head. During the progress of extraction the direction of traction is not changed so as to correspond exactly to the sequence of flexion, rotation, and extension required by the normal mechanism as the head passes from one pelvic plane into the next. A failure to adhere to these requirements always implies excessive and dangerous compression of the head in one or another direction.

The forceps should never be employed in an effort to overcome difficulties arising from a marked disproportion between head and pelvis. The traction should be made slowly and with interruptions sufficient to permit the head to mold gradually. If the head is larger than normal, or the forceps' blades, on the incompletely or abnormally rotated head, cannot be applied along the shorter transverse diameters, the forceps' handles necessarily will not lie in close apposition. They must be steadied in their relative position to avoid their compression during extraction.

MANAGEMENT OF THE AFTERCOMING HEAD.—Statistics from large maternity services show an immediate infant mortality varying between 1.5 and 4 per cent for breech labors. It is conservatively estimated that the mortality under less skilled management in general practice probably averages 15 per cent. The clinical diagnosis of the cause of death still commonly is given as asphyxiation, presumably the result of the compression of the umbilical cord between the pelvic wall and the aftercoming head. Post-mortem studies, however, leave no doubt that in the majority of cases death is actually caused by spinal and intracerebral injuries, chiefly tentorial lacerations. If we take into consideration that outside of such fatal traumatism also many slighter lesions, such as fractures of the upper extremities, or of the clavicle, damage to the sternomastoid muscle or brachial plexus are fairly common sequelae of labors in breech presentation, it becomes obvious that correction of the malpresentation by external version, before labor has started, represents a measure of distinct value in the prevention of birth injuries. Next in importance from the standpoint of prophylaxis is strict adherence to definite principles during the extraction of the aftercoming head. It must be kept in perfect flexion, and at the proper moment the occiput must be rotated forward to avoid excessive compression by forcibly pulling too long a head diameter through comparatively too

small a pelvic diameter. It has been shown in foregoing pages how, in the delivery of the aftercoming head, a tentorial tear may result if the cerebellum is brusquely pushed upwards against the tentorium. This might be done if the occipital squama were forced against the pubic arch either by undue efforts to protect the perineum or by a very quick extraction of the head. It seems reasonable to assume that slow and deliberate extraction of the head through a vagina and vulvar ring, either dilated after the method of Potter or, if necessary, properly widened by means of an episiotomy, will greatly lessen the danger of intracephalic traumatization without unduly increasing the risk of asphyxiation. In some instances the application of the forceps to the aftercoming head may prove useful.

PREMATURE LABOR.—There probably is no exaggeration in Vaglio's statement that the notoriously high immediate mortality of premature infants may be due rather to the trauma of birth than, as is customarily assumed, to the immaturity of the newborn. Routine autopsies on all newborn infants bear evidence for the frequency of severe injuries after normal and quick labors in premature babies and even very small feti. The induction of premature labor, as a method of dealing with minor contractions of the pelvis, now is rather generally discouraged on account of the great loss of children in this procedure.

The evident vulnerability of the immature child renders it necessary also in spontaneous labors to avoid so far as possible any severe or abrupt compression of the skull. Even a small dose of pituitrin, if sufficient quickly to force the head through an incompletely dilated cervix or rigid vulvar opening, probably proves disastrous to many an infant.

PROTECTION OF THE PERINEUM.—The attempt to push the head through the vulvar ring between uterine contractions by means of strong pressure against the child's forehead either over the distended perineum or with a finger introduced into the mother's rectum, may cause an intracranial injury, most likely a tentorial tear. In this maneuver the occiput is too forcibly pressed against the subpubic arch. Injuries probably also prove an asymmetric pressure against the forehead, exerted more strongly over the one than over the other frontal or parietal bone. It is prone to strain the falx in an oblique direction. Passage of the head over an unyielding perineum is best retarded by symmetric pressure with the palm against the entire

exposed surface of the occiput. One familiar with the etiology of cranial birth injuries will most readily resort to an episiotomy.

**RESUSCITATION.**—All brusque manipulations must be shunned during the resuscitation of the asphyxiated newborn. In textbooks of obstetrics Schultze's swingings should not be given the prominent place among the methods of artificial respiration still accorded to them. The method should never be described or recommended without proper warning against its various dangers, which can be avoided only by the experienced. If the asphyxiation of the newborn is not due to an intracerebral lesion, such simpler procedures as clearing of the pharynx of mucus, skin stimulation, including the hot and cold bath, tongue traction, or possibly careful compression of the thorax, etc., will prove sufficient to establish suspended respiration. In every case of seemingly deep asphyxia the possibility of a cranial trauma as the cause of the asphyxiation must be kept in mind. All vigorous manipulations in such cases and all procedures in which the baby is held by its feet, head down, are prone to aggravate the condition.

**TWILIGHT SLEEP.**—Even the most enthusiastic advocates of twilight sleep seem willing to concede the following facts: Twilight sleep lengthens labor, necessitates the application of forceps in many cases in spite of the administration of pituitrin and causes some degree of asphyxiation in a considerable number of newborn infants. Thus the procedure includes four distinct factors whose etiologic importance in the causation of intracranial injuries is generally recognized. As a matter of fact, conclusive evidence for a direct relation of twilight sleep to physical or mental deficiency later in life is still wanting, but various writers express the opinion that such a relation will some day be established. In the reports of individual observations of later defects presumably the result of nonfatal lesions sustained at birth, one often meets with the assertion of the writers that twilight sleep was responsible for the injury.

**Treatment**—**INJECTION OF BLOOD OR SERUM.**—Much clinical evidence has been adduced in preceding pages to prove that a hemorrhagic tendency necessarily represents a most serious complication of any, even the slightest, traumatic injury within the skull of the newborn. It must be conceded that Rodda's simple method of ascertaining the clotting time, and Duke's method of establishing the bleeding time are not scientifically accurate, and indeed may be misleading in some cases. On the other hand, it can

be asserted that especially the subcutaneous injection of whole blood is void of any danger to the child. It, therefore, seems entirely logical that all authors who have investigated the relation of hemorrhagic diathesis to birth hemorrhages, without any exceptions, insist on the early and repeated injection of human whole blood, or of animal serum, thromboplastin, etc., whenever an anomaly of coagulability is reasonably suspected. Most of these investigators strongly advocate, without any preliminary test, subcutaneous injection of from 10 to 30 cubic centimeters of the father's blood, if necessary repeated in intervals of from four to eight hours, in every instance in which, immediately after birth or later, certain symptoms indicate an intracranial hemorrhage. This therapy is not only likely to check a further extravasation of blood but is also a very rational and useful prophylactic procedure in case further developments should make surgical interference inevitable. Seemingly excellent results are recorded in literature with blood injections. Foote obtained 6 permanent cures in 7 cases treated in this manner after the hemorrhage had been definitely established by spinal puncture.

The value of the prophylactic injection of blood before operation is well illustrated in 2 observations cited by Rodda: Case IV. Spontaneous delivery after long labor. Cephalhematoma, petechial hemorrhages on arms, chest, and abdomen. Spasticity of right hand. Twelve hours post partum, 30 cubic centimeters of blood injected. Seven hours later another 20 cubic centimeters. Coagulation time, four minutes, bleeding time eleven minutes. On second day: First convulsions. Spinal fluid bloody. Coagulation time unchanged, bleeding time reduced to four and one-half minutes. Third day: Coagulation and bleeding time unchanged. Another 22 cubic centimeters of blood injected. Fourth day: Operation. By means of an osteoplastic flap a large blood clot is removed. Twenty-five cubic centimeters of blood injected. From then on gradual recovery. At the age of ten months child seems normal in every respect, though its head is rather large.

Case V. Baby born asphyxiated in breech presentation. Coagulation time twelve minutes, bleeding time nineteen minutes. Injection of 43 cubic centimeters of blood. Second day: Coagulation time six minutes, bleeding time six minutes. Injection of 15 cubic centimeters of blood. Third day: First convulsions. Twenty-five cubic centimeters of blood injected twice. Fourth day: Frequent convulsions. Coagulation time six minutes, bleeding time ten min-

utes. Twenty and twenty-five cubic centimeters of blood injected. Fifth day: Coagulation time eight minutes. After injection of 25 cubic centimeters of blood, operation. Brain bulges through a dura incision, hemorrhage presumably on the other side. Child recovers and at the age of four months seems perfectly well.

**SPINAL PUNCTURE.**—Quite unlike the subcutaneous injection of blood, spinal puncture on the newborn is a procedure both comparatively difficult in execution and not entirely free of danger to the child. These two facts seem to be appreciated by all the writers who have extensively tested the method, and nevertheless they show no hesitation in recommending, some of them most extravagantly, spinal puncture as a routine procedure for all cases in which an intracerebral hemorrhage is suspected. A justification for this attitude can be found in the double advantage of spinal puncture both in the early diagnosis and in the treatment of these hemorrhages. The value of the method as a diagnostic aid already has been amply discussed in this chapter. There is considerable evidence available to prove that the withdrawal of blood by means of the puncture in many instances is followed by an immediate, and not so rarely by a permanent, subsidence of all the symptoms of the intracranial hypertension, especially if the puncture is repeated. Even when no blood is found, the withdrawal of spinal fluid tends to relieve the pressure symptoms. Only favorable results are likely to appear in literature, but observations like those recorded, e.g., by Brady, convincingly prove the curative effect of spinal puncture at least in some of the cases, particularly if the hemorrhage is of the infratentorial type.

Brady saw 9 cases of meningeal hemorrhages in the newborn. Four of them died quickly. Two survived and now are typical examples of Little's disease. In the 3 remaining cases, spinal puncture was tried, resulting in the recovery of two: (1) First convulsions twenty-four hours after delivery. Twelve hours later 30 cubic centimeters of pure blood are removed from the spinal canal. All symptoms disappear promptly, the distended fontanel recedes. Rapid and permanent recovery. (2) Child first seen six days after a difficult forceps delivery. Stupor. Large fontanel bulging, sutures widely separated. Embarrassed respiration. Infant apparently is in a hopeless condition. Lumbar puncture. Approximately 60 cubic centimeters of blood fairly gush from the needle. Fontanel recedes promptly, but gradually begins to bulge again. Next day another lumbar puncture. Blood escapes freely. On third and fourth days

puncture repeated, on account of renewed bulging of fontanel. Each puncture followed by satisfactory immediate result. A total of 240 cubic centimeters of blood and spinal fluid had been withdrawn by the several punctures. Infant now begins to improve and shortly afterwards to thrive. At age of fourteen months the child is perfectly normal in every respect.

Many illustrative cases of this sort could be quoted, but I shall limit myself to mention only a most instructive observation of Lippman: Premature infant, weighing  $5\frac{1}{2}$  pounds. A lumbar puncture yields 25 cubic centimeters of blood. The bulging fontanel flattens immediately. Child recovers and remains well.

A single or repeated lumbar puncture in the hands of many writers (R. M. Green, Devraigne, Brailon, Cathala, etc.) has yielded satisfactory, and often surprising, results so frequently that it seems perfectly logical that other investigators went a step further. A spinal puncture between second and third cervical vertebrae would hold greater promise than a lumbar puncture to relieve the dangerous pressure of a peribulbar hematoma against the medulla oblongata. Results so far recorded justify this procedure in selected cases. The same holds true for cranial punctures made through a fontanel or suture, which have been mentioned in preceding pages as aids in diagnosis. Such punctures are advocated by Frazier, Doazen, Green and others; their technic is described in detail in the paper of Henschen.

**SYMPTOMATIC TREATMENT.**—The following protective measures can be usefully applied in all cases of actual or suspected intracranial trauma: the infant is manipulated as little as possible. He is kept in a warm and quiet room, and protected against all irritation including that of glaring light. An icebag is placed on his head. Bromides and chloral hydrate are given as indicated by spasticity or convulsions. If the infant is unable to nurse, he is fed through a catheter used as a stomach tube.

**OPERATIVE TREATMENT.**—In a volume like this, designed chiefly for the needs of the obstetricians and of all practitioners attending women in labor, it would be inconsistent to dwell in detail on the various operative methods which have been evolved for the treatment of intracranial birth hemorrhages. Their adequate description will be found in many modern treatises of surgery and in the writings of Brindeau, Carmichael, Cushing, Doazen, Frazier, Gilles, Sharpe, and Vignes, to mention but a few of the most important contribu-

tions. The operations recommended by these writers comprise small incisions, small and large trephine openings and large osteoplastic flaps.

It also seems superfluous, if not impossible, to quote here percentage figures of surgical cures. It can be readily admitted that the operative results so far obtained are far from satisfactory. This fault, however, in general should not be charged either against the operator or any specific operation. It is obviously due to the fact that most operations to-day are performed at a time when the condition of the child is practically hopeless. Every critical student of this more recent literature on cranial birth trauma is forced to the conclusion that better results of all operative interference, including spinal puncture, can be hoped for only when the obstetrician will have come to a full realization of his responsibility for a most careful observation of the newborn infant immediately after birth and during the first few days of life. Such practice will necessarily lead to the early detection of signs of an intracerebral injury. It will also reveal all the symptoms which have preceded the first convulsion or the state of coma, the condition in which the surgeon at present usually finds his little patient when called for consultation. Only early therapeutic interference, surgical and nonsurgical, can be expected to lessen the immediate and delayed untoward effects of an intracranial birth trauma on the child's life and future health.

#### LITERATURE

- Abels. *Arch. f. Gynäk., Berl.*, 1913, 99:1.  
 Bailey. *Am. J. Obst. & Gynec.*, 1920, 1:52.  
 Bauereisen. *Zentralbl. f. Gynäk., Leipz.*, 1911, 35:1149.  
 Idem. *München. med. Wchnschr.*, 1912, 59:1035.  
 Baum. *Arch. f. Gynäk., Berl.*, 1917, 107:353.  
 Beneke. *München. med. Wchnschr.*, 1910, 57:2125.  
 Beneke and Zausch. *Zentralbl. f. Gynäk., Leipz.*, 1920, 44:34.  
 Benthin. *Monatschr. f. Geburtsh. u. Gynäk., Berl.*, 1912, 36:308.  
 Bonhoff and Esch. *Ztschr. f. Geburtsh. u. Gynäk., Stuttg.*, 1912, 70:886.  
 Brady. *J. Am. M. Ass., Chicago*, 1918, 71:347.  
 Brindeau. *Arch. men. d'obst. et de gynéc., Par.*, 1918, 10:103.  
 Carmichael. *Scot. M. & S. J., Edinb.*, 1906, p. 524.  
 Couvelaire. *Ann. de gynéc. et d'obst., Par.*, 1903, 59:253.



- Idem. *Ibidem*, 1907, Deux. Ser. 4:7.
- Cushing. *Am. J. M. Sc.*, Phila., 1905, 130:580.
- Doazen. *Arch. gén. de chir.*, Par., 1913, 9:10.
- Engelken. *Nederl. Tijdschr. v. Geneesk.*, Amst., 1920, 2:1538.
- Esch. *Arch. f. Gynäk.*, Berl., 1909, 88:60.
- Idem. *Ztschr. f. Geburtsh. u. Gynäk.*, Stuttg., 1910, 65:52.
- Finkelstein. *Berl. Klinik*, 1902, No. 168.
- Fischer. *Ztschr. f. Kinderh.*, Berl., 1911, 2:248.
- Foote. *Am. J. Dis. Child.*, Chicago, 1920, 20:17.
- Frazier. *Tr. Am. Surg. Ass.*, Phila., 1906, 24:316.
- Idem. *J. Am. M. Ass.*, Chicago, 1913, 61:2096.
- Gans. *Monatschr. f. Geburtsh. u. Gynäk.*, Berl., 1908, 27:430.
- Gilles. *Abstr. in J. Am. M. Ass.*, Chicago, 1912, 59:1494.
- Green. *Boston M. & S. J.*, 1914, 170:682.
- Idem. *Ibidem*, 1916, 174:947.
- Groszmann. *N. York M. J.*, 1917, 105:827.
- Hannes. *Ztschr. f. Geburtsh. u. Gynäk.*, Stuttg., 1911, 68:680.
- Idem. *Zentralbl. f. Gynäk.*, Leipz., 1921, 45:1037.
- Hedrén. *Förh. Svens. Läk.-Sällsk. Sammakn.*, 1918, 44:43. (Author's abstract in German.)
- Henschen. *Zentralbl. f. Gynäk.*, Leipz., 1913, 37:925.
- Herff. *Ibidem*, 1912, 36:1241.
- Herrman. *Arch. Pediat.* 1915, 32:583.
- Kearney. *Am. J. Obst.*, N. Y., 1917, 76:904.
- Klotz. *Ztschr. f. Neurol. u. Psychiat.*, 1913, 8:1.
- Kowitz. *Virchow's Arch. f. path. Anat. (etc.)*, Berl., 1914, 215:233.
- Kwozek. *Dissertation*, Breslau, 1914, abstr. in *Zentralbl. f. Gynäk.*, Leipz., 1920, 44:983.
- Leclercq and Paput. *Gynécologie*, 1913, 17:213.
- Lippman. *N. York M. J.*, 1916, 103:263.
- Mayer. *Zentralbl. f. Gynäk.*, Leipz., 1915, 46:795.
- Meyer and Hauch. *Abstr. in Zentralbl. f. Gynäk.*, Leipz., 1913, 37:707.
- Idem. *Abstr. in J. Am. M. Ass.*, Chicago, 1912, 59:2196.
- Moreno. *Arch. mens. d'obst. et de gynéc.*, Par., 1915, 4:114.
- Morse. *Am. J. Dis. Child.*, Chicago, 1919, 18:73.
- Neustaedter. *Am. J. Obst.*, N. Y., 1915, 72:520.
- Oden. *J. Am. M. Ass.*, Chicago, 1915, 44:816.
- Paul. *Dissertation*, Halle, 1900, quoted by Seitz: *Zentralbl. f. Gynäk.*, Leipz., 1912, 36:1.

- Pott. *Ztschr. f. Geburtsh. u. Gynäk.*, Stuttg., 1911, 69:674.  
 Rechtschaft. Dissertation, 1918, abstr. in *Monatschr. f. Geburtsh. u. Gynäk.*, Berl., 1918, 48:283.
- Reuss. *Krankheiten des Neugeborenen*, Springer, Berlin, 1914.
- Rodda. *J. Am. M. Ass.*, Chicago, 1920, 75:452.
- Idem. *Am. J. Dis. Child.*, Chicago, 1920, 19:269.
- Sachs. *Therap. d. Gegenw.*, Berl., 1920, 61:16.
- Schaefer. *Zentralbl. f. Gynäk.*, Leipz., 1921, 45:829.
- Seitz. *Winckel's Handb. d. Geburtsh.*, 3:3, p. 70.
- Idem. *Arch. f. Gynäk.*, Berl., 1907, 82:528.
- Idem. *Arch. f. Gynäk.*, Berl., 1907, 83:701.
- Idem. *Zentralbl. f. Gynäk.*, Leipz., 1912, 36:1.
- Sharpe. Paper read before Clinical Society of New York Polyclinic Hospital, on October 1, 1917.
- Sidbury. *Arch. Pediat.*, 1920, 37:545.
- Stein. *J. Am. M. Ass.*, Chicago, 1917, 59:334.
- Stumpf and Sicherer. *Beitr. z. Geburtsh. u. Gynäk.*, Leipz., 1909, 13:408.
- Tucker. *J. Am. M. Ass.*, Chicago, 1913, 61:2096.
- Vaglio. Abstr. in *J. Am. M. Ass.*, Chicago, 1921, 76:688.
- Vignes. *Progrès méd.*, Par., Sept. 21, 1918.
- Vischer. *Cor.-Bl. Schweiz. Aerzte*, 1919, 49:230.
- Vogt. *Arch. f. Kinderh.*, Stuttg., 1908, 48:321.
- Volland. *Allg. Ztschr. f. Psychiat.*, Berl., 1906, 63:725.
- Waldstein. *Zentralbl. f. Gynäk.*, Leipz., 1912, 36:1704.
- Wallich. *Ann. de gynéc. et d'obst.*, Par., 1913, 70:600.
- Warwick. *Am. J. M. Sc.*, Phila., 1919, 158:95.
- Idem. *Am. J. Dis. Child.*, Chicago, 1921, 21:488.
- Wilcox. *Boston M. & S. J.*, 1913, 168:568.
- Wilke. *München. med. Wchnschr.*, 1912, 59: 1880.
- Winn. *J. Am. M. Ass.*, Chicago, 1913, 61:2096.

## CHAPTER VI

### INJURIES OF THE VERTEBRAL COLUMN AND SPINAL CORD

Pathology and etiology of injuries of vertebral column—Pathology and etiology of injuries of the spinal cord—Frequency of spinal and cord injuries—Prognosis—Symptomatology—Diagnosis—Prophylaxis—Treatment.

Probably never in spontaneous labors, but fairly often in manipulations necessitated by a dystocia, especially in version and extraction, the vertebral column of the child is fractured or ruptured. At times the injury is limited to a temporary luxation of a vertebra. In cases of injury to the spine the cord may be traumatized secondarily by a hemorrhage, or by a more or less complete compression. However, occasionally there is found a complete or partial rupture of the cord with smaller or larger medullary cord hemorrhages in the absence of any discoverable injury to the vertebral column. For this reason it seemed advantageous to consider in this chapter the frequency, prognosis, symptomatology, diagnosis and prophylaxis of the injuries of the spine conjointly with those of the cord.

### INJURIES OF THE VERTEBRAL COLUMN

**Pathology.**—Rokitansky, in 1874, claimed that experiments had convinced him that the spine of the newborn is immune against even most forceful axial traction. This view prevailed for a considerable time in obstetrical teaching in spite of the fact that Duncan approximately at the same time (1874) had announced that his experiments and observations had shown that the cervical portion was the most fragile part of the entire vertebral column in the newborn, and was liable to be fractured even by traction exerted exactly along the child's longitudinal axis. Only two years later Carl Ruge demonstrated the fact, never since refuted, that these obstetrical vertebral fractures really consist in the detachment of a vertebral epiphysis along the normal demarcation line. However, only recently this problem has been thoroughly studied and satisfactory explanations

have been offered for the mechanical causes of the various types of spinal injuries sustained during birth.

Hofbauer (1907) confirmed that injuries to the spine most often occur in its cervical section. With the sole exception of the first and seventh, all other cervical vertebra may be ruptured in an epiphyseal line. Hofbauer, probably as the first, laid stress on the now generally conceded fact that the sixth cervical vertebra most frequently is involved, e.g., in 3 of the 4 cases seen by him. In every instance he found the injury to consist in a separation of an epiphysis. As the result of a careful anatomic study he offered the following explanation of this fact: the fibrocartilage between the sixth and seventh cervical vertebra is almost twice as thick as the one between the fifth and the sixth, an anatomic finding apparently never mentioned in textbooks. If forcible traction is exerted on the shoulders, it presumably will manifest its injurious effect at the nearest point of least resistance, and this would be just above the particularly firm sixth intervertebral disc. The sixth cervical vertebra, in his opinion, also is the one least protected by muscle attachments. The anterior scalenus muscle is attached to the transverse processes of the third to the sixth vertebrae, another factor favoring the abruption of the epiphysis just below the edge of the muscle. Sachs, however, is unwilling to accept this explanation because it does not account for the evident fact that the seventh vertebra, exposed to injury in the same manner, apparently proves immune. Also Sachs' experiments proved the specific vulnerability of the sixth vertebra and he knows of no other acceptable reason for it than that the sixth cervical vertebra probably is not so firmly constructed as the others. Much additional information concerning vertebral birth traumatization in the cervical section has been furnished by the anatomic investigations of Stoltzenberg. In all the 9 cases studied by him, one intervertebral joint capsule together with the adjoining portions of the intercrural ligaments was torn on one side. He assumes that in extreme cases this rupture then extends further into the cartilaginous portion of a vertebra.

Complete traumatic separation of the spine at the neck is possible. Not so rarely as would seem from records in literature, the aftercoming, and occasionally also the forecoming, head is torn off. This is not uncommon in macerated feti, but has been reported also in cases in which the children were living, some of them premature, others mature. Pinzani (quoted by Stumpf) speaks in a case of this

sort of an abnormal fragility. While comparatively slight traction was made with a forceps, the head of the living child suddenly separated from the trunk. In the attempt to remove the trunk by version and extraction, both lower extremities and one arm were torn out, though, as he asserts, no noteworthy resistance or difficulty was met in the procedure. In a case recorded by Rewolinski, a woman giving birth to her first child without any assistance, actually tore off the already delivered head while endeavoring to pull out the shoulders. The deliberate cutting off of the newborn's head is known in forensic literature but undeniably represents a most unusual type of infanticide. Cases have been repeatedly reported in which midwives and occasionally also physicians, unable to deliver the aftercoming head, had severed the child's neck to facilitate the transport of the mother to a hospital.

Decidedly rarer are fractures in other than the cervical section of the vertebral column. Among them injuries in the thoracic section are more common than those involving the lumbar spine.

A dislocation of a vertebra, apparently occurring only in the lower thoracic and the upper lumbar sections, occasionally is mentioned by writers as the presumable cause for certain types of paraplegic palsies. Holman recently described a case of persisting dislocation which illustrates this accident unusually well. In a child, four years old, suffering since birth from a flaccid paralysis of both legs, below the knees, the responsible luxation of the first lumbar vertebra was still visible in roentgenograms. He felt justified in the conclusion that the luxation represented a birth injury. The child was born prematurely, with an estimated weight of two and a half pounds, after version and extraction. Within a month the mother had observed a "sinking in of the child's back" and also the paralysis of the lower extremities. Holman suspects that such cases do not appear more frequently in literature because the victim of such a severe injury is not likely to survive. It is possible, in his opinion, that a case reported by Kleinberg (*J. Am. M. Ass.*, 1916, 66:736) as "congenital anterior curvature of the spine" is of the identical character. Also in this case the child was delivered in breech presentation. It seems likely to him that some of the cases of so-called "congenital scoliosis" really are instances of birth injuries of the spinal column.

**Etiology.**—The anatomic characteristics of these injuries, and the noteworthy fact that they have been observed only after operative

deliveries, mostly after version followed by extraction, occasionally after difficult forceps extractions, but never after spontaneous labors, clearly emphasize the mechanical element in their causation. Prematurity evidently is a predisposing factor of considerable importance. These injuries obviously are the result of a strong pulling force, but most investigators agree that straight traction in the axis of the spine probably is insufficient to cause the damage, even if in a breech delivery an aftercoming head, or in a normal vertex labor very broad shoulders or an abnormally distended abdomen necessitate forceful traction. Rather generally it is claimed, as the result of observation and experiment, that a spinal injury occurs only if strong traction is made in a lateral direction, or is combined either with a torsion or a sharp bending of the spine, especially backwards towards hyperextension. It thus is obvious that some of the common maneuvers in the management of breech labor offer ample opportunity for traumatic injury to the spine.

**LATERAL TRACTION.**—Stolzenberg's anatomic findings of joint capsule rupture on one side only well justify his claim that the damage to the largest extent and most often is due to a lateral deviation in the direction of traction. He considers difficulty in freeing an arm, caught in the nape of the neck, the most favorable situation for the unilateral rupture of a cervical joint capsule. With the head fixed, the spine is pushed sharply over to one side and this deviation now is further aggravated by traction on the arm already born posteriorly. He lays emphasis on the fact that serious damage thus already has been done before the Veit-Smellie maneuver is resorted to, which probably is responsible for a final epiphyseal detachment after the intervertebral ligaments have been torn. In cases of vertex presentation, in Stolzenberg's opinion, forcible traction on the head with simultaneous lateral flexion is particularly dangerous, if the anterior shoulder is held fast behind the symphysis.

**TORSION OF THE VERTEBRAL COLUMN.**—This torsion can cause a laceration of ligaments or a fracture only if the normal and marked ability of the spine of the newborn to be twisted is transgressed. Such overtwisting requires a very firm fixation of the vertebral column at its upper end. This would seem possible only at the level of the shoulders, and, therefore, torsion, according to Sachs, is the predominant factor in the causation of injuries, especially of the thoracic portion, which, in comparison, also is the least flexible portion of the spine. This very fixation of the column at the level of the shoulders pro-

fects the cervical vertebrae against injury from torsion. Excessive turning of either the spine against a fixed head, or of the head against the fixed spine can but rarely cause cervical injury because the atlanto-occipital articulation allows a very wide rotation. Nevertheless a torsion injury of a cervical vertebra in a breech labor after the shoulders are born, might conceivably occur in the following situation: The head may have reached the outlet unrotated and its transverse position is overlooked. In the attempt to bring the back forward the neck is overtwisted. If strong traction is applied to the overrotated head—and it is likely to be strong on account of the unrecognized malposition of the head—a cervical vertebra might be ruptured.

**HYPEREXTENSION.**—Hyperextension rather than traction in a lateral direction, in the belief of Sachs, renders the Veit-Smellie maneuver dangerous. Hofbauer thinks that one attempt, with careful avoidance of any simultaneous rotation, can be tried, but, if ineffective, any further traction should be combined with pressure from above. Sachs goes further and warns emphatically against any attempt with the Veit-Smellie when the head still is above the brim.

The newborn's vertebral column is better adapted to flexion than to extension, a point also made by Laffont. It was held in flexion practically during the entire intra-uterine life. Sachs is certain that the amount of traction required for complete extension probably is sufficient to produce laceration of some of the structures on the ventral aspect of the spine. The same holds true for the ligaments on the lateral aspect of the spine when they are exposed to excessive tension by strong lateral flexion. This, e.g., is the case when the lower end of the fetus is too brusquely lifted above the mother's symphysis while the posterior arm is freed. Manipulations of this sort endanger particularly the thoracic portion of the column.

In the Veit-Smellie procedure the risk to the cervical vertebrae consists in the hyperextension of the cervical column section while it is held under severe tension by the simultaneous traction. This is the case particularly if the head still is high in the pelvic inlet. If in this position of the head, the shoulders are pulled down forcibly with the fingers hooked over them, the traction is exerted chiefly on the upper thoracic vertebrae. In this situation traction is required in a downward direction, but if at this time, incorrectly, the spine is bent back, the chance for injury of the thoracic section is great.

With the head near the outlet in a breech labor, a rigid pelvic

floor, and too great an effort to save the perineum may be responsible for a degree of sagittal hyperextension that can prove disastrous to one of the cervical vertebrae.

Among the many justified and unjustified accusations that have been raised against Schultze's swinging of the asphyxiated newborn, one, often disputed, is the claim (e.g., made by Burekhard) that the forced extension in the moment when the child's legs are thrown backwards may injure the lumbar spine. A similar danger is ascribed to the method of artificial respiration which consists in the alternation of strong flexion of the trunk of the asphyxiated baby with extension (method of Hervie-Byrd).

### INJURIES OF THE SPINAL CORD

**Pathology.**—Approximately in accord with a suggestion made by Kooy the traumatic birth lesions of the spinal cord can be divided into three groups:

The first group comprises secondary cord lesions resulting from a primary injury of the vertebral column, a fracture, rupture, or luxation. The cord may be torn or merely compressed, both either partially or completely, permanently or only temporarily. The pressure is exerted by a vertebra or by a hematoma.

In a second group the cord had become compressed or ruptured, partially or completely, within an apparently intact vertebral column. Very likely in some of these cases the cord injury is caused by a temporary dislocation of a vertebra, not any longer discoverable in a roentgenogram taken at a later date. Kooy thinks that the cases of this group can be compared best with observations occasionally made in the adult subsequent to severe traction at the nerve root, e.g., in stretching the sciatic nerve in tabetics, or by the reposition of a hipjoint dislocation; or in cases in which the spine had been bent acutely and extremely in an accident. In the individual case, later on, it often is impossible to decide by macro- or microscopical examination whether the partial or total destruction of a section of the cord was due to a primary hematomyelia or directly to the injury, since a traumatic rupture of the cord necessarily would be associated with a hemorrhage.

In a third group, small, disseminated hemorrhages are discovered in various sections of the cord, often accompanied by similar minute



extravasations in other parts of the central nervous system, the typical instances of true hematomyelia. Rather generally they still are ascribed to a mechanical rupture of congested veins in the course of various obstetrical manipulations.

If we exclude the cases in which blood enters the spinal canal from a profuse intracranial hemorrhage, most likely produced by a tentorial tear, spinal hemorrhages are either intramedullary, intermeningeal or epidural, the latter representing the most common type (Litzmann). In contradistinction to injuries of the vertebral column which distinctly prevail in the cervical region, spinal cord hemorrhages may be situated in any section of the cord, and this is particularly true of hematomyelia.

**Etiology.**—The pronounced flexibility and elasticity of the vertebral column of the newborn renders it unusually resistant to flexion and torsion. The spinal cord, on the other hand, as pointed out by Goett, fixed as it is at the top by its connection with the brain and along its entire course by the exit of the numerous spinal nerve roots on either side, is less able to yield without harm to pronounced bending and twisting of the spine. Therefore, in general, the same mechanical factors which in foregoing pages have been shown responsible for the traumatic birth injuries of the vertebral column also account for cord lesions, though it is evident that less distortion of the column is required to damage the cord than the spine.

Cord lesions most often have been observed subsequent to breech labors, or especially when breech extraction was preceded by version. Rather generally the fact is noted that the infants were asphyxiated at birth. There is some dissension whether this asphyxiation favors spinal cord lesions directly as the result of venous congestion of cord vessels or indirectly by necessitating certain manipulations during resuscitation. Lawatschek, among others, accuses improper swinging as likely to cause cord apoplexies. Reuss, Leclerq and Paput and others lay stress on the prematurity of the infants in many of these cases.

The generally noted association of small medullary hemorrhages with scattered hemorrhages in the entire central nervous system and in other parts of the body suggests the possible etiologic relation to hemorrhagic disease. Investigations along these lines are still wanting. They can be expected to prove that, at least in some instances, a hematomyelia, as well as intracranial hemorrhages, might be only the local evidence of a hemorrhagic tendency.

**Frequency of Spinal and Cord Injuries.**—No reliable information is available concerning the frequency of birth injuries of the spine. Even careful examination at autopsy rarely includes the study of the spine and the cord. Therefore, the assertion can be made that in the larger number of instances the injury remains unrecognized. By way of illustration we may quote the following observation of Groene. The firstborn of twins became cyanotic on the second day of life and died on the following day. A clinical diagnosis of congenital debility was made. Greatly interested in the problem of vertebral birth injuries Groene included in the post-mortem examination a study of the spine and was surprised to find a large epidural hematoma. Schaeffer (*Archiv für Gynäkologie*, 1897, liii) discovered 10 cases of cord hemorrhage in 100 autopsies of newborn infants. Spencer (as quoted by Burr) in a series of 130 necropsies on fresh, mostly stillborn feti, examined the spinal cord in 44 instances. In 30 of them he found a hemorrhage somewhere within the spinal canal, in 6 cases it was within the spinal cord. Spencer concluded from his observations that a spinal hemorrhage is greatly favored by the presentation of the lower extremity in labor. Stoltzenberg saw in 75 post-mortem examinations of newborn babies 9 lacerations of joint capsules between cervical vertebrae and considered this a typical birth injury especially for children born in breech presentation. Among these 9 cases was but 1 vertex labor in which considerable difficulty had been experienced in the extraction of the shoulders. Sachs' thorough studies concerning the causation of spinal injuries were based on 16 cases seen in a large maternity service within ten years, in a material handled exclusively by more or less expert obstetricians.

**Prognosis.**—The prognosis in the individual case is determined by the size and chiefly by the location of the hematoma. A large effusion of blood near the upper end of the cord, involving the centers in the medulla oblongata, probably proves immediately fatal in the overwhelming majority of all cases, that is, in most cases of rupture or dislocation of a cerebral vertebra. A case like the one recorded by Parrot (1870), and often quoted, represents the exception. In this case the spinal cord was completely severed near the sixth cervical vertebra, but the child lived for six days.

In severe injuries of the thoracic portion of the vertebral column the infant but rarely survives. This undeniably is the reason that neurologic and orthopedic literature contains only a very small num-

ber of well-authenticated observations of palsy of the lower extremities due to parturitional traumatization of the spine. The most carefully analyzed case of Kooy is almost unique. It deals with a child, nine years old, in whom the responsible traumatic lesion at the level of the ninth and tenth thoracic vertebra still could be clearly demonstrated in the X-ray pictures. Burr reported the case of a child four and a half months old, in whom the cord was found degenerated from the root of the fourth cervical to that of the first dorsal nerve. In general, however, the assertion of Lawatschek seems correct, that in birth injuries affecting the upper portion of the vertebral column, as a rule, death ensues within from two to ten days, but is correspondingly delayed the lower down the lesion is located, with the least immediate danger to life in the rarer instances of traumatism of the lower dorsal and lumbar section.

Still less we know concerning the prognosis of smaller intramedullary hemorrhages not due to a primary column injury. If very small, they are generally supposed to cause neither immediate symptoms, nor later sequelae pronounced enough to permit their recognition. Many writers (e.g., Zappert, Birnbaum, Stoltzenberg), however, assume an etiologic relation of syringomyelia to larger cord hemorrhages, caused by a birth traumatism but not recognized at the time of birth. Kooy showed in his case how cord degeneration progresses upwards and downwards from the original focus of the lesion until in older cases it becomes impossible to determine whether the injury originally was a hematomyelia or a rupture of the cord.

Many of the infants, who survive the immediate effect of a cord injury, later succumb to a septicemia from decubitus sores or to an ascending pyelonephritis from a cystitis which develops as a result of the paralysis of the bladder sphincter.

**Symptomatology.**—When death does not occur immediately the clinical picture is characterized by the paraplegia of both lower extremities usually associated with a paralysis of the muscles of the abdominal wall. A few days later the incontinence of bladder and anus will be noticed. In some instances a gradual improvement of the palsy and a final disappearance of all symptoms has been recorded, presumably coincident with the gradual absorption of an epidural hematoma that had compressed the cord from without.

In the cases in which the cord lesion is situated higher up, the upper extremities also are paralyzed and with them some of the

respiratory muscles. Under these conditions an incorrect diagnosis of deep asphyxiation is most likely to be made. The child is pale and limp, its heart beating, but there are no efforts at respiration. Attempts to start respiration artificially of necessity fail, at best the child will gasp a few times under stimulation before the heart stops beating. Hofbauer emphasizes that in suspected cases of injuries to the cervical section all motions of the head—almost unavoidable during resuscitation—should be avoided as prone to cause further effusion of blood.

The clinical picture of a flaccid paraplegia in some cases of cord apoplexy is confused by a simultaneous cortex irritation from coincident intracranial hemorrhages. Thus there might be seen, unexpected in a cord lesion, an opisthotonos, twitching or spasms in certain muscle groups, or even a general convulsion. In this connection the fact must be remembered that in cases of excessive intracranial hemorrhage, especially of the infratentorial type, some of the blood may flow down into the spinal canal and may give rise to such cord symptoms as erection of the penis and contraction of the scrotal skin.

Being of no specific interest to the obstetrician, a detailed discussion of the later sequelae of degenerative processes in the cord, of atrophy and degeneration of the paralyzed muscles with their subsequent contractions as seen in older children by neurologists and orthopedists will be omitted here. In neurological literature one often finds lesions of this sort classed as congenital. This is incorrect. Lesions due to a birth trauma cannot be properly designated congenital. The term congenital should be reserved for those cases of central and peripheral palsy in which the responsible defects in the cerebrospinal system have developed during intra-uterine life.

**Diagnosis.**—A paraplegia, noticeable immediately at birth of a child born by forcible means, practically permits the definite diagnosis of a lesion of the cord by rupture or pressure. The extent of the paralysis will allow its localization. In the absence of any paralysis, e.g., in the case of small scattered hemorrhages, cord lesions necessarily will remain unrecognized, possibly until much later in life.

Spinal puncture, advocated by Birnbaum, and also mentioned by Hofbauer, has its limited value in establishing the diagnosis. Of greater help would seem the X-ray examination of the entire spine in every case of suspected spinal injury, a method apparently not

so generally and promptly applied as it well deserves to be. As a matter of fact, the correct diagnosis is made but rarely in the living newborn, and after death only if examination of the vertebral column and of the spinal cord is part of the autopsy routine.

**Prophylaxis.**—Since these injuries occur most frequently in the course of breech labors, suggestions towards their prevention necessarily deal chiefly with certain details of manipulation during extraction, the freeing of the arms and the delivery of the aftercoming head. Considering the mechanical factors directly responsible for the injury, as set forth in the preceding pages, it becomes necessary never to exert very strong traction especially in a direction which does not accurately coincide with the long axis of the fetus; to avoid torsion of the trunk when the upper end of the spine is firmly fixed and thus cannot follow the twisting motion; and to prevent hyperextension, i.e., deflexion (especially under simultaneous traction) of any portion of the spine which at birth still is in that state of flexion that was maintained all during intra-uterine life.

The Deventer-Mueller method of delivering the shoulders in a breech presentation often has been accused of favoring traumatic injuries of the thoracic section of the spine. Such damage can be prevented if excessive tension is avoided, particularly in the moment when, by torsion of the trunk, the one shoulder is rotated posteriorly. The Mueller method becomes dangerous only if a narrow pelvic inlet, or possibly also a tight contraction ring, or an incompletely dilated cervix holds the shoulders and with them the upper column end so firmly that it cannot follow the turning motion imparted to it by the rotation of the trunk. Unfortunately just in this situation the abnormal resistance is likely to prompt the operator to make stronger traction. In this manner the specifically dangerous combination of forceful traction and torsion is created.

In the maneuver of freeing the posterior arm the infant's trunk must not be bent too brusquely sideways while its legs are pulled high above the maternal symphysis.

No traction should be made while the arm, caught in the nape of the neck, is gently pushed over the infant's face and downwards.

In the delivery of the aftercoming head the dangerous combination of hyperextension and traction must also be shunned, especially any traction in a lateral direction. The Veit-Smellie procedure may be tried, but preferably, if an assistant is available, with simultaneous gentle pressure against the head from above. If the rotated

head is held fast higher up than at the pelvic floor, it will be more advantageous to resort at once to the Martin-Wiegand method. A dangerous degree of hyperextension, and with it unnecessary risk to the cervical spine, is entailed in the entirely superfluous effort to protect a high and rigid perineum by bending the trunk of the infant far back over the mother's abdomen. One should feel no hesitancy in facilitating the delivery of the head in the primipara by a deep episiotomy, if vagina and vulva have not been dilated in the manner suggested by Potter.

Fear of compression of the umbilical cord too often prompts the obstetrician to hurry unnecessarily with the extraction of the after-coming head. He should remember that in the management of a breech labor neglect to perform the various maneuvers gently and precisely implies greater danger to the child than mere asphyxiation.

In vertex presentations the cervical section of the vertebral column is exposed to damage near the end of the second stage at two definite times: (1) if in a forceps extraction the head is forcibly pulled down and at the same time bent far back to protect the perineum; and (2) if, with the anterior shoulder firmly caught behind the symphysis, delivery of the posterior shoulder is effected by pulling the rotated head upwards, a maneuver that will lead to excessive lateral flexion under tension.

Injuries of the lower portions of the spine as the result of hyperextension by brusque execution either of Schultze's swingings or in the Hervie-Byrd method of resuscitation, certainly are preventable.

**Treatment.**—Very little can be said concerning the immediate treatment of spinal birth injuries. In every case of diagnosed or even only suspected injury immobilization is an essential requirement. A spinal puncture by which a part of the effused blood is withdrawn is likely to procure immediate relief of the symptoms, and in a few well-authenticated cases has led to complete and permanent recovery.

## LITERATURE

- Burr. *Am. J. Dis. Child.*, Chicago, 1920, 19:473.  
Goett. *Jahrb. f. Kinderh.*, Leipz., 1909, 69:422.  
Groene. *Zentrabl. f. Gynäk.*, Leipz., 1913, 37:1849.

Hofbauer. *Ibidem*, 1907, 31:354.

Holman. *J. Am. M. Ass.*, Chicago, 1919, 73:1351.

Kooy. *J. Nerv. & Ment. Dis.*, N. Y., 1920, 52:1.

Laffont. *Arch. mens. d'obst. et de gynéc.*, Par., 1919, 8:62.

Lawatschek. *Arch. f. Kinderh.*, Stuttg., 1911, 56:1.

Mueller. *Zentralbl. f. Gynäk.*, Leipz., 1921, 45:550.

Potter. *Am. J. Obst. & Gynec.*, St. Louis, 1921, 1:560.

Sachs. *Ztschr. f. Geburtsh. u. Gynäk.*, Stuttg., 1917, 79:450.

Stoltzenberg. *Berl. klin. Wchnschr.*, 1911, 48:1741.

## CHAPTER VII

### FACIAL PARALYSIS

Central and peripheral origin—Intracranial, cranial and extracranial traumatization of the nerve—Frequency—Symptomatology—Diagnosis — Prognosis — Prophylaxis—Treatment.

From its origin in the nucleus in the pons, the seventh (facial) cranial nerve sweeps around the nucleus of the sixth (abducens) and emerges from the brain with the eighth (auditory) nerve. Together with the latter entering the internal auditory meatus, it passes through the facial canal in the petrous portion of the temporal bone which it leaves at the stylomastoid foramen. From there the nerve runs forward into the substance of the parotid gland, dividing behind the ramus of the lower jaw into the various branches which supply the superficial muscles of the face and the upper part of the neck.

Lesions of the facial nerve, manifested in a facial palsy of the newborn, therefore, may be situated (1) in its supranuclear portion, i.e., somewhere between the cortical motor area and the pontile nucleus; (2) in the cranial center itself; or (3) in the peripheral, lower motor neuron, i.e., somewhere between the center in the pons and the periphery.

The term "central" thus can correctly be applied only to the lesions of group one and two, and the term "peripheral" should imply that the lesion occurred anywhere in the course of the nerve from its nucleus to its peripheral endings.

Proper attention to exact and correct terminology is wanting in much of the obstetric literature dealing with facial paralysis. It seems that most commonly the typical facial paralysis of the newborn is designated as peripheral to express a peripheral injury as its cause. In this same loose manner, obstetricians often speak of central lesions of the facialis when they wish to convey the idea that they are due to intracranial injuries. Obviously all truly central lesions are intracranial, but peripheral lesions of the facial nerve



may be either intracranial or extracranial, and may be due to either a peripheral or an intracranial trauma.

Some excuse for this prevailing inaccuracy of terminology can be found in the fact that our more exact knowledge concerning the semeiology of intracephalic birth injuries is of comparatively recent date. Greater accuracy is requisite for a proper understanding of the problem of facial palsy of the newborn.

**Etiology**—CENTRAL LESIONS.—In rare instances central lesions are not traumatic, but truly *congenital*, representing maldevelopment of some part of the upper neuron or a nuclear aplasia. Some of these cases have been reported as instances of congenital facial paralysis (Fry). According to Vogt—in contrast to some cases of the traumatic bulbar type—in the congenital form the muscles supplied by the upper branch of the facial nerve remain permanently motionless, while the region about the mouth retains either partial or complete mobility. When this difference between the upper and the lower part of the face is great, it results in a characteristic facial expression: full, protruding lips contrasting with the masklike, expressionless face. "The appearance of the face makes the condition often recognizable at the time of birth and gives one the impression of an advanced atrophy or aplasia of the face muscles; also the skin over the muscles is unusually smooth, peculiarly shiny and colorless."

In this connection, however, we are concerned solely with *traumatic central lesions* of the facial nerve in the newborn. They are caused by intramedullary or cortical hemorrhages, and for this very reason are not commonly seen in the living infant. It has been shown in the foregoing pages (Chapter V) that hemorrhages within the substance of the brain seem to occur only in very small, premature infants, or in connection with severe crushing of the skull, which almost without fail will terminate fatally.

A peripheral motor disturbance, confined to the facial nerve, in case of a cortical hemorrhage, would be possible only with strict limitation of the subdural hematoma to the area of the anterior frontal convolution, which is most unlikely to occur. Therefore, in cases of central traumatic injuries a facial palsy, as a rule, will be seen only as one among the other signs of serious intracranial damage.

**PERIPHERAL LESIONS.**—In a discussion limited to the traumatic lesions of the facial nerve sustained during birth, it is essential to differentiate clearly, as already pointed out, between the three portions of the peripheral neuron as the site of the injury.

*Intracranial Traumatization.*—Between its exit from the base of the brain and its entrance into the internal acoustic meatus, the seventh cranial nerve most commonly is affected by a hemorrhage, which, as a rule, originates from a tentorial tear and spreads forward underneath the lower surface of the brain. It has been previously mentioned that under these conditions frequently the twelfth (hypoglossus) and the third (oculomotorius) cranial nerves become involved, a fact of considerable importance for the more exact diagnosis of the character and localization of the intracephalic parturitional injury.

*Cranial Traumatization.*—In its course through the petrous portion of the temporal bone the facialis can be injured only by a fracture of the bone, an uncommon but exceedingly grave injury which practically always causes the death of the infant.

*Peripheral Traumatization.*—In the overwhelming majority of cases the facial palsy of the newborn is due to traumatization of the nerve at or near its exit from the stylomastoid foramen. Most commonly the nerve trunk in this region is severely bruised by a forceps blade. In some cases the nerve seemingly is compressed by a hematoma, which may be found lying in the upper end of the sternomastoid muscle (Schultze, Stein). Under these conditions the facial palsy may be found associated with the signs of a torticollis. A dense edema, probably the result of severe pressure exerted by a forceps blade, seemed to account for the facial paralysis in a case described by Knapp.

The claim has been made that extreme external pressure might cause the complete crushing of the nerve, a fact further discussed later on in this chapter.

Facial palsy is most commonly seen subsequently to forceps extractions. If the forceps, either carelessly or at times unavoidably, are applied to the fetal head in an oblique direction, the posterior blade will come to lie over the infant's ear. This brings the tip of the blade close to the mastoid or parotid region. The mastoid process in the newborn is but poorly developed and thus affords only slight protection to the nerve emerging from the stylomastoid foramen. In a recently published paper Rossenbeck pointed out the fact that usually it is assumed that the facial nerve is injured somewhere between the foramen and the place where it enters the parotid gland. Careful anatomic investigations, however, have convinced him that this seems hardly possible. Within this particular area the facial nerve lies in a deep groove, well covered by muscle, connective tissue, and fat. This groove is formed anteriorly by the angle and ramus of the mandible, and posteriorly by that high

bony ridge which later develops into the thick mastoid process. In no known, or possible, form of forceps application could the tip of a blade reach the nerve just in this location. The nerve is most exposed to traumatization in the parotid itself where all or only some of its branches may be injured. However, the very fact that they are imbedded in soft tissue affords them protection against serious injury. These anatomic findings plausibly explain the fact that not necessarily all the branches of the nerve are involved and that the restoration of function occurs promptly and, with rarest exceptions, is complete.

These anatomic findings, in my belief, are of great importance in accounting for the occasional observation of a *facial palsy subsequent to a nonoperative, spontaneous labor*. The first case of this sort probably has been reported by Kennedy in 1836. Further observations have been put on record by Beriel, Carlsson, Frank, Kehrer, Ludwig, Roulland, Schuetze, Stein, Vogel, and others. In the most recent of thorough studies of the problem, presented by Carlsson in 1912, 34 observations of facial palsies after spontaneous labor are quoted from literature and two new observations added. They presumably occur more often than search through the literature would indicate, because, as already surmised by Kehrer in 1901, the prompt recovery of the child probably leads the observer to the conclusion that the case is not worth reporting.

At present the occurrence of a facial paralysis after a spontaneous labor rather generally is ascribed to pressure exerted by some projecting portion of a contracted pelvis, chiefly of the flat rachitic type, possibly also by an exostosis, or a protruding symphyseal cartilage (Birnbaum-Blackler).

Very few reports specifically assert that the pelvis was roomy and normal in every way. Disregarding for the moment these few exceptions, the etiologic significance of the flat pelvis is evident. A careful analysis of the reported cases shows that apparently only in a small number of instances the nerve is traumatized by direct pressure from the protruding promontory. In the larger number the palsy occurs on the side of the face which during labor is directed towards the symphysis. The exact mechanism by which the infant's facial nerve is injured in the course of a spontaneous labor presumably is the following: the protruding promontory of the contracted pelvis posteriorly interferes with the descent of the head into the pelvic inlet. Through the resulting lateral flexion of the still transverse head an abnormal presentation of the anterior parietal bone is

produced. An ear comes to lie behind the symphysis against which it is firmly pushed during each contraction (Vogel).

A similar situation is created also in the rarer case of a normal pelvis, by an extreme relaxation of the abdominal wall which permits the uterus to fall into exaggerated anteversion. This is well illustrated, e.g., in the observation recorded by Frank. When the head in the course of the labor becomes transversely fixed in the inlet, the excessive uterine antelexion will bend the fetal neck laterally. Therefore, both in the case of an abnormal anterior parietal presentation (anterior asynclitism), and of pendulous abdomen, a forced lateroflexion will cause the fetal shoulder to press strongly against the ear behind the pubes. It seems probable that this shoulder pressure is of etiologic importance in the development of a facial palsy in a spontaneous labor (Bonnaire). The unique observation of Vogel of a typical facial palsy in a child born by cesarean section (osteomalacic pelvis) clearly establishes the fact that the trunk of the seventh cranial nerve might be injured even without passage of the head through the pelvic channel, i.e., as the result of an abnormal attitude of the head at the pelvic inlet.

Since a facial palsy can be seen after a spontaneous labor, it is obvious that in the case of forceps extraction the blame for the injury cannot always be placed on the operation.

It must furthermore be emphasized in this connection that a facial palsy subsequent to a spontaneous and seemingly normal labor, not necessarily is due to a traumatization of the face portion of the nerve. We have also demonstrated in Chapter V that spontaneous labors may cause serious intracranial lesions. Therefore, in some rarer instances a facial palsy after a spontaneous labor actually is the result of the traumatization of the intracranial portion of the seventh nerve. As a good example of this type of injury may be cited a case seen by Stein: the baby, born spontaneously, exhibited a paralysis both of the facial and hypoglossus nerve, the latter evidenced by a deviation of the tongue towards the side on which the face was palsied. A doughy swelling around the upper end of the sternomastoid muscle indicated an external trauma, but the condition of the tongue left no doubt concerning the concomitant intracranial lesion. All symptoms improved rapidly in this case—a fact which emphasizes the necessity of careful observations of all the anomalies exhibited by an infant immediately after birth.

The foregoing discussion of the various etiologic factors re-

sponsible for an injury of the lower neuron of the facial nerve renders the fact apparent that usually the palsy is unilateral. But it also explains the rarer occurrence of a bilateral affection.

Both facial nerves may become involved in one of the following ways: (1) the nerve trunk is injured on one side by the forceps, on the other by a projecting portion of the contracted or otherwise deformed pelvis; (2) both trunks are affected by an intracranial hemorrhage which in the case of a supratentorial basal hematoma may directly compress both intracranial portions of the peripheral neurons, or in the case of a cortical or intramedullary hemorrhage, may on one side, directly, affect the upper or lower neuron, on the other side, indirectly, the intracranial part of the lower neuron as the result of the exaggerated intracephalic tension; (3) there might be a combination of a peripheral and an intracranial traumatization.

**Frequency.**—It is estimated that a functional impairment of the facial muscles of one side is seen approximately in 10 per cent of all forceps extractions. Gans discovered in 562 extractions, made in Winter's clinic, 40 facial palsies, 25 being on the left and 15 on the right side.

**Symptomatology.**—The infant suffering from a paralysis of the facial muscles offers a very characteristic appearance, especially while it is crying: The angle of the mouth is drawn over to the unaffected side; on the affected side the eye cannot be closed (lagophthalmus); on the same side the folds in the skin of the forehead and the nasolabial fold are obliterated, contrasting strikingly with a corresponding deepening of these folds on the sound side. If not all of the branches of the facial nerve are involved, this typical picture is accordingly varied. In some cases the muscles of facial expression are not actually paralyzed but only paretic, and then the existing anomaly might be recognizable only when the child is made to cry.

**Diagnosis.**—No difficulty is experienced in the diagnosis of a typical facial palsy.

From the standpoint of prognosis and therapy, however, it is necessary in every case to establish definitely whether the responsible traumatization has occurred extracranially or intracranially, and in the latter case, whether it is central or peripheral.

The presence of such marks of an external trauma in the region of the mastoid process or of the parotid as a depression, a hematoma

or an edema, are presumptive, but not conclusive, evidence of the traumatization of the nerve at this place.

Simultaneous involvement of other cranial nerves (especially of hypoglossus and oculomotorius) or any signs of intracerebral hypertension suggest strongly an intracranial cause for the facial palsy. Since under these conditions the labor is likely to have been terminated with instruments, in some cases great, and even insuperable, difficulty may be experienced in determining whether the facial paralysis surely is due to the evident external trauma. A careful interpretation of all symptoms, revealed by thorough examination, is essential.

The axones of the facial nerve, as emphasized by Carlsson, which run to the muscles of the forehead and to the orbicularis oculi, are connected with cortical centers of both hemispheres, while all others connect only with the cortex of the opposite side. Injury of the upper neuron in the case of a truly central lesion, therefore, will leave the muscles of the forehead and the sphincter of the eye unaffected, a fact—unfortunately for the purpose of exact diagnosis—often not easily ascertainable in a newborn infant.

While extracranial peripheral lesions lead to a palsy present immediately after birth and improving promptly, intracranial lesions, whether central or peripheral, at times manifest themselves only a short time after birth, and in general tend to become more pronounced, a differential point already duly emphasized in Chapter V.

From the standpoint of differential diagnosis, it is noteworthy that in some well-authenticated cases such a delayed facial paralysis of a newborn child was unmistakably caused by a nasal diphtheria presumably acquired during birth from an infected vagina. In a systematic search, Kirstein discovered diphtheria bacilli in three instances on the first day of life.

Again, in connection with the cases of facial palsy noticeable immediately after birth, it must be remembered that at times, though very rarely, the anomaly of the face is truly congenital, due to a nuclear aplasia, maldevelopment of the os petrosum with occlusion of the auditory meatus (mentioned by Carlsson), or caused during intra-uterine life by the pressure of an amniotic band against the cheek of the fetus (Olshausen, Geyl). In the cases of a congenital nuclear aplasia, the palsy apparently tends to be bilateral. Koester (quoted by Kehrer) observed this condition in two brothers, and

Ballantyne mentions Thomas as having seen familial congenital palsy with distinct aplasia of the face muscles.

**Prognosis.**—The prognosis is very favorable for all peripheral extracranial lesions. Improvement, as a rule, sets in immediately and continues from day to day, with complete restoration of function usually attained within the first two weeks of life. In some instances the signs of, at least a slight, paresis may continue up to the fourth and sixth week. The palsy but rarely impairs the infant's ability of sucking to such a degree that nutrition is seriously interfered with.

Observations of permanent palsy appear only in older contributions in which obviously more stress is laid on the extracranial injuries and a possible intracranial lesion, congenital or traumatic, is but rarely considered. Therefore, rather generally a failure of functional restoration of the face muscles has been ascribed to a complete crushing of the nerve by the forceps blade, an accident which is not likely to occur in view of the newer anatomic findings of Rossenbeck.

In all cases of intracranial traumatization of the facial nerve, the prognosis rests entirely upon the nature of the lesion.

**Prophylaxis.**—Though undeniably a traumatization of the facial nerve by the forceps cannot positively be avoided in every instance, even by the most expert operator, there cannot be any doubt that it is more prone to happen when the instrument is handled by a less skilled physician. At least in some instances such an injury is prevented by the careful application of the forceps exactly across the transverse diameters of the head, whenever this is possible—granted the forceps is properly constructed. Too strong a cephalic curve in many instruments on the market reduces the distance between the tips of the blades, thus causing them to dig unnecessarily deep into the skull. This same effect might be produced with a properly constructed instrument, properly applied on an abnormally large head, if during the extraction the somewhat separated handles are forced against each other. This the experienced obstetrician will avoid. An approach of the separated handles can be obviated by placing a folded towel between them. Instruments which permit a fixation of this separation by means of a special set screw between the handles in this respect offer a distinct advantage.

**Treatment.**—The typical facial palsy of the newborn, caused by an external injury, practically always disappears without any special

therapeutic attention. In those cases in which the symptoms continue for more than two weeks, the affected muscles may be stimulated prophylactically with the galvanic current to prevent their secondary atrophy and a, possibly permanent, slight asymmetry of the face.

In a case of lagophthalmus it is well to protect the eye against traumatization and infection by means of a moist boric acid pack.

### LITERATURE

- Ballantyne. *Antenatal Pathology and Hygiene*, Edinburgh, 1902.  
 Beriel. *Abstr. in Zentralbl. f. Gynäk.*, Leipz., 1907, 31:1522.  
 Carlsson. *Ibidem*, 1912, 36:1508.  
 Frank. *Ibidem*, 1901, 25:509.  
 Fry. *J. Am. M. Ass.*, Chicago, 1920, 74:1699.  
 Gans. *Monatschr. f. Geburtsh. u. Gynäk.*, Berl., 1908, 27:430.  
 Kehrer. *Zentralbl. f. Gynäk.*, Leipz., 1901, 25:1082.  
 Kirstein. *Ibidem*, 1918, 42:821.  
 Rossenbeck. *Ibidem*, 1921, 45:981.  
 Souchon. *Abstr. Ibidem*, 1909, 33:1717.  
 Stein. *Ibidem*, 1905, 29:321.  
 Vogel. *Monatschr. f. Geburtsh. u. Gynäk.*, Berl., 1900, 12:609.  
 Idem. *Ztschr. f. Geburtsh. u. Gynäk.*, Stuttg., 1903, 48:474.  
 Vogt. *Handb. d. Neurol.*, Berl., 1911.



## CHAPTER VIII

### INJURIES OF THE FACE

Injuries—Face—Skin—Nose—Mouth—Lower jaw—Masseter muscle—Ear.

#### SKIN

Red spots and linear streaks on the face of the newborn, usually in locations determined by the mechanism of the labor, represent pressure marks and pressure grooves due to disproportion between head and pelvis in some diameter. In rarer instances they are produced by an exostosis, or by the horizontal ramus of the pubis, or in a funnel-shaped pelvis by the ischial spines.

Deeper depressions of the skin and cuts through its entire thickness down to the underlying bone are made by the tip or lower edge of a forceps blade, if this edge is unnecessarily sharp, and especially, if it was necessary to rotate the head with the forceps. Such forceps indentations, and pressure marks, if the contact with the protruding bone ridge was of long duration, may become gangrenous. After elimination of the necrotic area, a scar forms which usually is permanent.

#### NOSE

If the forceps have to be applied to an unrotated head, standing transversely high in the pelvic inlet, the tip of one blade is likely to lie very close to the base of the nose. Fractures of one or both nasal bones will occur under these conditions and must be regarded as unavoidable accidents. That a nasal fracture may occur in a spontaneous labor has been proved by an observation of Olshausen (quoted by Kuestner).

As the most protruding feature of the face, writes Sluder, the nose is exposed to injury far more than the rest of the face, and probably during birth the nose is often injured despite its elastic, pliable consistency. This seems the only etiology for the dislocation of the anterior part of the septum from the wings of the pre-

maxillary bones, giving rise to a deflected septum in patients who have never sustained an injury.

## MOUTH

Mouth injuries of some sort are fairly common. Unfortunately in many instances, particularly of severer traumatization, the blame lies squarely with the attending physician. The most common source for such injuries seemingly is the Mauriceau-Veit-Smellie method of delivery of the aftercoming head. If the one or two fingers introduced into the baby's mouth are improperly employed for the purpose of extraction, the resulting injuries, according to reports in literature, seem to range between laceration of the mucosa in one corner of the mouth to the complete evulsion of the lower jaw. Literature contains many records of serious injuries of the tongue and of mandibular fractures. Ruge (quoted by Kuestner) saw a deep tear of the genioglossus muscle on one side of the tongue. Strassmann, as cited by Reuss, observed a fatal hemorrhage from a torn frenum of the tongue.

Such injuries can be avoided if the obstetrician will only remember that in the course of the extraction of the aftercoming head the fingers in the infant's mouth serve solely the purpose of maintaining flexion.

Mouth injuries of the newborn are known in forensic literature as occasional findings in cases in which the mother gave birth to a child without the help of a physician or midwife. Instinctively she might attempt to hasten the painful moment of expulsion by hooking a finger into the mouth of the delivered head to pull out the rest of the body. Obviously, by such an effort it is more likely the roof of the mouth and the palate that will be traumatized, a point of medico-legal importance.

However, traumatization of the palate more commonly, and indeed quite frequently, is due to unnecessary roughness in clearing the mouth and pharynx from aspirated mucus. A piece of coarse dry gauze employed for this purpose more often than generally suspected will account for bruises and excoriations on the hard palate and tongue. Their frequent presence will be surprising to every physician who will look for them whenever a baby seems reluctant or refuses to suck on the nipple. Injuries of the hard and the soft palate, including lacerations of the latter, have been observed after

the use of aspirators equipped with stiff hard rubber or metal tips. All such injuries can be effectively avoided by the use of moist cotton and solely of soft rubber catheters for the removal and aspiration of mucus from the pharynx. Thus speaking of resuscitation, mention may be made here of injuries of the tongue caused by the Laborde method of rhythmic tractions of the tongue. Slight sugillations and excoriations of the tip of the tongue are unavoidable, while all serious traumatisms of the base of the tongue, as recorded in literature (Peronne), must be attributed to lack of care or skill. The question whether tongue injuries, sustained at birth, may account for certain speech defects, has been answered in the negative at least by Knapp.

Haberda (as quoted by Stumpf) reported two cases of severe laceration of the soft palate and pharynx walls caused by very rough manipulation in clearing the throat of asphyxiated newborn infants. He emphasized in this connection the forensic importance of such an injury in its differentiation from injuries produced by the stuffing of soft materials down the baby's throat in cases of infanticide.

### LOWER JAW

Fractures, dislocations and even complete evulsions of the mandible have been reported as the direct result of unskilled, one might say for certain cases, of brutal traction made in the Veit-Smellie maneuver. A separation of the two portions of the lower jaw in the midline has been specifically ascribed to an asymmetric pull on the jaw in a lateral direction in the attempt to obtain a complete rotation of the after-coming head.

Fracture as the result of direct pressure by a forceps blade occurs rarely, but its possibility is well exemplified, e.g., in the case of Tissier. The mandible was found broken on the right side between the midline and the angle, exactly at the point where the instrument had left a very deep skin impression.

### MASSETER MUSCLE

Hofstaetter described a hematoma in the masseter muscle as a typical birth injury seemingly so far not mentioned in medical literature. The pathology in all four cases was the same. Soon after birth a small, well-circumscribed mass was discovered lying within the masseter muscle of one side, easily palpable from without and

also from the interior of the mouth. As ascertained by puncture, it was a hematoma which did not seem to give rise to any noticeable discomfort and gradually disappeared by resorption.

This traumatic swelling must be differentiated from the following conditions which have been observed in newborn infants: inflammatory processes in the parotid, including syphilis and tuberculosis; retention cyst due either to inflammatory occlusion or to obstruction, by a concretion, of Steno's duct; and new growths, benign and malignant, of the parotid gland.

All 4 of Hofstaetter's observations were made in infants born in breech presentation, 2 of them after version. The condition is ascribed by him to a severe stretching of one masseter, when with the finger held in the baby's mouth to maintain flexion, strong lateral pressure is exerted against the inner surface of the cheek for the purpose of rotating the head completely.

Since this hematoma apparently does not affect the child, it does not demand therapeutic attention. A cold pack during the first few days might prevent a further extravasation of blood, and hot applications later on will possibly hasten resorption.

## EAR

Serious injuries of the external ear, as a rule, are caused by a slipping forceps and, therefore, in general are avoided by care and skill. Complete avulsion of the auricle, as recorded by Erskine, is rare. In this case the entire ear was torn off by the sudden and unexpected slipping of the forceps, and later had to be replaced by an artificial auricle.

## LITERATURE

- Erskine. *Brit. M. J.*, Lond., 1902, 1:14.  
 Hofstaetter. *Beitr. z. Geburtsh. u. Gynäk.*, Leipz., 1911, 16:332.  
 Knapp. *Scheintod des Neugeborenen*, Vienna, 1904.  
 Kuestner. *Encykl. d. Geburtsh. u. Gynäk.*, Leipz., 1909.  
 Sluder. *Headaches and Eye Disorders of Nasal Origin*, St. Louis, 1917.  
 Stumpf. *Winckel's Handb. d. Geburtsh.*, 3:3, p. 622.  
 Tissier. *Bulletin de la Soc. d'Obstétrique de Par.*, December 16, 1909.

## CHAPTER IX

### INJURIES OF THE EYE

**Definition**—Injuries of eyelids and orbit—Functional anomalies of the external muscles expressed in nystagmus, strabismus, and paralysis of the external rectus—Luxation and avulsion of the eyeball—Crushing of the eyeball—Corneal injuries—Hemorrhages into the anterior chamber—Retinal hemorrhages—Injuries of optic nerve—Visual defects in later life.

Under the term parturitional injuries of the eye there must logically be included the injuries of the eyelids, of the lacrymal ducts, of the orbit, of the external eye muscles, and of the various structures of the eyeball, which occur as the direct result of traumatism incident to normal and pathological labor. Therefore, the statement, often met in obstetrical writings and also in textbooks of obstetrics, that birth injuries of the eye are extremely rare, is both incorrect and misleading. If Bruno Wolff was able to find in the records of the Charité Hospital (Berlin) only 6 cases of eye injuries mentioned in a total of 39,317 deliveries, this fact solely proves that but very serious and most obvious injuries had been considered worth mentioning in these history records.

Ophthalmic literature is not wanting in reports of such injuries. To the 112 cases collected by Wolff from this literature Goldwasser in a more recent survey of the literature, up to 1914, was able to add another 132 observations. But even this seemingly imposing number of 244 reported cases certainly does not adequately express the actual incidence of parturitional damage to the infant's eyes. It is generally conceded that in approximately 10 per cent of cases a forceps extraction is followed by lagophthalmus. By an examination of the eyes of newborn infants within the first twenty-four hours of life, Lequeux discovered a disturbance of some sort in 25 per cent. He enumerates the anomalies in order of their respective frequency as: paralytic conditions of muscles, corneal lesions and retinal hemorrhages. Paul asserted that a systematic ophthalmoscopic examination of newborn babies revealed evidences of a retinal hemorrhage in 50 per cent of the infants born through a contracted

pelvis, in 40 per cent of premature infants, in 40 per cent of protracted and difficult labors, and in 20 per cent of normal-sized babies born spontaneously after normal labors. Such figures hardly justify writers to assert that eye injuries are rare.

In Chapter V, I had occasion to point to the advantage of a routine examination of the eyeground in the early diagnosis of intracranial birth lesions. Such a routine, admittedly feasible only for a large and well-equipped maternity, would probably also offer an opportunity of clearing up the etiology of many disturbances in eye function manifesting themselves only later in life. This problem is aptly stated by Thomson and Buchanan: "The term congenital is frequently applied to various conditions of the cornea, choroid, retina, and eye muscles, because we are yet in ignorance of the true cause of these defects; but it is our opinion that many of these may be due to the traumatism of birth."

The indifference of the obstetrician towards the problem of parturitional eye trauma up to recent years is quite evident. While the results of systematic studies of the eyeground of the newborn have been recorded in ophthalmologic literature from time to time ever since Jaeger's first investigations, in 1861, the interest of the obstetrician in eye injuries incident to labor seems to have been aroused only by Wolff's splendid presentation of this problem in 1905. Many single observations and etiologic studies have since appeared also in obstetric literature. The latest systematic and exhaustive survey of the entire literature, based on a total of 246 cases, was presented by Goldwasser.

Runge in his excellent book dealing with the "Relations of Gynecology and Obstetrics to Ophthalmology" (1908) divided the parturitional injuries of the eye into those seen subsequently to spontaneous labors, and those undoubtedly due to the artificial means employed in the termination of labor. Such an etiological classification does not seem practical. As a matter of fact, a traumatization of the eyes is more commonly observed after labors terminated by artificial means, chiefly after forceps extractions, but the very serious injuries, such as luxation or complete avulsion of the bulbus are rarely directly connected with the actual delivery and more generally due to unskilled and rough vaginal examinations made in the course of the labor by attending physicians and more often by midwives.

A regional arrangement of all the known lesions, as adopted here, offers the opportunity for a discussion not only of the etiology

of each individual type of lesion, but also of their possible prevention—one of the chief general aims of this volume. If not otherwise indicated in the appended bibliography, the papers and cases mentioned in the following pages are cited from Runge's and Goldwasser's comprehensive monographs.

## INJURIES OF THE EYELIDS

*Edema, suffusions and suffusions* of the skin surface of the lids and of the conjunctiva palpebrarum are commonly seen in cases of face and brow presentation when the caput succedaneum involves the region of the eyes. They disappear readily without treatment.

I shall not enter into a detailed consideration of the problem of a *conjunctivitis* of the newborn subsequent to the instillation of a silver solution immediately after birth in accord with the suggestion of Credé. In general this undesirable sequela can be avoided by the use of fresh solutions. Cramer warned against the prophylactic instillation after labors in face or brow presentations. Hemorrhages from the conjunctiva, as mentioned in literature, seem invariably to have been caused by rubbing of the lids after the instillation, which is not in accord with a proper technic. I reported a fatal conjunctival hemorrhage after correct Credé treatment with a 2 per cent solution of silver nitrate, observed in a full-term child born spontaneously in vertex presentation.

The lids or their immediate surroundings often are injured by the tip of the forceps. Strong pressure may cause an *ecchymosis* or small hematoma to form in the lid. Cones saw, after a forceps extraction, a *hematoma* in the upper lid which in his opinion stood in direct etiologic relation to a cephalhematoma on the parietal bone of the same side.

Unique is the observation of Saemisch (quoted by Goldwasser) of an *angioma cavernosum* of the conjunctiva developed subsequent to, and as he assumed, as the result of, a forceps extraction.

Whenever the suffusion of the eyelids is very pronounced, one should think of a possible deeper injury to the orbit itself.

An actual laceration of the lid, followed by scar contraction accounted in the case of Steinheim for an *ectropion* of the upper lid, in the case of Truc for an ectropion of the lower lid. Schmidt-Rimpler noticed a secondary ectropion of the lower lid developing after the fracture of the upper jaw by the forceps.

A *lagophthalmus* occurs in about 10 per cent of forceps extractions, from a facial paralysis.

Reese ascribed a *contraction of the palpebral fissure*, associated with enophthalmus and myosis, to traumatization of sympathetic nerve fibers by the forceps.

A *ptosis* of the upper lid, caused by paralysis of the oculomotor nerve, has already been mentioned in the symptomatology of intracranial hemorrhages (Chapter V).

*Laceration of the lacrymal duct* was reported by Peters, in this case accompanied by a facial palsy and corneal opacities, all representing forceps injuries.

## INJURIES OF THE ORBIT

In Chapter IV, I have described fractures of the frontal bone, as a rule the result of a high forceps extraction in which the instrument unavoidably had to be applied to an unrotated head. Through direct pressure by the apex of one blade the supra-orbital margin, the lacrymal bone, or the roof of the orbit may be fractured in this manner. In other instances a basal fracture may extend posteriorly into the orbital surface of the frontal bone, and then will involve the various fissures and foramina through which the optic, abducens and other nerves, and blood vessels are passing to and from the eyeball. In the majority of instances such severe injuries cause the immediate death of the child.

In surviving infants the immediate and later symptomatology of traumatization of the orbit comprises: changes in the eyelids, such as edema, suggillations or hematomas, already described; functional anomalies of the eye muscles expressed in the lack of coördinate movements; exophthalmos; and a great variety of lesions in the interior of the bulbus, usually noticed only later in life in the form of deficient vision, if not ascertained by an ophthalmoscopic examination immediately after birth.

Among these various symptoms *exophthalmus* becomes one of particular significance in the diagnosis of orbital lesions, because disturbances in coördinate eye movements, as will be shown later, often are discovered in the newborn only accidentally. The pathology of this exophthalmus has been thoroughly studied by Levy and Hintzy on newborn infants and in experiments. Levy asserts that careful examination will reveal at least a slight exophthalmus in a large number of babies extracted with the forceps. This is the result of a temporary strangulation of the ophthalmic veins in the upper orbital



fissure, causing an engorgement of the veins in the retrobulbar space. A contraction of this fissure is likely to occur whenever the forceps are applied in an oblique direction. If some of the congested veins rupture, a retrobulbar hematoma forms. Both Levy and Hintzy conclude that for this reason, subsequent to a forceps extraction, a pronounced exophthalmus may develop with or without an orbital fracture. In other instances the exophthalmus may be absent despite a fracture of the orbital roof. The blood, extravasated behind the eyeball, they explained, may fail to press the latter forward, if a hole in the roof allows for its partial escape into the space opened up by the fracture. Among the 244 cases of serious eye injuries collected from literature by Wolff and Goldwasser there were 28 orbital fractures, and 30 instances of traumatic exophthalmus, both writers excluding from this number the cases of actual luxation of the eyeball.

In some rare cases the bulbus has been found partially pushed out of the eye socket by a fragment of the fractured orbit. They all terminated fatally.

## FUNCTIONAL ANOMALIES OF THE MUSCLES OF THE EYEBALL

A marked *nystagmus* is occasionally seen in intracephalic hemorrhages (see Chapter V). However, great care must be exercised in the interpretation of this symptom. Rhythmic lateral movements of one or both eyes are commonly observed in seemingly normal newborn babies. It is an open question whether or not this nystagmus is the expression of a physiological traumatization of the brain as the result of the compression of the skull during molding. More acceptably this phenomenon seems explained by a lack of that perfect balance of synergistic and antagonistic muscle function essential for coördinate motion of both eyes. The external eye muscles at birth are not fully developed and only after several weeks are controlled voluntarily by the child.

*Strabismus* in the newborn is always due to extreme weakness, actual paralysis or anatomic defect, but never, as may be the case in the adult, to overaction or contraction of an eye muscle.

On account of the evident weakness of the eye muscles at birth, and as the result of the infant's inability to focus on an object, an

apparent alternating or periodic squint is fairly frequent within the first few days of life.

A true squint may be evident immediately after birth, but often is noticed only accidentally when the eyes happen to turn into the direction into which the one bulb cannot follow on account of the paralysis of an eye muscle, most commonly of the external rectus. This fact is of great importance in the differentiation of a paralysis developing only a few days after delivery as the result of a slowly progressing intracranial hemorrhage, from one actually existing at birth but overlooked until turning of the eyes into a certain direction reveals the heretofore unsuspected inability of the one eye to follow its fellow.

The involvement of the oculomotorius in cases of intracephalic lesions has been amply discussed in Chapter V. Disturbances of eye movements caused by oculomotor lesions are complex and, under the special conditions and difficulties met in the newborn, rarely permit of accurate interpretation.

Much more obvious and of great interest to the obstetrician is the fairly frequent internal squint due to the paralysis of one of the external recti muscles innervated by the abducens.

*A paralysis of the external rectus*, one may conclude from extremely scanty statistical information offered in ophthalmologic literature, represents the cause of approximately one-half of those cases of squint, seen by ophthalmologists, in which the impaired mobility of an eye is caused by the palsy of but one of the nerves controlling eye movements (Nettleship).

In looking for enlightenment concerning a possible relation of abducens palsy to a parturitional trauma, one will discover that text books of ophthalmology usually limit themselves to the bare statement that in many cases this defect is congenital, and that textbooks of obstetrics, seemingly without any exceptions, entirely fail to mention this condition. This clearly points to indifference or lack of information concerning any possible etiological relation of the palsy to birth. As far as I was able to ascertain, only the paper of Bloch, published in 1891, and still frequently quoted, deals specifically with the problem. Of 438 cases of abducens paralysis seen in Hirschberg's eye clinic, Bloch classified 31 as congenital because the defect was known to have existed since birth. Three of them he claimed to represent birth injuries, for no other reason than that these three children were delivered by means of forceps. He reasoned that

there were three possible types of birth trauma which might account for a paralysis of the external rectus muscle:

1. A laceration of the muscle by the instrument, which does not seem likely without serious damage to the eye itself;
2. Injuries of the abducens nerve in the orbit, which to him appeared to be the most plausible origin of such a palsy; and,
3. Possibly intracranial injuries which directly affect the nucleus of the abducens.

The assertion of DeWecker or Scrinì that forceps extraction and difficult labor in the presence of a contracted pelvis are of significant etiologic importance was not based on conclusive clinical or anatomic evidence. It clearly is unjustifiable to quote Bloch in support of this contention; three forceps extractions for 31 cases of abducens palsy noticed since birth. Bloch's figures, however, seem to express with fair accuracy actual conditions. Every obstetrician with a large personal experience knows that he has seen this palsy very often after perfectly normal and spontaneous labors—and most likely has considered it of so little importance and interest that he has not even noted the fact on his records. There are apparently no statistical reports extant from large maternity services concerning the incidence of a paralysis of the external rectus after normal or abnormal labors.

This is another problem the satisfactory solution of which rests with the obstetrician.

The abducens has the longest course of any of the cranial nerves and, in the adult, is frequently affected by basal lesions. We may assume that in connection with the common tentorial lacerations—which, as I have shown in Chapter V, occur also in spontaneous labors—a small amount of the extravasated blood may reach the intracranial portion of the abducens. The involvement thus may be slight and but temporary. As a matter of fact, an internal strabismus noticed at birth in many instances disappears gradually, especially at the time when eye movements begin to become voluntary and coördinate, proving that the primary functional impairment of the external rectus was not of a serious nature.

A palsy of the external rectus muscle, temporary or permanent, may follow the traumatization of the abducens in the sphenoidal fissure through which it enters the orbit. In speaking in the foregoing pages of exophthalmus, I referred to the experimental studies of Levy which tend to establish a narrowing of this fissure during

an asymmetric compression of the skull chiefly as the result of application of the forceps in an oblique direction. Assuming that Levy's observations are correct, it is justifiable to conclude that such a narrowing of the upper orbital fissure might occur under certain conditions also in a labor ending spontaneously (in spite of mechanical disproportion between head and pelvis), and that in this manner not only veins but also the abducens is compressed and traumatized.

A simultaneous palsy of both the abducens and facialis nerves of the same side is not rarely seen after forceps extraction. Spitz (quoted by Goldwasser) thought that this combination probably was due to traumatization (by hemorrhage) of the nuclei of both nerves which lie closely together in the pons. This might happen but perhaps is the exception. Both nerves more easily are injured peripherally, as well illustrated in a recent personal observation. Immediately after a fairly high forceps extraction, necessitated by unusually advanced ossification of the cranial bones, the fresh, not asphyxiated baby showed a complete left facial paralysis and a palsy of the left external rectus muscle, also pronounced nystagmus. Most careful observation during the next few days, however, failed to reveal any signs of an intracranial lesion. At the end of the fifth week the baby began to follow light and both eyes moved coördinately far out to the left. At that time the facial palsy had greatly improved with a paretic condition still noticeable on crying. In this case the lesions of both nerves undoubtedly were peripheral. The facial palsy was evidently due to severe forceps pressure. The abducens palsy could not have been due to direct trauma of the rectus muscle, because the blade was not lying over the eye. There were no skin lesions immediately around the orbit, no conjunctival changes, edema or hemorrhage, to indicate traumatization of the eyeball itself. Presumably the abducens was compressed in the sphenoidal fissure. In this case the instrument unavoidably had been applied obliquely.

Personally I am of the opinion that a paralysis of the external rectus muscle, present at birth, is more likely to be of traumatic origin than truly "congenital," i.e., due to deficient intra-uterine development of muscle or nerve. Further investigations into this problem will have to take into account the fact, now well established, that organs and structures of the infant are injured also in the course of normal and spontaneous labors.

## LUXATION AND AVULSION OF THE EYEBALL

Luxation and avulsion of the bulbus represent the most serious types of parturitional eye injuries. In the case of a *luxation* the eye has been forced through the palpebral fissure and thus comes to lie in front of the lids, which are tightly contracted behind it. In an *avulsion* the eyeball is torn out of the orbit. It sometimes is found still hanging on the socket by one or the other of the muscles or by mere shreds of tissue; in other instances the separation is complete. Thus in a case reported by Zangarol (cited by Friedenwald) the missing eyeball was finally discovered among the bed linens, and in a case observed by Goldwasser, in the placenta.

This subject has been thoroughly covered more recently in a paper by Friedenwald, in which he presented brief histories of 29 instances of this kind recorded in literature, and added the detailed description of a personal observation.

A variety of causes can be found to account for this horrible mutilation of the child:

1. The attending physician, and more often a midwife, mistook a brow or face presentation for a breech and injured the eye in the attempt to explore the anus for the purpose of confirming the diagnosis. Practically all cases of avulsion will be found to belong in this group. In general they evidence inexcusable and, therefore, avoidable roughness during examination.

However, it has been claimed by Rothenspieler and Birch-Hirschfeld that under certain conditions, even if the pressure is not excessive, the bulb might be wedged through the palpebral fissure if the pressure is exerted in a lateral direction around the edge of the eyeball. This is a point of great importance for the medicolegal expert in a possible action for malpractice against the attending physician.

2. In an identical manner the luxation may be produced by the tip of a forceps blade lying unfortunately so that it dips on one side under the periphery of the eyeball. This particular accident seems to be very rare and can occur only in a high forceps application.

3. In cases of fracture of the orbit, perhaps always caused by a forceps, bone fragments or a very large retrobulbar hematoma may push the eyeball completely from the orbit. In such a case, described by Maygrier, a bone fragment seemed to have cut off all connections

of the bulb to the eye socket. In a case of Gad the optic nerve was severed in this manner.

4. There are some rarer reports of luxation of the eye after normal and spontaneous labors. It would be impossible to determine in which of these cases the injury, after all, might have been due to manipulations. Bugge saw a luxation after the quick expulsion of a hydrocephalic child and ascribed the accident to the abnormal softness of the skull bones. Goldwasser quoted the following interesting observation of Fage: The child was born with a simple luxation of one eye. On the day before labor the mother had sustained a blow against her abdomen by the tongue of a wagon, severe enough to throw her over on her back. Since labor was perfectly normal, it seems possible that in this instance the luxation of the bulbus was caused by the external trauma. Most writers quote a unique case reported by Hofmann in which two successive children of the same mother were born with complete avulsion of one eye. The first delivery was managed by a midwife. It ended spontaneously in a left vertex presentation. When the head, supported by the midwife, was expelled, the right bulbus fell into her hands. It was hanging over the infant's cheek attached to the orbit solely by the partially torn inferior rectus muscle. Several doses of ergot had been administered by the midwife to overcome an inertia. A later examination revealed a sharp-edged promontory jutting deeply into the pelvis. Hofmann assumed that the eye was torn out by the promontory, over which it had been quickly pushed by the strong uterine contractions excited by the ergot. During the next labor he was forced to apply forceps. While unwinding the cord, lying tightly around the neck, the right bulbus slipped into his hand. Presumably also in this labor the avulsion was produced by the promontory and not by the forceps.

The prognosis of a luxation or avulsion in each case is dependent on certain conditions. In cases of extensive fracture of the orbit, as a rule, the infant succumbs to the coincident intracranial traumatization.

In cases of *avulsion* immediate enucleation of the eye most likely will result in the recovery of the infant. Mere replacement of an avulsed eye into the socket is prone to lead to panophthalmitis, which will terminate in a phthisis bulbi or will necessitate later enucleation. In some instances the infant died from a general septicemia.

The merely *luxated* eye has been successfully replaced, as well illus-

trated in Friedenwald's case. To avoid traumatization of the eye, and, in most cases, to make replacement actually possible, it becomes necessary to enlarge temporarily the palpebral fissure by an incision at the outer canthus (external canthotomy). Before a reposition is attempted in this manner, it is essential to look for and remove all fragments of bone from an orbital fracture.

## INJURIES OF THE EYEBALL

**Crushing of the Eyeball.**—The bulbus may be crushed, ruptured, or only flattened by direct pressure. Reports of this injury are not numerous. Spaeth (quoted by Berger) and Berger himself saw complete crushing of both eyes, caused by a high forceps; in the case of Spaeth this was associated with extensive fracture of the nose. In a case of Steinheim one eye was completely destroyed by the forceps blade, resulting finally in the death of the infant. Peck found one eye flattened by a hematoma; later a cataract developed in this eye. Other observations of this kind have been mentioned by Finkelstein, Wolff, Wulsten, and others. In case of recovery from the severe insult, the eye, as a rule, will exhibit some permanent defect, such as cataract, corneal opacities, etc.

**Injuries of the Cornea.**—The question of parturitional traumatization of the cornea has been most thoroughly studied by Thomson and Buchanan, and the entire literature dealing with this type of injury was more recently compiled by Goldwasser.

Corneal injuries apparently are relatively common. There were 31 instances of corneal opacity or injury among the 112 cases collected by Wolff, and 12 (including keratoconus) in the 132 observations considered by Goldwasser.

*Opacities* appear in a diffuse form or in the shape of streaks. If diffuse they vary greatly in density. At times the entire cornea is involved, in some cases only the center or periphery is opacified. In the majority of instances only one cornea is affected, though bilaterality is not exceptional. Most frequently the opacities are the result of an edema and infiltration, and are then prone to be diffuse and milky.

If due to a direct trauma, the opaque area more likely is long and narrow. In the latter case the corneal change often is found associated with other evidence of traumatization, such as fracture of the

supra-orbital arch with a tear running through the midst of the cornea into the sclera, finally leading to a phthisis bulbi (case of Cramer); diffuse corneal opacities with exophthalmus and abrasions on the forehead (Pick).

In their anatomic studies Thomson and Buchanan discovered the opaqueness and inflammatory infiltration to be lying in the substantia propria of the cornea often with coincident *tears in the membrane of Descemet*. Breaks in the latter probably account for permanent thin linear or ribbonlike (striate) opacities. Peters (according to Goldwasser) saw extensive laceration of this membrane and, in one case, its partial detachment and curling into the anterior eye chamber, an occurrence also noticed by Thomson and Buchanan. Rupprecht compares this laceration of Descemet's membrane without a concomitant lesion of the peripheral layers of the cornea to the isolated splitting of the tabula interna in some cranial fractures. Pressure against the center of the convex surface of the cornea stretches in particular the concave membrane of Descemet as the result of the effected flattening.

While disappearing quickly in most instances when the result of an edema, the corneal opacities may fade out gradually (in a case of DeWecker within three months) or become permanent, though rarely. Fine scars after direct injury with the forceps may result in a distortion of the cornea leading to a complicated astigmatism as seen by De Schweinitz, Steffenson, Thomson and Buchanan, and others.

*Corneal ulcerations* have been recorded as immediate sequelae of instrumental injuries (Thomson and Buchanan). They show a tendency to heal very slowly. Lack of proper care of the exposed eye in cases of lagophthalmus has been followed by the development of a corneal ulcer. Only rarely the corneal destruction from an ulceration is extensive enough to terminate in a complete leucoma of the cornea (seen by Truc in a child four years old).

The exceptional observation of corneal opacities immediately after spontaneous, though at times protracted, labors has been explained by Sidler-Huguenin on the basis of general circulatory disturbances and chiefly of congestion in various portions of the head, and in particular by the sudden influx of blood into the eye immediately after the expulsion of the head. In his opinion, in this manner temporarily a condition of glaucomalike hypertension is created in the bulbus. Other writers, e.g., Berger-Loewy, and



Wagenmann, are inclined to accept Thomson's claim that also subsequent to spontaneous labors such corneal opacities more likely are due to traumatization of the eye. In a deformed pelvis some protruding bone ridge might exert undue pressure against the eyeball, while it passes over this prominence during the expulsion stage.

A large keratoconus noticed by De Schweinitz (also by Stock) in association with diffuse cloudiness was ascribed to an edema of the cornea.

**Hemorrhages into the Anterior Chamber.**—In some of the comparatively frequent cases of internal hemorrhages of the eye, a part of the extravasated blood escapes into the anterior chamber. In some other instances the hemorrhage seems limited to the anterior chamber. The condition is readily ascertained by mere inspection of the eyes of the newborn. The presence of blood in the anterior chamber is satisfactorily explained as of traumatic origin in cases of extensive external injuries, especially of orbital fractures, of lacerations of the lids, etc.

Difficulty is experienced in accounting for such hemorrhages in the eyes of infants born without operative aid. Under these conditions they have been ascribed to excessive congestion in the infant's head. Such an etiology is strongly suggested, e.g., in a recent observation of Voorhees: the child was born with its cord coiled tightly six times around its neck; the intense congestion and discoloration of the face was striking; the conjunctiva of one eye was suffused and blood was noticed in its anterior chamber. This hemorrhage was resorbed slowly and left a permanent opacity still visible at the age of six years.

It will be shown later that a congestion of certain veins plays an important part in the causation of hemorrhages in other structures of the bulbus, especially of the retina. The strongly congested vessels might conceivably be injured by a trauma not necessarily excessive.

The blood in the anterior chamber may originate from a *laceration of the iris* or of the ciliary body. A case carefully studied by Wintersteiner thus showed free blood in both anterior chambers, and hemorrhages in the ciliary body, iris, between the ciliary body and sclera, in the subarachnoidal and episcleral tissue. In a stillborn infant, extracted with forceps in a case of contracted pelvis, Mizuno observed a meningeal hematoma, and in addition, subarachnoidal hemorrhages and pronounced hyperemia of the iris and ciliary body,

and dilation of the veins in the retina and optic nerve—in my opinion a most instructive illustration of the close relation of intracranial birth trauma to lesions within the eyeball. A tearing of the iris with extravasation of blood into the anterior chamber, subsequent to a forceps extraction, was described by Bylsma. However, defects in the iris might be truly congenital, and still be found associated with other traumatic eye injuries, as presumably was the case in a newborn baby seen by Hippel. There was a circumscribed defect in the iris, a complete detachment of the retina and a subretinal hematoma.

*Luxation of the lens*, as the result of direct pressure of the apex of a forceps blade, probably is very rare. It was discovered in one instance by Thomson and Buchanan. The lens was lying in the vitreous body, the integuments of the bulbus were intact.

In this connection may be mentioned the possibility of a typical *traumatic cataract* either in the interior of the lens or in its anterior capsule, observed after, and presumably the result of, forceps extraction (among others, two cases reported by Posey).

**Retinal Hemorrhages.**—In Chapter V, I have dwelt in detail on certain retinal changes ascertained by ophthalmoscopic examination of the eyegrounds in newborn infants. They have been discussed in that connection chiefly from the viewpoint of their apparent relation to intracranial hypertension, and of their importance in the diagnosis of intracranial birth hemorrhages. I shall, therefore, in this connection consider them rather in regard to their direct relation to parturitional traumatization of the eye itself and their probable relation to some of the visual defects of later life, still customarily classified as congenital.

The question of retinal edema, congestion, hemorrhage, and detachment is most thoroughly elucidated in the paper of Goldwasser.

Systematic studies of the eyegrounds on large numbers of newborn infants leave no doubt concerning the frequency of retinal hemorrhages. They have been ascertained, e.g., by Schleich in 32 per cent, by Paul in 34.5 per cent of children examined in this manner. They are likely to be discovered in a much larger number of instances if the examinations are made within the first twenty-four hours, because in many cases the changes are slight and disappear quickly. The retina of both eyes was found involved in 29 out of 49 cases examined by Schleich, in 23 of 65 studied by Sicherer. In an anatomic study of the eyes of newborn infants Naumoff dis-

covered pathological conditions in 25 per cent, the majority of them being retinal hemorrhages. A still higher percentage is given in a pathologic-anatomic investigation of Coburn, made on the eyes of 37 infants, either stillborn or dying within the first three days. He found 17 cases of retinal hemorrhage (46 per cent), 13 subretinal and 4 subhyaloidal hematomas. It seems obvious that the anatomic study of the eyes of infants, stillborn or dying within a few days, will yield a relatively higher percentage of retinal hemorrhages than the ophthalmoscopic examination of living infants. In a considerable number of those available for anatomic investigation, death probably was caused by serious cranial or intracranial injuries. As amply explained in foregoing pages such traumatic lesions frequently result in changes within the eye.

Histologic research has demonstrated that these hemorrhages are situated more commonly in the posterior portion of the retina near the macula, and that they are most extensive in the layers of the nerve fibers and ganglion cells.

Larger hemorrhages are readily seen by the ophthalmoscope in the form of hemorrhagic spots, at times occupying the entire eye-ground, or running in a radial direction along larger vessels. The minor changes due to small hemorrhages and to congestion have been described in detail in Chapter V.

Various theories have been advanced to explain their causation. Koenigstein's theory of an active hyperemization of the eye with the first cry now seems obsolete. Most of the modern writers incline to the belief that retinal hemorrhages, when not obviously produced by a direct insult to the bulb, are due to an excessive congestion of certain vessels during labor. Naumoff assumed that during the compression of the head in the molding process some of the cerebrospinal fluid is pressed into the subvagal (intersheath) space of the optic nerve, and there causes a strangulation of the vena centralis retinae. Such a widening of the subvagal space, however, has been denied by Hippel, who, indeed, found this space to be very narrow or even entirely absent in some cases of retinal hemorrhage. He failed to discover any anatomic condition to explain the predilection of the region of the papilla and macula as seats of these hemorrhages. Thomson and Buchanan, on the other hand, found no traces of retinal hemorrhage in some cases in which the bulb undeniably had been exposed to severe pressure and, therefore, they assumed an etiologic importance of increased fetal blood pressure due to

interference with cord or placental circulation during intra-uterine life. This explanation has been refuted by Stumpf, Sicherer and Schleich, who emphasized that the freshness of the extravasated blood proves that the hemorrhage is of natal and not of prenatal origin. Paul blamed strong compression of the jugular vein by a tight coiling of the cord around the neck. But he was answered by Stumpf, who found retinal hemorrhages in only 14 per cent of cases in which the cord was around the neck, but in 24 per cent without any cord anomaly. Naumoff's findings of fresh hemorrhages in infants, who had died during labor, evidently justify his conclusion that retinal hemorrhages are truly natal, neither prenatal as claimed by Thomson and Buchanan, nor immediately postnatal as explained by Koenigstein.

The still irrefuted theories of Stumpf and Sicherer, and Schleich of the origin of the congestion of retinal vessels in the course of labor have been fully presented in Chapter V.

An analysis of this entire literature strongly suggests that, like in the origin of intracranial hemorrhages, also in the etiology of retinal hemorrhages, we probably will have to differentiate between direct mechanical factors and certain predisposing causes.

The notorious thinness of the walls of the retinal blood vessels is often mentioned by writers. But it seems that the fact that a child, exhibiting a retinal hemorrhage, was premature often is only incidentally mentioned by a writer, chiefly to emphasize that this small head could not have been subjected to excessive compression during its passage through a normal pelvic canal (e.g., a case of Thomson and Buchanan). The evident vulnerability of all vessels within the cranium of a premature and small infant, is to-day of recognized significance in the occurrence of intracranial hemorrhages and might also be found of importance in the origin of retinal hemorrhages. Paul placed the incidence of retinal hemorrhage for premature infants at the strikingly high figure of 40 per cent. Stumpf, apparently laying more stress on the luetic infection, proved an apparent predisposition of the premature, congenital luetic infant for retinal hemorrhage.

Wolff pointed to the possible or even probable relation of these ocular hemorrhages to asphyxiation, the latter, in his opinion, being evidenced by the frequency of concomitant ecchymoses in other organs of the infant. Does this not possibly indicate that a hemorrhagic diathesis of the newborn is of a still unrecognized importance

in the origin also of retinal hemorrhages? Might not retinal hemorrhages stand in the same relation to a hemorrhagic tendency of the newborn as now established at least for some of the intracerebral hemorrhages, namely, that they represent only the local expression of the anomalous coagulability of the blood? Further investigations along these lines would seem promising and might be expected to add greatly to our present insufficient knowledge concerning the origin of the retinal hemorrhages which undeniably occur in many infants born after normal labors.

It seems unessential for the purpose of this volume to quote here occasional observations of choroidal, subchoroidal and other hemorrhages mentioned in the exhaustive reviews of the literature by Runge and Goldwasser.

**Injuries of the Optic Nerve.**—Mention has been made in this chapter of complete severance of the optic nerve in cases of avulsion of the eyeball, or in instances of orbital fracture by a sharp splinter of bone. It has been shown that under such conditions the optic nerve usually is torn some distance from the bulb, e.g., approximately one-half inch in the cases of Wicherkiewicz, Gueniot and Gad, one inch in the case of Snell.

The optic nerve is necessarily also traumatized by a luxation of the eyeball. However, in some cases of operative restoration, vision later has been found normal, if not disturbed by corneal opacities.

Literature contains several records of atrophy of the optic nerve, observed in young children and presumably standing in relation to a difficult labor, usually terminated by forceps. In some of the cases evidences of injury around the eye strongly suggested an etiologic connection to traumatization at birth. In a case of Muehsam's, a girl of seven years with partial atrophy of one optic nerve, amblyopia with contraction of the visual field, the history revealed a difficult forceps extraction, followed by pronounced swelling of the one eye. Naumoff saw a bilateral optic atrophy in a child eighteen months old apparently blind since birth. Labor had been terminated by means of difficult forceps extraction in this case. Sidler-Huguenin reported a left-sided complete atrophy of the optic nerve with scar formation in the papillomacular region in a woman of forty-nine years, who told him that when she was born her eye was hanging out of its socket. Similar observations have been recorded by Nordquist and Pihl (unilateral optic atrophy, empyema of sinus frontalis with deep forceps scars on the forehead), by Sidler-Huguenin (atrophic dis-

coloration of both papillae, convergent strabismus and nystagmus subsequent to a forceps extraction which consumed three hours), and others.

**Visual Defects.**—Visual defects, as a rule, can be ascertained only later in life. Of interest to the obstetrician, therefore, is only their possible relation to injuries sustained in birth. I have cited in the foregoing pages many instances in which corneal opacities, cataract, retinal hemorrhages, optic atrophy, etc., were evidently due to definite injuries of the bulbus, usually inflicted in the course of instrumental deliveries. In many other cases such a relation of visual defect to a birth trauma is only problematical, often only assumed by the ophthalmologist on account of a history of difficult or instrumental labor. Berger-Loewy considered it as not improbable that in cases of "congenital" amblyopia the structural changes in the region of the macula at times are due to retinal birth hemorrhages. Partial retinal detachment and abnormal pigmentations have been explained in this manner when there was no other acceptable explanation available. Partial atrophy of the choroid in the form of a coloboma of the macula has been ascribed to intra-ocular hemorrhages *intra partum* (Naumann).

As already emphasized in the introductory remarks to this chapter, a systematic study of the eyegrounds of all newborn babies, which could be easily done in all well-equipped maternity services, would certainly throw much light on the problem of the etiology of many of the more obscure ocular lesions which to-day still are classed as congenital. A proper understanding of their relation to mechanical elements during birth and to certain predisposing factors within the infant might well be expected to result in their prevention in many instances.

#### LITERATURE

- Bloch. *Zentralbl. f. prakt. Augenh.*, 1891, 15:134.  
 Bugge. *Abstr. in Zentralbl. f. Gynäk.*, Leipz., 1908, 32:29.  
 Ehrenfest. *St. Louis M. Rev.*, April 25, 1903.  
 Friedenwald. *Am. J. Ophth.*, St. Louis, 1918, ser. 3, 1:9.  
 Gad. *Abstr. in München. med. Wehnschr.*, 1908, 53:834.  
 Goldwasser. *Brit. z. Geburtsh. u. Gynäk.*, Leipz., 1914, 19:365.  
 Hintzy. *Abstr. in Zentralbl. f. d. ges. Gynäk. u. Grenzgeb.*, Berl., 1914, 5:281.

- Jardine. *J. Obst. & Gynec. Brit. Emp., Lond.*, 1903, 3:530.
- Kearney. *Am. J. Obst., N. Y.*, 1917, 70:904.
- Lequeux. *Ann. de gynéc. et d'obst., Par.*, 1911, 8:740.
- Levy. *Ibidem*, 1913, 10:561.
- Naumoff. *Arch. f. Ophth., Berl.*, 1890, 36:180.
- Paul. Quoted by Seitz: *Zentralbl. f. Gynäk., Leipz.*, 1912, 36:1.
- Runge. *Geburtshilfe und Gynäkologie in ihren Beziehungen zur Ophthalmologie, Leipz.*, 1908.
- Serini. *Gaz. d. hôp., Par.*, 1901, 74:492.
- Stumpf and Sicherer. *Beitr. z. Geburtsh. u. Gynäk., Leipz.*, 1909, 13:408.
- Thomson and Buchanan. *Ophthalmological Society's Transactions (London)*, XXIII, Abstr. in *J. Obst. & Gynec. Brit. Emp., Lond.*, 1904, 5:367.
- Voorhees. *Am. J. Obst., N. Y.*, 1916, 74:299.
- Wolff. *Die Augenverletzungen des Kindes bei der Geburt, Leipz.*, 1905.

## CHAPTER X

### INJURIES OF THE NECK

Forceps trauma—Forensic importance of strangulation marks—Traumatization of the parathyroid glands.

Contusions, suggillations, and furrows visible on the neck of a newborn, dead infant are of significant importance especially in forensic medicine. There are three possible sources for their origin.

(1) The tip of a forceps blade may reach up to the neck. This is more likely to occur in cases of slight or complete extension of the head. The typical sharp-edged skin depression or abrasion will be readily recognized, usually situated on one side below the ear or lower jaw. If the instrument is applied to an unrotated head in face presentation, the end of the one blade may come to lie over the larynx. A unique injury caused in this manner has been recorded by Kouwer. The patient was a ix-para. Before admission to the hospital futile attempts had been made to extract the infant, lying in a face presentation, by means of the forceps. In the hospital the malpresentation was corrected manually and the child extracted in a vertex presentation. It was deeply asphyxiated. All efforts to start respiration failed, though the heart continued to beat regularly for some time. At the post-mortem examination a marked suggillation and edema was discovered in the larynx exactly at the spot where from the outside it had been compressed by the tip of the blade during the preceding forceps attempts. The infant evidently had succumbed to an acute edema of the glottis. Kouwer closed his report with the valuable advice, to think of such an injury of the larynx under these conditions; because tracheotomy certainly would have saved this infant.

(2) In speaking of injuries of the face in Chapter VIII, I had occasion to refer to suggillations below the jaw. In some instances they have been seen as a result of the efforts of a mother, delivering herself in the absence of an attendant, to pull out the rest of the child by pushing against the lower jaw of the already born head (Hohl



as cited by Halban). The medicolegal expert may be called upon to differentiate this type of injury from another produced in the attempt to kill the newborn.

(3) During the effort to kill the infant the mother or an accomplice is likely to grab the neck firmly from the front. Therefore, under these conditions, suggillations, as a rule, will be found on either side of the neck and hemorrhages also in the deeper tissues. If at the same time, with the infant's head lying against a resisting base, severe pressure is exerted in a backward direction, the larynx is also prone to be injured. This combination of severe traumatization of the larynx with bilateral suggillations not only in the skin, but in underlying tissues, apparently represents the almost characteristic picture at autopsy of the newborn killed by strangulation with the hand.

In other cases, many of them mentioned in forensic literature, a handkerchief or rope had been used for the purpose. The strangulation mark then will run around the entire neck, and post mortem findings in general will be practically identical with those seen after hanging. In some authentic observations the newborn had been deliberately strangled by means of its own cord. A cord tightly coiled several times around the neck, however, may cause the child's death during labor.

Two points seem of value in differentiating between such unavoidable and accidental death and the deliberate strangulation of the born baby with the cord. According to Liman-Skrzetzka (quoted by Stumpf) deep, visible furrows are formed in the neck by the cord coiled around it only after the child is dead. Excoriations of the skin could be produced only in the deliberate strangulation with the cord.

In some cases a test of the lungs for air will help to decide the question. In the instances of tight coiling of the cord the infant most likely was stillborn, and the lungs will be found in a state of complete atelectasis. On the other hand, in instances of infanticide it will but rarely have been possible to effect the strangulation so quickly that the infant had no chance of breathing before it was killed.

Brief reference has been made in Chapter V to a possible *traumatization of the parathyroid glands* in birth. This question is discussed in detail by Reuss. The occasional appearance of symptoms of tetany in young infants suggested the possibility of a parturitional injury of

the epithelial bodies, because their artificial traumatization in animals was known to lead to tetany-like symptoms. Erdheim (1904), as the first, had called attention to the frequent presence of hemorrhages in the parathyroid glands of newborn infants and considered them to be caused by deep asphyxiation. In 1908 Yanase advanced further arguments in favor of this theory by discovering in 37 per cent of 89 infants, dying at various ages, remnants of extravasated blood or typical histological changes pointing clearly to a hemorrhage in the epithelial bodies during early postfetal life. Though also in his opinion, deep asphyxiation most likely represented the cause for the hemorrhage, he acknowledged that in some instances the parathyroids were found normal in the presence of characteristic ecchymoses of asphyxiation in pleura and pericardium. Negative findings in the parathyroid glands of children dying with the typical symptoms of tetany have induced most pediatricians to repudiate the theory of a parathyroid traumatization as the underlying cause of this disease. Reuss contends that such negative findings in older children do not preclude the possibility that a parturitional injury of the epithelial bodies create a definite predisposition in the child to develop a tetany only much later, when all local evidences of the trauma might have disappeared.

#### LITERATURE

- Halban. *Real-Encykl. d. ges. Heilk.*, 3d Ed., 12:281.  
Kouwer. *Zentralbl. f. Gynäk.*, Leipz., 1901, 25:771.  
Kuestner. *Encykl. d. Geburtsh. u. Gynäk.*, 2:444.  
Reuss. *Die Krankheiten des Neugeborenen*, 1914, p. 305.  
Stumpf. *Winckel's Handb. d. Geburtsh.*, 3:3, p. 623.

## CHAPTER XI

### INJURIES OF THE STERNOMASTOID MUSCLE

Pathology—Etiology—Frequency—Symptomatology and diagnosis—Prognosis—  
Prophylaxis—Treatment.

The term *torticollis* (*caput obstipum*, wry-neck) connotes a characteristic attitude of the head caused by a shortened sternomastoid muscle. The head is drawn forward and towards the shoulder of the affected side while at the same time it is rotated so that the chin points towards the opposite side.

Rather commonly *torticollis* is mentioned as one of the more important and frequent types of birth injuries of the child. This statement, at least in this form, is incorrect in view of the fact (1) that seemingly in the overwhelming majority of instances a wry-neck, noticeable at birth, represents a truly congenital anomaly and not a parturitional injury, and (2) that only exceptionally a traumatization of the sternomastoid muscle during birth finally leads to a *torticollis*. As a matter of fact, most often it is a congenitally malformed sternomastoid muscle which is injured during birth. Therefore, we can speak in connection with birth trauma not of *torticollis*, but only of injuries of the sternomastoid muscle.

**Pathology.**—Two pathologic-anatomic anomalies of this muscle, observed in the newborn, must be clearly differentiated.

(1) There is a shortened and rigid muscle which on microscopic examination is found to contain an abnormal amount of fibrous tissue with a corresponding reduction of muscular elements. The fibrous degeneration involves more or less the entire muscle and does not represent a typical scar within a limited area.

(2) A fairly well-circumscribed hematoma, varying in size between a hazelnut and a pigeon egg, is found in a sternomastoid muscle, which either is otherwise normal in every respect, or exhibits the already described fibrous degeneration. The hematoma is formed by the extravasation of blood from a partial or complete laceration of the muscle.

**Etiology.**—The first references to wry-neck, according to Sippel, can be found in the medical literature of the end of the seventeenth century. In 1668 and 1680 Van Roonhuysen, as the first, pronounced the shortening of the sternomastoid muscle to be congenital, and he corrected the deformity by cutting the shortened muscle. In 1830 Dieffenbach still expounded practically the identical view. He felt certain that the *caput obstipum* developed during intra-uterine life and had no connection to birth itself. Stromeyer (in 1839) announced his theory of the injury of the muscle in birth, and his idea remained the prevailing one for many decades. In 1884 Peterson reverted to the original conception that a developmental anomaly actually is responsible for the abnormal attitude of the head. Mickulicz (1895) suggested that severe pulling might tear any muscle while in the state of firm contraction. The hematoma resulting from this injury may become infected and a *myositis fibrosa* terminate in a permanent shortening of the muscle. This seemed plausible to Koester (1897). Strong traction would in birth naturally endanger those muscles which are contracted in premature efforts of respiration during a state of asphyxiation. Since healing of a traumatic tear of no other muscle is known to result in a pronounced and permanent shortening, he was willing to accept Mickulicz's (and Kader's) idea of a postnatal traumatic *myositis fibrosa*. Koester also expressed doubt whether the swelling at times palpable in the sternomastoid of the newborn always actually represents a hematoma and not only an infiltration. As a matter of fact, such swelling in most instances disappears without leading to later disturbances in the function of the muscle.

Quite recently (1921) Meyerding considered a trauma of the sternomastoid muscle, sustained at or preceding birth, a most important etiological factor in congenital torticollis. He ascribed the shortening to a chronic interstitial myositis, the result of an ischemia which is brought on either by compression of the sternomastoid branch of the superior thyroid artery, or by a hematoma into or around the sheath of the affected muscle.

From time to time the suggestion had been made that an intra-uterine inflammatory affection of the sternomastoid had caused its shortness and fibrous degeneration manifest at birth. In 1905 Pincus expressed the opinion that a prenatal myositis accounts only in the minority of instances for a congenital *caput obstipum*. Couvelaire (1911), on the other hand, asserted that an intra-uterine myo-

pathia with subsequent degeneration often represents the underlying cause for the hematoma discovered in the sternomastoid muscle immediately after birth.

Voelcker (1901), from an exhaustive study of the etiology of congenital torticollis, had arrived at the conclusion that the shortening of the sternomastoid is the result of a degenerative process during intra-uterine life, the result of direct pressure of the uterine wall, if the head is held in a lateral flexion, and insufficient protection against this pressure is caused by a scarcity of amniotic fluid.

Kehrer (1907), in a critical analysis of Voelcker's work, refused to accept this theory, coming from a surgeon, until verification had been obtained for the assumption that a fetus will maintain this faulty attitude of the head for a considerable time during pregnancy. This seemed to him improbable, particularly in view of the acknowledged frequency of breech labors in cases of congenital caput obstipum. Stumpf already had emphasized that the roomier fundus portion of the uterus certainly would offer to the head of the fetus a better opportunity for unhindered movement than the narrower lower uterine segment.

The proof demanded by Kehrer that the fetal head does maintain for a long time an abnormal attitude, now seems to have been most satisfactorily furnished by Sippel. He recently presented in two papers (1920 and 1921) the results of his exhaustive investigations concerning positions of the fetus, made by means of roentgenograms obtained by the method of Warnekros. In a lecture given before the Gynecological Society of Berlin, Sippel demonstrated many pictures (not yet published) which showed a typical torticollis distortion of the neck in feti lying in transverse and breech presentations. Several pictures taken in succession on the same patient irrefutably established the fact that at times this anomalous attitude of the head persists. Such direct observation of the fetus *in utero* together with the examination of the infant immediately after birth and, in some instances, followed by the histological study of excised portions of a shortened sternomastoid muscle, have enabled Sippel to bring the much-disputed question of the etiology of congenital wry-neck close to its final solution.

As soon as the fetus fills the uterine cavity, which is about in the fifth or sixth month of pregnancy, it is subjected to the effects of direct pressure by the uterine wall if the amniotic fluid is scanty. All the mechanical factors required for the development of a con-

genital torticollis now are present. Voelcker and Zedel actually have described typical examples of this deformity in fetuses, respectively six and seven months old, and also Sippel saw this condition in a seven months' fetus.

If, as the result of crowding, the fetal head continuously is held in a position which shortens the distance between the points of attachment of the sternomastoid, the muscle is prevented, and may fail, to grow to its normal length. It seems possible that the sternomastoid branch of the thyroid artery will participate in this underdevelopment or is prevented by mechanical conditions from supplying the muscle with the required amount of blood. The restriction or actual hindrance of head movements by limitation of space causes an atrophy of the already developed muscle, a fibrous degeneration and shrinkage of the sternomastoid. In this process often also the deeper muscles and fascias of the neck become involved. Fascia changes probably account for the failures in the attempts of correction of the deformity by redressment later in life.

In regard to the traumatization of the congenitally shortened and weakened muscle during birth, Sippel accepts the views repeatedly advanced by older writers. Traction is prone to tear such a muscle more or less completely, and probably the force sufficient to result in the injury is small. One can readily assume that in this manner merely a torsion of the head may cause the laceration. However, Sippel is of the opinion that in many cases a hematoma in the shortened muscle does not indicate an actual tearing of tissue. During labor, especially in a vertex presentation, a shortened sternomastoid muscle is exposed to strong pressure by the shoulder held in close approximation as the result of the shortening. In this way the muscle can be severely bruised.

Undeniably the sternomastoid muscle more often after breech extractions than after vertex labors is found injured, but this fact does not prove the prevailing erroneous contention that breech labors play an important rôle in the causation of congenital torticollis. Maldevelopment of the sternomastoid, in the main due to definite mechanical conditions within the uterine cavity, forces the fetal head permanently into an attitude which often prevents its normal adaptation to the pelvic inlet. Therefore, a congenital torticollis is not the result, but rather the cause, of breech presentations.

Sippel agrees that also the normal sternomastoid muscle might

be torn, but only by extreme force greatly exceeding the amount of traction permissible in any correct technic of obstetric manipulations. Reports of apparent traumatic rupture of a normal muscle comparatively often state that the injury was bilateral. This is well exemplified, e.g., in an observation reported by Hildebrand. In this case the head was firmly fixed in the pelvis and presumably first one and then the other sternomastoid muscle was torn by the difficult rotation of the shoulders required in the effort to bring the arm backwards for freeing.

A case of bilateral torticollis, described by Morse, however, clearly sustains the Voelcker-Sippel contention of maldevelopment as the underlying cause. This baby was delivered at the very onset of labor by means of a cesarean section. Immediately after extraction it was noticed that the head was held in partial extension by the shortened sternomastoid muscles. This attitude presumably had been maintained for some time before labor. The deformity disappeared gradually within four months.

The frequency of the combination of a congenital torticollis with other evident malformations of the infant, apparently most often a spina bifida, seems to support the maldevelopment theory. I saw a wry-neck, very marked immediately after birth, in a hydrocephalic child, born spontaneously in a normal vertex presentation.

Summarizing the information at present available concerning the etiology of traumatic injuries of the sternomastoid muscle in birth, the following statements can be made:

(1) In the majority of instances the injured muscle is congenitally malformed, abnormally short, weak and more or less degenerated.

(2) The developmental defect is perhaps always due to an anomalous attitude of the head, maintained for a long time during intra-uterine life as the result merely of mechanical conditions.

(3) The resulting typical deformity of the neck seemingly favors a breech presentation.

(4) Comparatively slight traction suffices to lacerate more or less completely the abnormal muscle. In some instances such a muscle probably is traumatized during labor by the pressure of the shoulder which lies close to the muscle on account of its shortening.

(5) A normal sternomastoid muscle may be torn in delivery, but only by excessive traction.

**Frequency.**—Meyerding, in a recent paper (1921), certainly was not justified in asserting that congenital torticollis is a very rare condition because it was seen only in 26 of 212,000 patients examined in Mayo's clinic. Sippel found 13 cases of torticollis (8 on the right, and 5 on the left side) subsequent to 8 labors in breech, 4 in transverse and 1 in normal vertex presentation in a series of 3179 deliveries which included 100 transverse, 276 breech, and 56 foot presentations. Sippel's figures well express the generally conceded higher frequency of wry-neck subsequent to breech labors. This same fact is also to be noticed in the statistics of five observers (quoted by Stumpf) who in a total of 114 cases of torticollis, present at birth, recorded 71 breech labors.

It may be well to emphasize that this information concerning the incidence of congenital torticollis obviously does not express the frequency of birth traumatism of the sternomastoid muscle, the question with which we are here concerned.

According to Stumpf, in the Obstetric Clinic of Munich, one hematoma of the sternomastoid was discovered approximately for every 7477 deliveries. After vertex labors hematomas seemed to occur practically only when forceps had been applied. However, in reexamining 232 children, delivered by forceps, Engelken a few years later was able to discover only 1 child with a torticollis.

**Symptomatology and Diagnosis.**—Sippel's work, definitely confirming the more or less theoretical assumptions of many preceding investigators, renders it necessary to alter the prevailing custom, and to omit the characteristic torticollis attitude of the newborn as a symptom of parturitional injury of the sternomastoid muscle.

A traumatization of this muscle during birth will be indicated by a localized swelling, in most instances representing a hematoma. This hematoma might be the result of an actual laceration or only of a severe bruising of the muscle. The mass, when small, occasionally is hidden for the first few days by a diffuse edema. Most commonly it lies at the outer edge of the muscle, is movable with the muscle, and apparently is not tender to pressure.

Since in most instances a tear is but partial, a hematoma will be found (1) in combination with the typical wry-neck deformity whenever a congenitally abnormal muscle has been traumatized and (2) with a normal attitude of the head if the injured muscle is otherwise normal.



In the rare instances of a complete laceration, unlikely to occur except when the muscle is extremely short and weak, the traumatic severance of the muscle actually may completely correct the deformity if the maldevelopment does not extend to the deeper muscles and fascias of the neck.

Theoretically one could assume that in the case of a complete tear the intact muscle of the other side, by overaction without an antagonist, might turn the head in such a manner as to suggest an existing shortness of the muscle on the intact side. I was unable to find any recorded observation of this occurrence. This situation, however, will always be difficult to determine in the newborn whose head, in the absence of all voluntary control, falls from one shoulder to the other as the infant is turned, or forward, when the child is held up.

**Prognosis.**—Injuries of the sternomastoid muscle at birth, as a rule, are not of noteworthy significance to the child. As has been shown in foregoing pages, the injury, with but rare exceptions, involves a congenitally abnormal muscle. The hematoma, caused by laceration or pressure, is gradually resorbed. Laceration of a congenitally shortened muscle might actually improve the condition, either temporarily or permanently. In cases in which the shortening of the muscle is slight, the deformity often disappears spontaneously or, at least, becomes unnoticeable, especially in short-necked children.

It remains an open question whether a torticollis may develop as a direct sequela of a birth injury of the sternomastoid muscle, in cases in which the hematoma had become infected, and when a myositis fibrosa finally may cause a permanent scar contraction of the muscle. Pincus assumed that in some instances the swelling in the muscle, noticed immediately after birth, is not a hematoma but an infiltration, indicating the beginning myositis.

**Prophylaxis.**—Traumatization of a weak and shortened sternomastoid muscle cannot be prevented either during the various manipulations required in the management of a breech presentation or in a forceps extraction.

The injury of the normal muscle generally is ascribed to excessive force employed in rotating the shoulders against an aftercoming head fixed firmly in the pelvis. Schauta mentioned as particularly favorable for such an injury the fixation of the unrotated head in an

anteroposterior direction. If this situation is not recognized by the operator, the shoulders are actually rotated fully 90 degrees.

In view of the fact, generally conceded, that a normal sternomastoid muscle could be torn only by most severe traction, I doubt whether the evident traumatization of the newborn, under such conditions as are mentioned in the preceding paragraph, actually consists in an injury of the sternomastoid muscle.

It is striking that practically identical mechanical factors are mentioned in literature as the direct causes also of injuries of the cervical portion of the vertebral column (confer Chapter VI). Recently Feil (1921) directed attention to an ankylosis of the atlas to the occipital bone (*occipitalisation de l'atlas*), causing in the adult a condition that can be differentiated from a typical torticollis solely by a roentgenogram which will show a bone shadow between the diffused atlas and occipital bone. Naturally the question suggests itself whether this final ankylosis, at least in some cases, is not the result of a severe traumatization of the atlanto-occipital articulation, particularly after a difficult extraction of the aftercoming head. A systematic roentgenographic examination of the cervical region of all newborn infants, exhibiting seemingly a congenital torticollis or an injury of the sternomastoid muscle, might occasionally reveal a unilateral subluxation or other injury of this articulation, and thus help to clear up some of the still obscure points in the causation of torticollis.

**Treatment.**—Parturitional injuries of the sternomastoid muscle require no specific therapeutic attention. The treatment of torticollis in the older child properly belongs to the surgeon or orthopedist. To the obstetrician the fact will prove interesting that, in the belief of Sippel, the congenital caput obstipum should be surgically corrected when the infant is four to five weeks old.

## LITERATURE

- Couvelaire. *Ann. de gynéc. et d'obst.*, Par., 1911, 68:1.  
Engelken. *Nederl. Tijdschr. v. Geneesk.*, Amst., 1920, 2:1538.  
Feil. *Presse méd.*, Par., June 29, 1921, p. 515.  
Hildebrand. *Deutsche Ztschr. f. Chir.*, Leipz., 1897, 45:584.  
Kehrer. *Beitr. z. Geburtsh. u. Gynäk.*, Leipz., 1907, 11:179.  
Koester. *Deutsche med. Wchnschr.*, Berl., 1895, 21:117.

- Meyerding. *J. Orthop. Surg.*, 1921, 3:91.  
Morse. *Surg. Gynee. & Obst.*, Chicago, 1915, 20:74.  
Pincus. *Zentralbl. f. Gynäk.*, Leipz., 1905, 29:618.  
Sippel. *Deutsche Ztschr. f. Chir.*, Leipz., 1920, 155:1.  
Idem. *Ztschr. f. Geburtsh. u. Gynäk.*, Stuttg., 1921, 84:225.  
Stumpf. *Winckel's Handb. d. Geburtsh.*, 3:3, p. 502.

## CHAPTER XII

### INJURIES OF THE CLAVICLE

Pathology—Etiology—Frequency—Symptomatology and diagnosis—Prognosis—  
Prophylaxis—Treatment.

Most frequent among parturitional injuries of the infant's shoulder girdle are the fractures of the clavicle. It was the common belief that these fractures occur only in breech labors until Riether, in 1902, called attention to their frequency in spontaneous vertex labors. The thorough investigations of Muus (1903) and Hauch (1905), implying an active search for this injury in every labor case, not only confirmed Riether's findings but demonstrated that a clavicular traumatization is more common in vertex than in breech labors, and undoubtedly more often is overlooked than actually diagnosed by the attending physician.

**Pathology.**—The clavicle is broken transversely, either straight across or in an oblique direction. Usually the lesion is subperiosteal, and situated in the middle third or between the middle and outer third of the bone. A mere infraction is not infrequent. Only exceptionally both clavicles are found fractured. Comparatively rare is a luxation in the sternoclavicular articulation, or an epiphyseal detachment at the sternal or acromial end.

**Etiology.**—The clavicle is traumatized, directly or indirectly, under the following conditions:

(1) During extraction of the aftercoming head. If in the Veit-Smellie maneuver the index or middle finger, forked around the neck, presses too forcibly against the clavicle, it may break at the point of pressure, which will be approximately in the middle. This mechanism corresponds exactly to that of a direct fracture of the clavicle in the adult, resulting from a blow striking the diaphysis in a perpendicular direction.

(2) In the management of a breech labor the clavicle can be fractured also indirectly. If strong pressure against the humerus is required to bring the arm down, this pressure may be indirectly

transmitted, along the shaft of the humerus, to the clavicle. Its sternal end being firmly fixed, this force will cause the clavicle to bend and possibly to break. In Kuestner's belief, this is the more common mechanism for the fracture in a pelvic end presentation. Such an injury of the clavicle is comparable with the clavicular fracture of the adult sustained in a fall on the extended arm. In the infant the clavicle under these conditions is prone to break at its weakest point, i.e., between its middle and outer third.

(3) It is assumed, but not definitely proved, that a clavicle may be fractured indirectly, both in vertex and breech labors, by the excessive compression of the shoulder girdle in its passage through a narrow pelvic outlet. During manual help in a breech labor this noxious compression might be brought about by the introduction of the operator's hand along the shoulders which are firmly crowded into the pelvis. In general this mechanism would correspond to the clavicular fracture of the adult produced in a fall against the shoulder.

Muus, in presenting this last theory, felt that in no other manner could be explained the striking frequency of clavicle fractures after normal, spontaneous vertex labors. Pulling on the head by means of the forceps or during the extraction of the shoulders, would obviously increase this compression of the shoulder girdle, and thus satisfactorily explain the higher incidence of fractures under these conditions. He saw 5 fractures in a series of 100 vertex labors which were deliberately managed in such a manner that at no time was any traction exerted on the head, or pressure against the shoulders (in the protection of the perineum). These fractures, in his belief, then must have occurred during the passage of the shoulders through the pelvis.

(4) Hauch is unwilling to accept this explanation of Muus. If correct, clavicular fractures would be extremely common in instances of contracted pelvis, which is not the case. Delbecque reported entirely negative results from his experiments of producing clavicular fractures artificially by forcibly pulling normal-sized fetuses through an equally contracted pelvis. Both Muus and Hauch established the marked prevalence of an injury to the clavicle which in labor passes anteriorly through the pelvic outlet. From this fact, the latter deduced that the deciding factor in the causation of the fracture is the pressure of the anterior shoulder against the symphysis, which, as already emphasized by Riether, is augmented by

a pressure against the posterior shoulder in the effort to protect the perineum. Muus had established a frequency of 1.5 per cent of clavicular fractures for all deliveries. A reduction to 0.67 per cent in his service, Hauch ascribes to a radical change in the customary technic of managing the delivery of the shoulders. The entire personnel of the clinic was prohibited under any conditions, to exert downward traction on the head for the purpose of bringing the anterior shoulder under the symphysis or to hook a finger into the anterior axilla for the purpose of lifting the posterior shoulder over the perineum. All assistance was restricted to support of the perineum. However, Hauch was forced to the conclusion that not even this technic can entirely eliminate the occurrence of a clavicular fracture. The scapulae are very movable, and, when reaching the pelvic outlet, are pushed forward. The anterior scapula, caught behind the symphysis, may exert strong pressure in a perpendicular direction against the clavicle, causing it to bend beyond its limit of flexibility. With the release of the shoulders the scapulae spring back into their normal position.

The apparent prevalence of clavicular fractures in the infants of multiparous mothers, Hauch concedes, is not satisfactorily accounted for by his explanation of the mechanism, but if Muus' theory were correct, he insists, fractures should be more common in the primipara in whom also the rigid pelvic floor strongly compresses the passing shoulder girdle.

Strong traction on the arm in the management of a breech labor may injure the clavicle (Rielaender). It is under these conditions that the sternal epiphysis of the clavicle may be detached or, as in the adult, the sternoclavicular articulation be luxated.

The clavicle may be only fractured, or broken underneath the intact periosteum, which usually will hold the fractured ends in good apposition. Therefore, it is the rule that there is no visible dislocation or even palpable deformity.

**Frequency.**—Weltmann recorded in 29,500 deliveries in the Breslau clinic 24 clavicular fractures. He quoted among others the following statistics: in the clinic in Halle they discovered 2 clavicular fractures in 3982 deliveries (including 182 breech labors); Foesterling reported 19 fractures and 1 luxation at the sternal end in 6171 deliveries made in an out-door service (including 989 breech labors). The unreliability of these figures becomes evident by a comparison with the newer statistics published since Riether directed attention

to the frequency of these fractures in the course of normal labors in a vertex presentation.

Carefully examining 1700 living children, born in vertex presentation, Muus discovered 22 fractures of the clavicle, which represent an approximate incidence of 1.5 per cent. All were unilateral. Of 22 mothers, 9 were primiparous, 13 multiparous, which actually means that fractures were twice as common in the latter group, because the ratio between primiparous and multiparous in the service of the clinic was found to be 135 to 100. In the 18 cases in which the exact mechanism of labor could be ascertained, 15 times it was the anterior, and only 3 times the posterior clavicle that was broken. In general all the injured children were fairly large, 7 of them weighing more than 4000 grams, 9 between 3500 and 4000 grams, and only 1 less than 3000 grams. The delivery had occurred spontaneously in 17 cases, and had been terminated with forceps in 5 instances. Since these 5 forceps extractions belonged to a series of 60 forceps cases the incidence of clavicular fractures for this operation may be calculated as slightly over 8 per cent. The records of the spontaneous cases showed that 5 times difficulty was experienced in the delivery of the shoulders, and in 4 instances stated specifically that the shoulders were expelled spontaneously without any particular manipulation.

In 1 case the clavicular fracture was found complicated by a typical brachial palsy.

Muus also saw 2 cases of fracture of the clavicle after version and extraction. In both instances the freeing of the arms had proved difficult. These 2 cases belonged to a series of 30 versions followed by extraction, so that for this procedure a frequency of 6 per cent of clavicular fractures can be figured.

Hauch, after changing the customary technic of the clinic and eliminating for spontaneous vertex deliveries all manipulations except the protection of the perineum, still had 16 fractures in 2531 deliveries. As far as he was able to ascertain the anterior clavicle was injured 13 times, the posterior but twice.

These statistics of Muus and Hauch allow no further doubt that the injury occurs more frequently than now is assumed, and more often, both actually and relatively, in vertex than in breech labors.

**Symptomatology and Diagnosis.**—Conditions, already described in speaking of the pathology, explain the evident fact that, as a rule, the fracture fails to exhibit any noteworthy or even recognizable

clinical symptoms immediately after birth. There is but rarely a distinct dislocation of the bone ends and practically never that lowering and sinking forward of the shoulder so characteristic in the adult. No case so far reported mentions a hematoma in the newborn at the site of the fracture. Usually the infant moves the arm on the affected side as well as on the other, though he possibly will avoid extreme abduction. Even pressure against the clavicle does not necessarily elicit pain. During manipulations in bathing and dressing these infants do not appear markedly noisier than usually. In some instances, however, it has been noticed that the arm hangs limply from the shoulder, most likely when the trauma also has involved the brachial plexus or the shoulder joint, conditions which will be discussed in subsequent chapters.

Only seldom distinct mobility of the bone fragments against each other or actual crepitation can be found. If present, they offer the most reliable symptom for a definite diagnosis. As a matter of fact, in most cases the presence of the fracture is suggested only at the end of the first and the beginning of the second week of life by the formation of a callus, discovered usually by the mother or the nurse, only exceptionally by the attending physician.

Careful examination of every newborn would reveal an unsuspected clavicular fracture in many instances. As emphasized by Rielaender, roentgenography is a most valuable aid in establishing the diagnosis definitely in doubtful cases.

**Prognosis.**—All observers agree that the infraction or actual fracture of the clavicle in birth is void of any direct danger or consequent harm to the infant. The callus is resorbed gradually and the clavicle finally will show a normal configuration. Even in cases of actual displacement of the bone ends, the developmental expansion of the thorax invariably corrects the deformity that might have been noticed during the first few weeks of life (Riether).

A rarer complication of a clavicle fracture may result from the swinging of the asphyxiated child after the method of Schultze. Heydrich felt sure that at least in one case of clavicular fracture, observed by him, the one end of the broken bone during swinging had been driven through the thorax wall and had perforated the lung. However, Schultze himself, and also Beuttner and Keilmann have attempted to prove by means of experiments that also infants with fractured clavicles can be swung without fear of damage to the thorax or lungs if the method is executed exactly in accord with



the rules given by Schultze (quoted by Stumpf). In a subsequent chapter I shall speak of the relation of clavicular fracture to brachial birth palsy.

**Prophylaxis.**—For infants born in head presentation Hauch seemingly has established the fact that the number of fractures can be effectively reduced by avoiding traction on the head or with a finger hooked into the upper axilla during the delivery of the shoulders.

In a normal vertex labor there really does not exist any necessity for hurrying the expulsion of the shoulders. After the head is born, the baby's mouth can be cleansed and by means of a piece of gauze protected against the aspiration of amniotic fluid, mucus or blood when the child begins to breathe. Whenever there is difficulty with the shoulders, one should wait for the support of the next uterine contraction reinforced by a bearing down effort of the mother. It is needless to say that under these conditions a deep twilight sleep offers certain disadvantages.

In view of the acknowledged harmlessness of a clavicular fracture in the overwhelming majority of instances, it does not seem justifiable to advocate an episiotomy for the purpose of eliminating in every instance the necessity of pressure against the posterior shoulder for the protection of the perineum, though this procedure probably proves at times injurious to the anterior clavicle.

In the management of a breech presentation a fracture of the clavicle cannot always be avoided even with extreme care. The injury occurs under these conditions probably less often in the hands of the experienced obstetrician. Great care must be exercised in freeing the arm, especially when found caught in the nape of the neck. However, the obstetrician may well risk a fracture, if in his judgment the condition of the infant demands haste in the delivery. Even a deliberate fracture of the clavicle or humerus in such a situation may be a fully justified procedure.

**Treatment.**—Facts set forth in the preceding pages warrant the advice, generally given, to refrain from any specific orthopedic treatment of the fracture of the clavicle in the newborn. Of the 16 cases of Hauch, 13 showed complete union within 10 days without particular attention. Fixation of the shoulders with adhesive straps or of the upper arms against a T-shaped splint on the baby's back are not only unnecessary, but often most annoying and irritating to the child. If in rarer instances the mobility of the broken ends seems

to persist over the first week, the arm on the affected side may be immobilized.

#### LITERATURE

Hauch. *Zentralbl. f. Gynäk.*, Leipz., 1905, 29:1025.

Muus. *Ibidem*, 1903, 27:689.

Rielaender. *München. med. Wchnschr.*, 1905, 52:1564.

Riether. *Wien. klin. Wchnschr.*, 1902, 15:619.

Stumpf. *Winckel's Handb. d. Geburtsh.*, 3:3, p. 486.

Weltmann. *Inaugural Dissertation*, Breslau, 1917.

## CHAPTER XIII

### BRACHIAL BIRTH PALSY

Historical facts—Pathology—Of actual lesions of the plexus—Of injuries of the shoulder joint—Of the upper end of the humerus—Frequency—Etiology—Compression of the plexus—By forceps—By finger—By the clavicle—Partial or complete tearing of cervical nerves—Symptomatology—Diagnosis—Prognosis—Prophylaxis—Treatment.

The first exact description of a case of brachial birth palsy, according to Stransky, was published by Danyan in 1851.

In 1862 Duchenne (in the second edition of his work on electricity) spoke of a brachial palsy seen in a newborn infant in whom the plexus had been injured by the subacromial luxation of the shoulder in birth. In the next edition of this book (1872) he recognized three types of obstetrical paralysis of the upper extremity: (1) due to pressure of the forceps against the plexus, (2) in which the plexus is injured during certain obstetrical manipulations (lowering of the arm in a breech presentation, hooking of the finger into the upper axilla for traction in vertex presentation, during delivery of the shoulders), and (3) traumatization of the plexus as the result of a subacromial luxation (quoted from Thomas).

Seeligmüller (1874), in a paper dealing mainly with injury of the facial nerve by the forceps, expressed the belief that in the same manner also the brachial plexus may be injured. The more frequent occurrence of a brachial palsy in breech labors he ascribed to excessive stretching of cervical nerves by severe pulling on the shoulders.

In the same year (1874) Erb published his classic monograph on traumatic brachial paralysis as seen in the adult. He discovered as its cause a lesion of the fifth and sixth cervical nerves at a point about two to three centimeters above the clavicle and just behind the posterior edge of the sternomastoid muscle—now well known as Erb's point. Approximately in this region the fifth and sixth cervical nerves emerge between the scaleni muscles and come to lie

closely underneath the skin. Passing with the rest of the plexus fibers behind the clavicle, they run towards the axilla.

On the basis of these anatomic facts, Schultze (1888) developed the theory of a compression of the brachial plexus by the clavicle, when the shoulder is forcibly elevated and the arm thrown upward and backward.

Erb considered the adult type identical with that described by Duchenne as an obstetrical lesion, and thus the condition became known in medical literature as the Duchenne-Erb type of brachial palsy, often simply referred to as Erb's palsy.

Though many other explanations concerning the origin of brachial birth palsy have been offered in the meantime, the Duchenne-Erb theory of a supraclavicular injury of the plexus in general has remained the predominating one in obstetrical literature up to recent years.

Kuestner (1889) could not deny the possibility of a true brachial paralysis being caused by the direct traumatization of the plexus, but felt forced to the deduction that in the larger number of cases the actual cause for the palsy was to be found in a more or less serious bone injury near the shoulder, a fracture or detachment of the upper epiphysis of the humerus. Only in this manner could he explain to his satisfaction the paradox that the facial nerve "injured by hard steel" almost always recuperated, while the brachial plexus traumatized "only by the pressure of the soft finger of the obstetrician" would prove irreparably damaged in so many instances. In all serious cases, he maintained, in which function remains permanently impaired or actually destroyed, the trauma to the plexus was complicated by an unrecognized injury of the humerus within the shoulder joint.

The suggestion made by Burr (in 1892) that the palsy is due to an injury of the cervical cord met with but scant consideration at the time. In view of our newer information concerning birth injuries of the cervical portion of the spine (see Chapter VI) the possibility cannot be denied that in some cases a cervical injury might produce a clinical picture closely resembling an Erb's paralysis.

Arens (1886) explained the palsy as the result of a hemorrhage in torn plexus fibers, and Carter (1892), seemingly as the first, spoke of severe stretching and eventual tearing of some of the cervical nerves at birth (quoted from Wright). This theory met with ready

acceptance when placed on a firmer basis by the experiments of Fieux (1897). The latter entirely rejected the idea that the palsy could be due to strong pressure exerted against Erb's point by the forceps, the clavicle or the finger of the obstetrician. He claimed that the fifth and sixth cervical nerves are partially or completely torn as the result of overstretching if strong traction is made on the fore-coming or after-coming head, not in an axial direction, but with the head bent towards one shoulder (*tractions asymclitiques*). Only in this manner, Fieux argued, could be explained the occasional limitation of the paralysis to the deltoid. Bollenhagen answered him that an isolated paralysis of this sort in an older child or adult patient would prove nothing, because even in a typical Duchenne-Erb palsy, manifest immediately after birth, function may be gradually restored in all muscles except the deltoid.

It seems pertinent to point out in this connection that much confusion and unnecessary controversy in obstetrical literature concerning the etiology of brachial palsy has been caused by indiscriminate quotations from the writings of orthopedic surgeons and neurologists who, as a rule, speak of symptoms and conditions seen in older children and adults. The obstetrician should base his deductions only on phenomena exhibited by the newborn. But exact information concerning these phenomena is deplorably defective on account of the evident lack of interest of the obstetrician in the problem which indifference in turn prevents the orthopedist from seeing these palsies in the very early stage.

Shoemaker (1899) repeating Fieux's experiments was able to confirm the latter's conclusions though he was inclined to believe, in accord with the older conception, that at least in some of the cases the injury consisted in a severe compression of the plexus near Erb's point.

This identical fact was brought out in a paper of Thoyer-Rozat (1904). Experiments had convinced him that oblique traction will cause a dangerous elongation and eventually a rupture of the roots of the plexus. The observation, however, of a typical palsy in a case in which the possibility of an injury by traction had to be positively excluded, forced the admission that in this case the palsy must have been the result of direct pressure against nerve trunks. He finally suggested that tension might render the nerves more susceptible to traumatization by pressure.

Anatomic facts, to be described later, irrefutably prove that partial or complete laceration of cervical nerves actually does occur.

Still another hypothesis concerning the origin of brachial paralysis has been offered by Strausky (1902). In a survey of the entire literature on the subject he became impressed by the striking frequency in which reports stated that these babies were born in a state of asphyxiation. Referring to the widely accepted view that circulatory disturbances together with toxic conditions play an important rôle in the causation of palsies as, e.g., seen in chronic alcoholics or after a general anesthesia, he suggested that possibly the increased vensity and thus abnormal toxicity of the blood of the asphyxiated newborn may stand in an etiologic connection to birth palsies.

Whitman (1905) is generally credited with having cleared up the relation of a posterior subluxation of the humerus to brachial palsy. He differentiated between a primary shoulder dislocation due to a direct trauma sustained during birth and a subluxation developing secondarily to a brachial paralysis.

A great mass of controversial literature has accumulated on this problem of luxation. There exists still a third type of luxation, a rare and truly congenital form, observed by Bramann, Zander, Luft, and others. In a comparison with the very similar congenital hip joint luxation, Kroenlein (as cited by Luft) calculated that there occur probably only 5 congenital shoulder subluxations to every 90 congenital hip dislocations. The X-ray pictures in the congenital type are likely to show defects of the scapula, a rudimentary development of the glenoid cavity, or a characteristic flattening of the head of the humerus.

Kuestner thought that a luxation as the direct result of a birth trauma was impossible, because a force sufficient to dislocate the head of the humerus would be more than sufficient to cause rather a detachment of the epiphysis which accident would prevent a luxation. Most elaborate experiments made on cadavers of newborn infants by Serrés (mentioned by Luft) have uniformly failed to produce a luxation of the humerus.

As a matter of fact, most of recent writers (with the possible exception of Thomas) look upon this subluxation merely as a sequela of a primary paralysis.

Much confusion, also in this question of luxation, however, has

resulted from the unwarranted but common practice of applying the term congenital to all lesions seemingly existing since birth.

In the majority of instances the phenomena of the paralysis disappear; nevertheless orthopedic surgeons and neurologists in adult patients very frequently meet with distortions of an arm seemingly caused by a brachial palsy. This evident discrepancy impressed Lange (1912). He concluded that in most instances the paralysis was only apparent—a pseudoparalysis. No actual lesion of the nerve trunk had occurred in these cases, but an extensive traumatization of the ligaments and of the capsule of the shoulder joint. A subsequent shrinkage of scar tissue has finally produced the condition seen in the adult, an inward rotation and slight abduction of the arm with limitation in outward rotation and elevation.

Lange's theory has met with approval and still is maintained especially by those orthopedic surgeons who endeavor to prove the advantage of surgical procedures, attacking the shoulder joint itself, over operations directed towards restoration of plexus function by the anastomosis of severed nerve trunks (typified in the work of Platt, Sever, Sharpe, Taylor, and others).

Other surgeons combine Kuestner's original conception with the teachings of Lange. They speak of birth injuries of the shoulder joint causing detachment or even fracture of the epiphysis (Peltasohn, Vulpius) or of other bony parts of the joint often associated with lacerations of ligaments or of the joint capsule, immediately causing only a pseudoparalysis. But subsequent embedding of nerves in scar tissue (Gaugele), or a callus from a fracture might in the opinion of others result in a secondary true paralysis of some of the nerve trunks passing through the axilla. In this latter manner the origin of types of brachial palsy can be explained which do not represent the characteristic Erb's palsy.

**Pathology.**—For the moment disregarding doubtful and unsettled points in the etiology, I shall speak from the standpoint of the obstetrician only of certain pathologic-anatomic findings in newborn babies in whom all or some of the muscles of the upper extremity are found in a condition of true or seeming paralysis.

The operative findings of Kennedy, Clark, Taylor and Prout, Fairbank, Platt, and many others, have definitely established the possibility of *actual lesions of the plexus* at or near the junction of the fifth and sixth primary division of the cervical nerves, as a rule, affecting most extensively the fifth.

Clark, Taylor, and Prout divided the neural lesions into immediate and remote. The immediate lesion consists in a tearing of the perineural sheath, surrounding and supporting the nerve trunk, and the incidental rupture of blood vessels belonging to it. There is, furthermore, a severance of some of the nerve strands, more or less complete, depending upon the severity and nature of the injury. The remote result is brought about and its extent determined by (1) the healing of the perineural sheath, (2) the organization of the blood clot, and (3) the ultimate contraction of the cicatrix upon the nerve strands which not only prevents their regeneration, but determines a pressure neuritis in those not severed and on which it may chance to impinge.

According to Fairbank the actual damage varies from a slight tearing of the perineural sheath and hemorrhage into the nerve itself, to complete laceration of the nerve trunk. The milder types are the more common and, therefore, in the majority of cases, complete recovery ensues. The anterior primary division of one or more nerves may be actually pulled out of the spinal cord.

Eversmann in a careful dissection found a tumorlike mass in which the fifth cervical nerve near its junction to the sixth was firmly embedded. The mass proved to be an extensive inflammatory infiltration which had led to the complete destruction of the nerve fibers within the mass.

Anatomic proof for a traumatization of plexus nerves can be assumed for clinical observations like the following: A case of brachial palsy seen by Stahl in which after a forceps extraction a deep scar had formed from a gangrene, running along the edge of the trapezius muscle. Fairbank pointed out that occasionally a tender swelling can be palpated on the neck of the newborn in the region of the plexus. A most remarkable recent observation of Kofferath must be mentioned in this connection: Immediately after an easy low forceps extraction, the infant showed the unmistakable signs of some respiratory embarrassment. On the right side of the neck suggillations could be seen from the pressure of the forceps blade. The next day the child had a typical Erb's palsy of the right arm. Further investigation under x-ray revealed that the right half of the diaphragm was standing higher than the left as the result of a paralysis of the right phrenic nerve. This combination, in the belief of Kofferath, permits no doubt concerning the trau-



matic origin of both paralyses in view of the fact that the phrenic nerve passes very closely to the brachial plexus.

Turning our attention next to the pathology of a primary *injury of the shoulder joint*, we must differentiate between laceration of the joint capsule, a detachment of the upper epiphysis of the humerus and a luxation of its head.

Lange's theory was not based on the actual observation of *capsule lesions* in the newborn. In one adult case operated by him he found the cause of the paralysis to be the embedding of the axillary nerves in dense connective tissue, presumably the result of a tear of the capsule. Identical findings in adults have been noted by Gaugele. Thomas, though arguing in favor of Lange's theory, does not mention the fact that he ever discovered in his operations anatomic evidence of capsule lacerations at birth. Platt makes the specific statement that anatomic confirmation of this injury from actual exploration of the joint is lacking. In his own experience with the open operation, performed for the relief of the secondary subluxation, he had never seen any signs pointing to the occurrence of a previous laceration of the joint capsule.

Anatomic proof of *epiphyseal injury* at birth in the main is based on radiographic findings, though occasionally the detached epiphysis can be palpated.

According to Peltasohn a radiogram may show a change in the direction of the diaphyseal axis at the level of the attachment of the epiphysis, an abnormal gap between diaphysis and the end of the clavicle, or a lateral dislocation of the very small bone nucleus of the epiphysis. As the infant grows older the nucleus does not seem to enlarge normally and later exhibits an irregularity in its contour. Haenisch demonstrated in roentgenograms of five cases that the small ossification center of the epiphysis could be seen lying laterally instead of in the median line above the upper end of the diaphysis. Of these five infants three were cured by operation. Van Neck claimed to have been able to differentiate by means of radiography (in 5 cases) between a mere detachment and a detachment complicated by an actual fracture of the epiphysis. Platt, however, considers the interpretation of such radiographic findings meticulous and unconvincing, and feels compelled to the conclusion that evidence in favor of a pure epiphyseal lesion, presenting the clinical picture of a brachial birth palsy, is wanting.

There cannot be any doubt that a *posterior subluxation of the head of the humerus* is commonly seen in the adult patients in whom the signs of a brachial palsy were manifest at birth.

In the opinion of the many advocates of a mechanical restoration of this dislocation in the younger child, and of its operative correction in the neglected adult case (Bonnaire and Ecalle, Fairbank, Gaugele, Platt, Sever, Thomas, Whitman, etc.) it is this particular feature of correcting the dislocation which explains the more permanent and better functional results of their technic over those obtainable by surgical procedures which solely aim to restore the interrupted nerve connections (Taylor, etc.).

As stated before, rather generally this dislocation to-day is looked upon as a secondary development. The obstetrician will be concerned only with pathologic evidence of a possible luxation of the humerus effected by the trauma of birth.

The posterior subluxation, which as a clinical phenomenon represents an undisputable fact, according to Platt, could be theoretically explained by the laceration of the antero-inferior part of the joint capsule by violent traction and torsion of the arm. This would provide all the factors necessary for the slow forcing back of the head of the humerus by the contracting cicatricial tissue.

Gaugele found in 4 operated cases of subluxation evidences of apparent injuries to the epiphysis and extensive scar formation around the joint. He assumed that the head of the humerus had been pushed from the glenoid cavity by a large hematoma. Realizing that so much blood could not have escaped from the epiphysis, he developed his hypothesis of the injury of the joint capsule which contains larger vessels. There was, however, no anatomic proof that a hematoma had caused the luxation at birth as he assumed.

The occasional palpation of the head of the humerus as a hard protrusion in the axilla of a newborn, exhibiting symptoms of a paralysis, cannot be accepted as proof of a luxation because, as emphasized by Bonnaire and Ecalle, the palpated hard body might be the detached epiphysis.

The presence of a true luxation in the newborn could be established only by the radiographic proof that the ossification nucleus of the upper epiphysis, discovered in an abnormal location, actually lies exactly in the axis of the diaphysis. To prove this it will be necessary to take X-ray pictures from different directions. I am not aware of the fact that such evidence has ever been furnished.

Present information concerning the pathology of brachial palsy can be summarized as follows: conclusive evidence has been furnished that in a large number of the cases the fifth and sixth cer-

vical nerves, usually near or at their junction, have been injured. Radiograms and certain findings during later operations prove that in some instances the upper epiphysis of the humerus is detached or even fractured during birth. In a very limited number of cases operative findings suggest an injury of the joint capsule. There is no anatomic or other proof extant that the posterior subluxation, so frequently seen in adult patients, has actually occurred during labor.

**Frequency.**—Statistics from large maternity services, as quoted in most textbooks, place the frequency of a brachial birth palsy as approximately 1 case from every 2000 deliveries. There cannot be any doubt that this figure falls far below its actual frequency. This figure unavoidably is calculated without the inclusion of the still-born infants, of whom many had been severely injured at birth and presumably would have exhibited a typical palsy, had they not succumbed to other injuries. This figure furthermore applies solely to an obstetric material managed more or less expertly. That inexpert attention during delivery necessarily increases the incidence of this injury has been most convincingly, one may say dramatically, shown by Prouff and Guillemot (*Annales de Gyn. et d'Obst.*, 1897) who reported an "endemic" of 30 cases of brachial palsy in the practice of a single midwife.

There are no reliable statistics available to determine whether this palsy is more common after vertex or breech labors, though the latter seemingly represents the view generally prevailing among obstetricians. Fairbank quotes Tubby and Sherren as respectively claiming to have found both types of labor equally or almost equally represented in the cases seen by them, while he found in his own series of 39 palsies, 32 subsequent to vertex and only 7 to breech presentations. Also Bullard ascertained for 43 cases, that 40 were vertex labors, in 28 instances terminated with forceps. There were but 3 breech cases in his series. Of the vertex labors the obstetric records stated in 18 instances that the shoulders offered difficulty, while in 5 it was expressly noted that the shoulders were not held.

Since these latter statistics have been furnished by orthopedic surgeons, and obstetricians in general continue to adhere to the belief that breech labor offers the better opportunity for an injury of the brachial plexus, the discrepancy would suggest the deduction that the injuries sustained in vertex labors possibly are severer and are more likely to result in a permanent deformity of the affected

arm. A further study of the problem from this angle would seem most desirable.

**Etiology.**—A critical analysis of the many theories concerning the origin of brachial birth palsy, given approximately in their chronological sequence in the introductory paragraphs of this chapter, readily permits their division into two groups, the one assuming a traumatization of cervical nerves by pressure or traction, the other surmising that the primary cause of the actual or seeming paralysis is found in an injury of the shoulder joint, a detachment of the epiphysis, a laceration of the capsule or a luxation of the humerus.

Such a critical analysis of the literature will further show that the real advantage of this grouping of the possible causative factors in the main applies to the problem of appropriate immediate or late efforts to cure the paralysis or functional deficiency of the affected arm. From this point of view these two groups will be considered later in this chapter.

The obstetrician is chiefly interested in the question of the etiology of brachial paralysis only in so far as it is definitely established that during delivery, both in vertex and breech labors, some of the cervical nerve roots are injured with fair frequency, and occasionally also the shoulder joint.

Considering first nerve injuries, which manifestly are the more common, I shall discuss the particular features of common obstetrical conditions and procedures which clinically or experimentally have been demonstrated either to cause severe pressure on nerves, supplying the muscles of the arm, or to lead to their excessive stretching and eventual tearing.

**DIRECT COMPRESSION BY THE END OF A FORCEPS' BLADE.**—This mode of injury, first discussed by Seeligmüller, has been confirmed by numerous clinical observations. Often the traumatization of the region of Erb's point is proved by suggillations or the formation of a palpable infiltration, or a localized gangrene, at times followed by a deep scar (case of Stahl). In most instances of this sort the lesion is slight and the paralytic symptoms disappear quickly. In other cases complete severance of the nerve fibers either as the result of the trauma itself or of their destruction by secondary inflammatory processes, or their continued compression by scar tissue precludes functional recovery. As well emphasized by Stolper, the tip of a forceps' blade is most likely to come to lie near Erb's point when the

forceps are applied as though the head were in a normal occipital presentation while actually it is deflexed.

**PRESSURE EXERTED BY THE FINGER.**—Erb, in his original contribution, mentioned the Mauriceau-Smiellie method of extraction of the aftercoming head as presumably the most common cause of a brachial palsy. This etiological factor is well recognized, and most teachers of obstetrics lay stress on the importance of placing the tips of index and middle fingers, forked above the shoulders, not on the sides of the neck, but on the sternum of the infant. Obstetricians, not enjoying the possession of long fingers, can prevent the end phalanx from lying just above Erb's point and from digging during forcible traction into the infant's neck, if they will deliberately keep the ends of these two fingers in extension and not flexed, as would be their more natural attitude in the attempt to pull on the shoulders.

**COMPRESSION BETWEEN CLAVICLE AND RIBS.**—In support of Erb's theory, Schultze tried to demonstrate that strong elevation of the arm with a simultaneous posterior flexion elevates the shoulder in such a manner that the clavicle is pressed laterally against the neck approximately in the region of Erb's point. A strong curvature of the clavicle and scant development of adipose tissue behind it would favor a harmful compression of the plexus between the clavicle and the underlying ribs. Approximately this situation obtains, if in a breech labor an arm is thrown above the head.

Experimental studies recently published by Weil support strongly this conception of a possible traumatization of the brachial plexus by the clavicle. He found that when in a newborn the shoulder is pressed against the neck, the outer third of the clavicle approaches the transverse processes of the fifth and sixth cervical vertebrae so closely that it actually squeezes the plexus fibers. This pressure is correspondingly increased when a lateral flexion of the head causes a convex curve of the cervical spine directed towards the same side. Thus the narrow space between clavicle and spinal processes is further reduced. In most instances he could notice that under this pressure the movable portion of the plexus would slip forward from underneath the clavicle. The fixed portion of the plexus, however, in this procedure, is severely compressed and may be injured. Excessive wedging of the shoulder against the neck, therefore, will always endanger the brachial plexus. A situation of this kind may develop both in vertex and breech labors. Indeed, this

attitude may have been maintained, for mechanical reasons, by the fetus before labor and then is likely to become exaggerated during labor.

Weil's investigations also furnish support for a hypothesis that in some instances the brachial palsy of the newborn may represent a truly congenital defect of development of the plexus as the result of continued malattitude of the shoulders during intra-uterine life. This possible origin of an Erb's palsy would be analogous to a congenital type of facial palsy and torticollis respectively described in Chapters VIII and XI.

In several instances recorded in literature the plexus was found to have been injured by one end of a broken clavicle (e.g., an observation of DeLee mentioned in his textbook).

**PARTIAL OR COMPLETE TEARING OF CERVICAL NERVES.**—The actual occurrence of such lesions has been irrefutably established by dissections made on newborn infants, and by anatomic findings in numerous operations. In experiments, variously arranged, the attempt has been made to reproduce the mechanical conditions under which, in the course of labor, cervical nerves are assumed to be dangerously stretched and eventually torn.

Clark, Taylor and Prout found that traction on the head, while the shoulders are firmly fixed, causes the nerves of the neck to become taut. On increasing the traction force the uppermost fibers begin to fray and to tear. Further force affects the lower fibers in the same manner. Whenever considerable violence is applied, finally also the deeper cervical fascia is torn.

In a repetition of these experiments by Sever it was shown that considerable force is required to rupture the fifth and cervical nerves. The supraclavicular nerve usually snapped before any other. Sever noted that the lower nerves could be placed in extreme tension only by a simultaneous abduction and elevation of the arm.

According to Weil, traction on an arm stretches the plexus only slightly, more so if the arm is in adduction than in abduction, but never to a degree that could prove dangerous to the integrity of the plexus. The fibers, however, became very tense if the head at the same time was forcibly bent to the other side. Weil thought that these experiments, though failing to result in actual tearing of nerve trunks, still do not exclude the possibility of serious damage to them. A structural lesion might be expected to develop in a nerve in which strong tension is maintained for a long time. Therefore, a brachial

palsy might not only be the result of certain violent manipulations during which cervical nerves had been actually torn, but possibly is also dependent upon the persistence of certain injurious positions of head, shoulder, or arm in the course of labor, in which plexus fibers remain in a state of excessive tension.

Summarizing the question of nerve injuries in the causation of brachial birth palsy, in the light of the foregoing information, we may state: (1) the plexus is occasionally injured by the forceps and (2) possibly more often traumatized by a finger during improperly executed traction in the delivery of the aftercoming head. (3) Cervical nerve fibers under special mechanical conditions are injured by compression between clavicle and underlying bone structures, both in vertex and breech labors, and (4) probably less often are actually lacerated by excessive traction. Cervical nerve roots, by violent manipulations, may be torn out of the spinal cord.

Less space will be required for an adequate consideration of the various shoulder lesions regarded of etiological importance in the causation of brachial palsy by some authors.

Since no evidence has been furnished to prove that during a spontaneous labor or through obstetrical manipulations the capsule of the shoulder joint can be torn or the head of the humerus pushed from the glenoidal fossa, the obstetrician does not need to concern himself with these injuries. Epiphyseal detachment, on the other hand, is widely recognized as a possible accident, especially when unusual difficulty is experienced in bringing down an arm caught in the nape of the neck. In most instances of this injury we are, however, dealing probably only with a pseudoparalysis. On account of the joint trauma the arm is held in a position which, on superficial examination, strongly simulates a typical brachial palsy.

For therapeutic reasons, it is essential to determine in each case, immediately after birth, whether the cause of the evident functional disability of the arm is an injury of the brachial plexus or a trauma of the shoulder joint.

**Symptomatology.**—Speaking solely of the newborn, the predominating feature in the clinical picture of a typical brachial birth palsy is the fact that the affected arm hangs limply from the shoulder in an inward rotation with the fingers usually held clenched. Continued observation will permit one to ascertain that the baby does not raise, abduct, or rotate the arm or bend it at the elbow. Since there is no possibility of determining in the newborn infant, as in the adult, that

this apparent impairment of mobility is due to a paralysis of certain muscles, it can be easily understood why without further careful investigation the incorrect diagnosis of brachial palsy is practically without exceptions made when in fact an injury of the shoulder is responsible for the anomalous attitude of the arm.

In the case of the typical Erb's palsy, the muscles innervated by the fifth and sixth cervical nerves are more or less involved, namely, the deltoid, supra- and infra-spinatus, teres major, biceps and supinator longus (brachioradialis), occasionally also the anterior serratus, coracobrachialis and supinator brevis.

If the traumatism in rarer instances extends to the seventh and eighth cervical and even first thoracic nerves, the muscles of the entire arm become paralyzed. Wrist drop and flaccid fingers are added as symptoms easily recognizable even in the newborn.

There exists still another form of brachial palsy of the newborn, only rarely observed, which is distinctly different from the Duchenne-Erb type. The paralysis is limited to the muscles of the lower arm innervated chiefly by the seventh and eighth cervical nerves (Klumpke's type). In these cases in addition to the paralysis of some or all of the muscles of the forearm, often fibers, as well, going from the lower cervical nerves to the cervical sympathetic may be affected, resulting in a miosis and narrowing of the palpebral fissure with endophthalmus (Sever, Fairbank).

At times only individual nerves of the arm are found to be paralyzed, usually as the result of localized traumatization. This condition will be dealt with in the next chapter.

**Diagnosis.**—Certain facts mentioned in foregoing pages make it obvious that the more or less characteristic attitude of the arm immediately after delivery will permit the definite diagnosis of an injury to the brachial plexus only if radiographic examination excludes a possible lesion of the shoulder joint.

In regard to the study of the shoulder of the newborn in the X-ray picture, it must be admitted that the problem offers many difficulties. Very little definite information is extant concerning the appearance of this joint and of the small ossification centers in the picture under normal conditions. Only roentgenograms taken from various angles can show that the small nucleus of the epiphysis does or does not lie exactly in the axis of the diaphysis, or that there is an abnormally wide gape between the diaphysis and epiphysis or the clavicle.



Nevertheless, I am convinced that careful radiographic study must be a part of the routine examination of every evident or suspected case of brachial birth palsy. In view of information presented in Chapter XII it can be reasonably expected that such a routine would bring to light in many of these cases an entirely unsuspected fracture of the clavicle. I have referred to the fact that in a few well-authenticated observations the end of a fractured clavicle had traumatized the plexus. A comparison of some of the mechanical factors in obstetrical manipulations, held responsible for the causation of clavicular fractures, with those supposed to account for plexus injuries, as, e.g., the asynclitic tractions, shows that they are practically identical. If fracture of the clavicle actually represents a common, though usually overlooked, birth injury, it would seem possible that it plays an important and still unrecognized rôle in the etiology of brachial palsy.

Only a radiographic examination will establish the correct diagnosis in a case of congenital deformity of the shoulder joint or allow the differentiation between a true palsy and a pseudoparalysis.

A fracture in the cervical portion of the spine (Burr) or an osteochondritis of congenital syphilitic origin (several cases mentioned by Backhaus) may present a clinical picture so closely resembling a brachial palsy that only the X-ray examination will furnish a clue for the correct diagnosis.

Pain is a symptom of very limited diagnostic value. Tenderness to pressure over Erb's point is occasionally mentioned as a sign of traumatization of the plexus. Subjective pain, indicated by fretfulness and irritability of the newborn, in the opinion of Taylor, proves the actual tearing of nerve trunks. From the viewpoint of prognosis such peevishness of the child then would suggest that in this case spontaneous restoration of function cannot be expected.

A joint injury would necessarily cause discomfort, and it seems possible that in such a case the child would hold the arm of the affected side more or less rigidly against the side of the thorax as an automatic protection against painful movements of the shoulder joint.

Symptoms of intracranial hypertension must suggest the possible central origin of a paralysis as the result of an intracephalic birth trauma.

Since authentic cases of infantile paralysis in newborn infants are

known in literature, this condition will also have to be considered in the differential diagnosis of a birth palsy.

**Prognosis.**—In by far the larger number of cases the symptoms of a brachial paralysis begin to improve promptly and disappear within a comparatively short time, exactly as in a facial palsy. However, much more frequently than in a facial palsy, the functional impairment becomes permanent. There are various reasons for this fact. In a few instances some of the nerve trunks have been completely crushed or actually torn apart. In others, as assumed by most writers, the paralytic condition of certain muscles, especially of the biceps, together with traction exerted by the weight of the arm, secondarily leads to a subluxation of the head. Cases seen soon after birth with undoubted palsy and no joint injury after some months are found with the palsy recovered but the joint subluxated (Fairbank). It also is possible that a primary pseudoparalysis develops into a true paralysis when some of the axillary nerves become embedded into a callus or into shrinking scar tissue developing in the immediate surroundings of an injured joint. Therefore, the obstetrician is not justified in rendering an absolutely favorable prognosis in any case.

The final outcome of the Klumpke type of forearm paralysis is notoriously bad. It is claimed that a spontaneous recovery of function has never been observed.

**Prophylaxis.**—In speaking of the etiology of brachial birth palsy I had occasion to emphasize certain details in the application of the forceps, in the execution of the Mauriceau-Smellie maneuver and in freeing an arm caught above the aftercoming head, which tend to minimize the danger to the cervical nerves.

As far as the injury of the plexus or of the shoulder by severe traction is concerned, only the general principle can be pointed out that wherever there is difficulty with the shoulders, either in vertex or breech presentations, forcible traction should be made, as far as such is possible, only along the long axis of the child and never against or on a head in lateral flexion.

**Treatment.**—Within the scope of this volume solely the immediate treatment can be considered. Sever, Platt, Fairbank, Boorstein and many other recent contributors have convincingly demonstrated the dangers and regrettable consequences of the still customary indifference of the obstetrician towards the anomaly, presumably due to his expectation of a spontaneous functional restora-

tion. All these writers insist upon prompt therapeutic attention in every case.

Since undeniably spontaneous recovery occurs in by far the larger number of cases, the early treatment necessarily must be conservative. The arm is placed in the position of physiologic rest (Sever), that is, held by means of an appropriate splint in abduction, elevation and outward rotation. Twice daily the arm is taken down for massage and the following manipulations: external rotation with or without abduction of the shoulder; full flexion and extension of the elbow; full supination of the forearm; dorsiflexion of wrist and fingers, and adduction of the externally rotated arm while the scapula is depressed by a hand on the shoulder (Fairbank).

In the belief of Sever a treatment of this sort, instituted without delay in every case, would leave but few instances in which the paralytic symptoms would not disappear, and would permit no infant to develop a secondary internal rotation contracture and a posterior subluxation of the shoulder joint.

Orthopedists differ as to the time during which such conservative efforts can be safely continued. Fairbank thinks that three months probably represent the limit, while Sever is willing to extend the trial to one year.

The still customary fixation of the affected arm along the thorax is rather generally deprecated by modern orthopedists. It has its very limited value only in certain types of shoulder injury.

## LITERATURE

Ashhurst. *Ann. Surg., Phila.*, 1918, 67:25.

Backhaus. *Zentralbl. f. Gynäk., Leipz.*, 1914, 38:1190.

Bonnaire and Ecalle. *Arch. mens. d'obst. et de gynéc., Par.*, 1913, 2:49.

Boorstein. *Med. Rec., N. Y.*, 1919, 96:790.

Bullard. *Am. J. M. Sc., Phila.*, 1907, 134:93.

Clark, Taylor, and Prout. *Ibidem*, 1905, 130:671.

Ehrenfest. *Interstate M. J., St. Louis*, 1908, 15:747.

Eversman. *Arch. f. Gynäk., Berl.*, 1903, 68:143.

Fairbank. *Lancet, Lond.*, 1913, 1:1217.

Idem. *J. Orthop. Surg.*, 1920, 2:284.

Fieux. *Ann. de gynéc. et d'obst., Par.*, 1897, 47:52.

- Friedman. Boston M. & S. J., 1921, 184:482.
- Gaugele. Ztschr. f. orthop. Chir., Stuttg., 1914, 34:511.
- Haenisch. Verhandlungen der deutschen Roentgenesellschaft, 1913, 9:86.
- Kofferath. Monatschr. f. Geburtsh. u. Gynäk., Berl., 1921, 55:33.
- Kuestner. Müller's Handb. d. Geburtsh., 1889, 3:301.
- Lange. München. med. Wchnschr., 1912, 59:1257.
- Luft. Monatschr. f. Geburtsh. u. Gynäk., Berl., 1909, 30:307.
- Peltesohn. Berl. klin. Wchnschr., 1914, 51:1162.
- Platt. J. Orthop. Surg., 1920, 2:272.
- Schultze. Arch. f. Gynäk., Berl., 1888, 32:410.
- Sever. Am. J. Dis. Child., Chicago, 1916, 12:541.
- Idem. Am. J. Orthop. Surg., 1918, 16:248.
- Sharpe. Meeting of Clinical Society of the New York Polyclinic Hospital, October 1, 1917. Reprint.
- Stahl. Winckel's Handb. d. Geburtsh., 3:1, p. 568.
- Stolper. Monatschr. f. Geburtsh. u. Gynäk., Berl., 1901, 14:49.
- Stransky. Zentralbl. f. d. Grenzgeb. d. Med. u. Chir., 1902, 5:497.
- Taylor. Surg., Gynec. & Obst., Chicago, 1920, 30:494.
- Thomas. Am. J. Obst., N. Y., 1916, 73:577.
- Thoyer-Rozat. L'Obstétrique, 1904, 9:413.
- Van Neck. J. méd. de Brux., 1914, 19:133.
- Vulpius. Berl. klin. Wchnschr., 1914, 51:1162.
- Weil. Zentralbl. f. Chir., Leipz., 1921, 48:1312.
- Whitman. Ann. Surg., Phila., 1905, 42:110.
- Wright. New York M. J., 1917, 105:788.

## CHAPTER XIV

### INJURIES OF THE UPPER EXTREMITIES

Brachiotomy—Congenital anomalies of the bones—Intra-uterine fractures—Fractures of humerus in birth—Resulting palsy—Injuries of elbow—Fractures of forearm—Treatment.

A consideration of birth injuries of the arm is appropriately started with historical references to the operation of brachiotomy—the amputation of a prolapsed arm to permit the extraction of a living child. This procedure is unknown in modern obstetrics. Baudeloque, as one of the first, severely condemned this unnecessary mutilation of the fetus, and most of subsequent writers concurred in his opinion. The question was widely discussed at the occasion of legal action against a physician who in 1825 had amputated both arms of a living child. While Leroux and Dupuytren maintained that under certain conditions this procedure was justified and necessary in the attempt to save the infant's life, Capuron (1828) announced the principle that an already born part can never be regarded a mechanical obstacle to the delivery of that part of the fetus still within the uterus. As late as 1836 Oehler, a German obstetrician, defended seriously brachiotomy as a legitimate operation, and in 1881 Vibert reflected on the legal and medical aspects of this operation in connection with a case in which a physician had amputated the arm of a living child and by the court had been found guilty of malpractice.

There are certain instances known in forensic literature in which arms were torn off or cut off by mothers endeavoring to deliver themselves without the aid of a physician (e.g., a case of Barbour mentioned by Stumpf).

Kratter studied experimentally, and admitted, the possibility of the complete avulsion of an arm by violent traction, when called upon to give expert opinion in legal proceedings against a physician, who, in the course of a breech labor, had torn out one arm of a living child. Blaettner, cited by Stumpf, saw a child in whom a

physician, after having unintentionally torn out the one arm, had cut off the other to extract the aftercoming head. Arnstein, twelve hours after delivery, found in the uterus of a moribund woman, handled by a midwife, an arm and a piece of clavicle. Doktor referred to a case in which an arm had been torn out during a version, presumably because the arm had been mistaken for a leg.

In the causation of such serious mutilating injuries, both lack of skill on the part of the attending physician or midwife, and their legal responsibility would seem self-evident.

Less certain is the responsibility of the attendant for the comparatively minor injuries of the upper extremity, like the possible luxation, fracture or detachment of the upper epiphysis, amply discussed in the preceding chapter, fractures of the diaphysis, fractures and dislocations in the region of the elbow, and the rarer instances of traumatization of the forearm.

In a limited number of these cases multiple fractures, occurring without any noteworthy external trauma, must be attributed to pathological conditions of the affected bones.

Chaussier (quoted by Ballantyne) in 1812 recorded the case of a spontaneously born child in whom between 50 and 100 fractures were counted in various bones of the skeleton, apparently due to an exceptional brittleness of the bones. Griffith described a typical example of *osteogenesis imperfecta* in which, without evident or sufficient cause, fractures of long bones began to occur within a few hours after birth, 17 of them developing within the first two years of life of this child. This tendency to be affected by numerous fractures (*osteopsathyrosis*) has been known to be transmitted by heredity. A practically identical observation more recently (1919) has been reported by Satanowsky.

Such an abnormal fragility of the bones accounted for the more than 30 fractures discovered by Linck in an infant born after but one strong uterine contraction. Further investigations in this instance showed that some of these fractures seemingly were fresh while others unquestionably were older and must have occurred during intra-uterine life. Burmeister, in a paper on "so-called" intra-uterine fractures, stressed the point that it is incorrect to speak in these cases of fractures. Ossification is not advanced far enough to permit the actual breaking of a bone. Usually there is no definite interruption in the continuity of a long bone. They are in most instances only bent and twisted by external pressure exerted by amniotic bands, a fibroid, or the uterine wall itself if amniotic fluid is scant.

The entire problem of intra-uterine fractures is covered in a most comprehensive paper of Richard R. Smith. The intra-uterine origin of the deformity of an extremity, evident at birth, is strongly suggested whenever it is found due, not to the dislocation of freely movable bone fragments, but to a firm angulation of a long bone. Its true character is definitely established when a radiogram reveals a callus or signs of retarded development of the extremity below the level of the assumed fracture. I shall not enter into a detailed discussion of the disputed etiology of such congenital deformities, whether actually the result of fractures or only resembling them. All extant information can be found in the paper of Smith. It is interesting to the obstetrician that in their origin amniotic bands probably play a definite rôle, and that in some instances intra-uterine fractures likely have been caused by severe traumatization of the pregnant uterus from without. From this latter viewpoint Smith analyzed 43 cases reported in literature as actual intra-uterine fractures of various bones of the skeleton including the skull. In 3 cases the fetus was directly injured by penetrating agents (gunshot, sickle, pitchfork). In 22 cases the fetal injury was supposed to have been due to a heavy fall, in 10 to blows, in 1 to abnormal pressure from the uterine wall. Fractures of this kind, as might be expected, are more likely to occur in the later months of pregnancy. Of these fractures, 32 affected only one bone (including 11 fractures of the skull) and 5 were multiple. In 4 of the cases the humerus, in 4 others the bones of the forearm had been broken.

In regard to the diagnosis of fractures actually sustained during birth, with which we are chiefly concerned, it is well to remember that (1) true intra-uterine fractures may occur, though rarely, (2) they can be often distinguished from parturitional injuries by definite X-ray findings and (3) an imperfect development of bones or their pathological fragility, favoring intra-uterine fractures, necessarily exposes the infant to an abnormal liability of sustaining fractures during labor.

**Fractures of the Humerus.**—Most often they are located in the diaphysis. At times they represent only partial infractions; in the larger number of cases, however, true transverse or oblique breaks. As a rule, injury of the periost allows distinct dislocation of the fragments. In most cases these fractures are the result of difficulties experienced with an arm in the management of a breech labor. With

greatest frequency they are observed after version followed by immediate extraction in cases of placenta previa or eclampsia.

While traction on the arm with simultaneous rotation (e.g., in turning the delivered posterior shoulder forward under the pubis to free the other arm in a breech presentation) is a mechanical factor of recognized etiologic significance, direct pressure against the humerus undoubtedly is the more common cause of its fracture. This fact plausibly explains the occurrence of fractures of the arm also in spontaneous vertex labors.

The humerus is liable to be broken if in a breech case an arm above the head is forcibly brought down by pressure against the upper arm instead of resorting to the proper procedure: pushing the body of the infant a little way back and then carefully wiping the arm over the child's face by passing the introduced hand gradually from the shoulder down over the elbow to the wrist. It does not need to be emphasized that the obstetrician will be justified in deliberately breaking the humerus if in his best judgment a quick delivery is required in the interest of the child. But, as stated in the preceding chapter, undue haste of the less-experienced operator in the management of the aftercoming head is more likely to cause the serious and even fatal traumatization of the infant than to result in the saving of an endangered fetal life.

More often than generally appreciated the humerus is broken both in vertex and breech labors by being firmly pressed against the symphysis, which pressure is exaggerated by traction on the fetus. In a case of Kogvaldson (quoted by Jaeger) the arm was fractured by compression between symphysis and aftercoming head. There could be no question in this particular case because on the side of the face a distinct pressure mark could be seen produced by the elevated arm.

With the anterior shoulder under the subpubic arch in a vertex presentation the humerus may be too forcibly pushed against the symphysis in the attempt to protect the perineum (1) by pushing the posterior shoulder through the distended perineum upward, (2) by raising the anterior shoulder with a finger hooked into the anterior axilla, and (3) by elevating the born head. Good illustrations of fractures resulting in this manner can be found, e.g., in Jaeger's paper. He also quoted an observation of Engel in which the injury to the humerus apparently was caused by traction from the axilla in a downward direction in the endeavor to release the upper shoulder caught behind the symphysis. It seems doubtful whether Engel was



justified in ascribing, in still another instance, a fracture of the humerus to an expression after Kristeller's method, in which, if correctly performed, the downward pressure during a uterine contraction by the two spread palms is exerted only over the fundus portion of the uterus.

Mere infractions of the humerus, as a rule, produce no symptoms immediately noticeable. Presumably infractions are overlooked in most instances, if not later a callus formation suggests their presence. In true fractures a marked dislocation of the fragments will unmistakably reveal the injury. But it seems that often the attendant's attention is called to the injury only by the fact that the child does not move the arm. Palpation of the bone ends, crepitation and X-ray findings establish the definite diagnosis. In many of the recorded cases the fact is mentioned that the physician distinctly felt the snapping of the bone. However, even if this snap or crack is noticed, it is at times difficult to locate the point of fracture. This is well illustrated in one of the observations of Pforte. Most careful palpation failed to reveal the fracture. It could not be seen in roentgenograms. Only three weeks later another picture showed the callus, and an autopsy, made at a later date, left no doubt that the epiphysis had been torn off.

Of great practical importance are the possible consequences of such a parturitional injury of the arm. In the largest number of cases the fracture, under proper care, results in a perfect consolidation of the fragments in from two to three weeks without any further disadvantage to the infant. A pseudo-arthritis develops only rarely. Occasionally one of the nerves of the arm becomes temporarily or permanently compressed by the callus or actually embedded into it. It may be mentioned in this connection that Budin, as quoted by Fueth, thought that in some instances the subsequent paralysis really is caused by an improperly applied plaster cast.

As explained in the preceding chapter, as sequelae of fractures and other localized traumas, in this manner, the rarer types of isolated paralysis of one nerve trunk of the arm may develop. Loviot (cited by Fueth) had observed a palsy strictly limited to the musculocutaneous nerve subsequently to a fracture of the humerus. Fueth himself an isolated palsy of the radial nerve which disappeared promptly after the operative release of the affected nerve at the site of injury. Kehrer mentioned 4 cases of unilateral and 1 of bilateral radial palsy in newborn infants.

Kehrer thought that strong circumscribed pressure from a symphysis, promontory or exostosis might injure individual nerve trunks. In some cases known in literature deep scars or grooves strongly suggested pressure from amniotic bands. Thus it seems probable that the latter case was responsible for the gangrene of the forearm seen in a newborn child by Winslow, necessitating amputation at the elbow.

A bilateral affection in some cases offers great difficulty for satisfactory explanation. No suggestion concerning its etiology is advanced by Gordon for his unique observation of a symmetrical palsy at birth limited to the flexors carpi ulnaris and carpi radialis of both arms. In a case of Merkel the paralysis of but one flexor carpi radialis was readily explained by a traumatic detachment of the one end of the muscle from its bone attachment (after a very difficult version).

**Injuries of Elbow.**—Few records can be found of serious traumatization of the elbow in birth. The question has been studied experimentally by Truesdell. He ascertained that such forcible manipulations of the arm, as might be required in the management of a breech presentation with an arm caught in the nape of the neck, can result either in fracture of the diaphysis of the humerus or an avulsion and backward dislocation of its lower epiphysis. Bonnaire and Metzger reported a case of actual luxation of the elbow and emphasized the value of the X-rays in the differentiation of this injury from a detachment of the lower epiphysis. An anterior luxation of both radii, possibly of intra-uterine origin, has been seen by Bar and Cautru (mentioned by Stumpf).

**Fractures of Forearm.**—They seem to be extremely rare. Pforte, describing in detail 2 cases of this sort, emphasized that his opportunity of observing 2 cases within a short time is merely a coincidence. In both instances after version and extraction ulna and radius were found fractured, though no difficulty whatever had been experienced with the arms. The fractures, in his belief, must have been due to severe pressure against the forearm directly at the site of the injury.

There seems to be but 1 case of parturitional injury of the wrist on record, a luxation seen by Winckel (Stumpf).

But little need be said concerning the therapy of birth injuries of the upper extremity, which, in my opinion, is better left to the experienced orthopedist. Various methods of treatment that have

proved particularly useful to individual surgeons are described in the papers of Dollinger, Oeri, Stuhl, Truesdell, Weltmann, etc., and are comprehensively discussed in a recent paper of Boorstein (1920).

Active treatment by means of splints or plaster casts with and without added extension is strictly required because untreated cases often result in a permanent deformity of the arm caused by angular healing, by a palsy, or sometimes by a pseudo-arthritis. In regard to the problem of the palsy, recent literature evidences a growing tendency in favor of operative restoration of the interrupted nerve function before muscle atrophy has advanced too far.

### LITERATURE

- Arnstein. Original in Polish, abstr. in *Jahresb. ü. d. Fortschr. d. Geburtsh. u. Gynäk.*, 1903, 16:1142.
- Bonnaire and Metzger. *Ann. de gynéc. et d'obst.*, Par., 1910, 7:236.
- Boorstein. *Am. J. Dis. Child.*, Chicago, 1920, 19:375.
- Burmeister. "So-called" Intra-uterine Fractures, Ruge's *Festschrift*, Karger, Berl., 1896, p. 215.
- Doktor. *Zentralbl. f. Gynäk.*, Leipz., 1902, 25:831.
- Dollinger. *Deutsche Ztschr. f. Chir.*, Leipz., 1902, 65:570.
- Fueth. *Zentralbl. f. Gynäk.*, Leipz., 1909, 33:1201.
- Gordon. *J. Am. M. Ass.*, Chicago, 1914, 63:2282.
- Griffith. *Am. J. M. Sc.*, Phila., 1897, 113:426.
- Jaeger. *Gynäk. Rundschau*, Vienna, 1912, 6:511.
- Kehrer. *Zentralbl. f. Gynäk.*, Leipz., 1919, 43:338.
- Kuestner. *Monatschr. f. Geburtsh. u. Gynäk.*, Berl., 1910, 32:90.
- Linck. *Arch. f. Gynäk.*, Berl., 1887, 30:264.
- Merkel. *München. med. Wchnschr.*, 1903, 50:1012.
- Oeri. *Beitr. z. Geburtsh. u. Gynäk.*, Leipz., 1909, 13:309.
- Pforte. *Gynäk. Rundschau*, Vienna, 1908, 2:285.
- Satanowsky. Original in Spanish, abstr. in *J. Am. M. Ass.*, Chicago, 1920, 74:212.
- Smith. *Surg., Gynec. & Obst.*, Chicago, 1913, 17:346.
- Stuhl. *Deutsche med. Wchnschr.*, Berl., 1907, 33:103.
- Stumpf. *Winckel's Handb. d. Geburtsh.*, 3:3, pp. 507, 629.
- Truesdell. *Am. J. Obst.*, N. Y., 1916, 73:1065.
- Weltmann. *Inaugural Dissertation*, Breslau, 1917.
- Winslow. *South. M. J.*, Nash., 1912, 5:64.

## CHAPTER XV

### INJURIES OF THE THORAX

Etiology—Danger from a clavicular fracture and from the various methods of resuscitation.

Berkeley and Bonney, without mentioning actual observations or quoting a reference, state that a hydrothorax of the fetus may prevent the shoulders from entering the pelvis, and that under these circumstances the perforation of the thorax is required to permit delivery. As a matter of fact, the thorax hardly ever gives rise to mechanical difficulties during labor and for this reason, in striking contrast to all the conditions discussed in the preceding chapters, parturitional injuries of the thorax do not occur in delivery but, with the fewest exceptions, are due solely to manipulations of the born child. Among them, efforts in the resuscitation of the asphyxiated newborn represent the most common cause for traumatization of the thoracic wall and the lungs.

I spoke in a previous chapter of the possible serious injury caused by the end of a broken clavicle being driven into the chest wall during Schultze's swingings. Stumpf mentioned a case of Heydrich in which the fragment of a clavicle during swinging had been forced into the lungs. In spite of the assertion of Schultze himself and also of Keilmann and Beuttner to the contrary, swinging of the asphyxiated child must always be regarded a risky method of resuscitation in the presence of a fractured clavicle. In this connection the undisputed fact must be reiterated that in the majority of instances clavicular fractures are overlooked in the newborn.

During swinging—to be sure, only if improperly done by the inexperienced—the larger pectoral muscle and the anterior wall of the chest may be injured (Knapp and Dittrich). Fractures of ribs produced in this manner have been repeatedly recorded in literature.

Another fairly common source of injury is the insufflation of air into the collapsed lungs. Stumpf, in a thorough discussion of this problem, gives a long list of observers who have seen many instances of traumatic emphysema, of rupture of alveoli, a pneumopericardium (Fritsch), a pneumothorax (Runge), and even a fatal air embolism

(Marchand). DeLee mentions in his textbook that Bichat has proved that it is possible by means of tracheal insufflation to blow air into the vessels of the heart and into the general circulation if too much force is used.

Occasionally air is unintentionally blown also into the stomach, which accident, however, according to Kupffer, never interferes seriously with the respiratory movements of the diaphragm.

Incidentally it may be mentioned in this connection that direct mouth to mouth inflation in some cases seemingly has resulted in the infection of the newborn with tuberculosis. Reich, in a most interesting contribution, furnished convincing evidence for his assumption that a tuberculous midwife, customarily resorting to this method, within thirteen months had become responsible for the death of 10 infants from tuberculous meningitis.

Fraught with danger to the integrity of the lungs is the forced inflation and deflation by means of variously constructed pumps. Theoretically they demand the amount of the injected air or oxygen to correspond exactly to the lung capacity of the infant to be resuscitated. Since the latter varies within considerable limits, these motor devices in practice as a whole have been found ineffective or actually harmful, and for this reason have lost much of their transient popularity of a few years ago.

The firm grip on the shoulders required for swinging has been held responsible for injuries of various muscles of the shoulder girdle. It cannot be denied that this is possible; however, of late such injuries have been satisfactorily explained in another manner. Investigations and experiments of Sellheim have shown that these muscles often are traumatized as the result of the pronounced mechanical modification of the shoulder girdle in the course of labor. Under the influence of circular compression by the birth channel the scapulae are pushed towards the head, and the clavicles turned steeply. In this way the lower portions of the trapezius muscle may be overstretched and eventually also torn. The consequence will be a permanent elevation of the shoulder blade, still customarily described as a congenital anomaly. Veit, in discussing Sellheim's paper, emphasized that confirmatory evidence for this theory is found in the occasional observation that several children of the same mother exhibit the identical deformity of the shoulder, strongly suggesting an abnormal pelvis as its original source.

Extremely rare are thorax injuries caused by the forceps.

Audebert saw a case in which the tip of improperly applied forceps had perforated the thoracic wall.

Worthy of serious consideration is an observation only recently reported by Kofferath (1921). A newborn infant immediately after birth showed a peculiar respiratory embarrassment. Respiratory movements seemed regular and deep, but there was a distinct stridor and marked quivering of the nasal alae, suggesting an obstacle rather in the upper air passages. All examinations proved negative. On the next day the child had a typical brachial palsy on the right side. A roentgenogram showed that the right half of the diaphragm was standing high. Both sides of the diaphragm moved with inspiration and expiration, but the right half in a distinctly abnormal manner, rising with inspiration and moving down in expiration. The diagnosis of an Erb's palsy on the right side with a simultaneous paralysis of the right phrenic nerve could be made. Within four days the noticeable symptoms of both palsies had practically disappeared. An X-ray picture made four months later showed the right half of the diaphragm still to be slightly higher than the left. Suggillations on the right side of the neck near Erb's point left no doubt that in this case both nerves, at that point lying in close proximity, had been traumatized by the tip of the one forceps' blade.

Kofferath refers to the fact that a phrenic paralysis has been often described in literature. Weigert (*Bruns' Beiträge zur klinischen Chirurgie*, 1920, 119:100) has collected the records of 30 cases. Only in Kofferath's case the existence of this palsy has been definitely demonstrated in the X-ray picture. Further investigations of such cases of temporary or permanent unilateral relaxation of the diaphragm, usually termed congenital, might reveal that at least at times the condition actually is the result of a birth injury of the phrenic nerve.

#### LITERATURE

- Audebert. Quoted by Wyder in Winckel's Handb. d. Geburtsh., 3:1, p. 568.
- Berkeley and Bonney. Difficulties and Emergencies of Obstetric Practice, Second Edition, Blakiston, 1915, p. 419.
- Kofferath. Monatschr. f. Geburtsh. u. Gynäk., Berl., 1921, 55:33.
- Reich. Berl. klin. Wchnschr., 1878, 15:551.
- Sellheim. Deutsche Ztschr. f. Chir., Leipz., 1909, 102:271.
- Stumpf. Winckel's Handb. d. Geburtsh., 3:3, p. 486.

## CHAPTER XVI

### INJURIES OF THE ABDOMEN

Medicolegal facts—Traumatization of abdominal viscera during resuscitation—General causes—Injuries of liver—Of suprarenal glands—Of spleen—Of pancreas—Of kidneys—Of intestines—Of bladder—Of uterus—Of testicles and scrotum—Of female external genitalia.

The most thorough investigations concerning visceral birth injuries recorded in medical literature have been made by Hedrén in a series of over 1000 autopsies of newborn infants. In two papers, embodying the results of his exhaustive studies, he laid particular stress on the forensic significance of these lesions.

In Chapter IV, I mentioned the fact that cranial injuries, inflicted in the criminal attempt to kill the newborn, often are found complicated with visceral traumatisms. The infant, as a rule, seems to be firmly grasped around the waist for the purpose of hitting his head against a firm object. An example of this fairly typical combination of fatal skull fractures with rupture of the liver in instances of infanticide was described, e.g., in a paper of Lindner.

Rarer, but well known in forensic literature, are serious intra-abdominal lesions with fatal hemorrhages resulting from blows and kicks against the abdomen of the infant. The origin of such injuries usually is clearly revealed by the external evidences of severe traumatization.

In a differentiation of criminal visceral injuries from those sustained in delivery the fact must be remembered that particularly liver and spleen of a dead infant are very easily traumatized. All investigators (Spencer, Hedrén, Geill, etc.) emphasize that certain intra-abdominal lesions discovered at post-mortem examinations unquestionably are solely due to rough handling of the corpse.

For the consideration of the pathology and etiology of abdominal injuries developing incident to birth it would be essential to distinguish the strictly natal ones, i.e., those sustained during labor and delivery, from neonatal lesions, inflicted chiefly during resuscitation of the asphyxiated newborn. In practice such clear differentiation is impossible

There are but few of the many types of birth injuries discussed in the preceding chapters which have not been attributed by some writers to Schultze's swinging. However, among the injuries blamed on this procedure, peritoneal hemorrhages originating from lacerations of the liver, spleen, adrenals and pancreas always held a prominent place in the controversy. Thus Ahlfeld very justly objected to the all too common practice of swinging newborn infants simply because they do not seem to breathe properly. Schultze himself, at various occasions, most emphatically had insisted that his method should be used only in the cases of deep pallid asphyxiation. This really means, only infants seemingly lifeless, limp and pale, and for this reason not differentiable from seriously injured infants with cerebral or abdominal hemorrhages, should be swung. Under such conditions it necessarily becomes impossible to determine whether serious intra-abdominal lesions, discovered at autopsy, were primarily responsible for the symptoms erroneously interpreted as deep asphyxiation, or are the result of the swinging. Ahlfeld drew the logical conclusion: although swinging, even when done exactly in accord with the precepts of Schultze, can never be regarded as free of danger, it is unjust to blame a visceral injury on the man who employed the method. Nacke, joining in the discussion, felt most certain that swinging might cause great harm. He had lost a child from a rupture of the liver, which could not have been caused by birth, because the infant had been born by a cesarean section quickly done on a dying woman not in labor. It was the contention of Schwab that merely theoretical considerations could leave no doubt that the forced flexion and extension during swinging unavoidably would imply a definite trauma to the abdominal viscera.

It seems likely, as Schwab assumed, that a systematic search for lesions of this sort in all necropsies of newborn infants would reveal their presence with unsuspected frequency. This has actually been found true in the investigations of Spencer in 130 and of Hedrén in 1000 autopsies. They and others discovered many instances of subcapsular hemorrhages and actual ruptures of the liver, ruptures of the spleen, hemorrhages in pancreas, kidneys, adrenals, uterus and testicles, ruptured bowels and tears of mesenteric vessels.

An analysis of these observations reveals certain general principles on which the etiology of such visceral lesions can be acceptably explained.

As the presenting part enters the pelvic canal and gradually



emerges from the tight vulvar opening, some of the fetal blood is backed up into the still unborn part of the child. The resulting passive congestion in vertex labors will be more marked in the organs of the lower abdomen (uterus, testicles), in breech labors, on the other hand, in the viscera of the upper abdomen—liver, spleen, pancreas, adrenals and kidneys. It is this fact which, at least in part, accounts for the recognized importance of breech presentation in the causation of visceral injuries.

This mechanical hyperemia, especially in association with abnormal fragility of vessels [prematurity (Lesser), syphilis (Spencer, Gaifami), etc.], or if added to an already existing congestion from intra-uterine asphyxiation, will lead to hemorrhages within the affected organs (subcapsular hematomas of liver, parenchymatous hemorrhages of pancreas, adrenals and kidneys).

Intraperitoneal hemorrhages develop only if an organ is actually ruptured. This in general seems impossible without an additional definite trauma. There are some cases on record in which this essential trauma presumably was supplied by a severe blow against the pregnant woman's abdomen. More often the trauma consists in undue pressure against the infant's abdomen during manipulations in artificial delivery or during resuscitation. Also this fact explains the evident preponderance of babies born in breech presentation among those in whom abdominal organs are found injured.

A congested, or diseased and abnormally large organ (spleen) of necessity will prove particularly vulnerable. The responsible trauma then might have been unimportant or inflicted unknowingly. This fact, in Hedrén's belief, accounts for the continued controversy concerning a question of great forensic importance, viz., whether serious visceral injuries can occur in spontaneous labors. Beyond question they do, if the term "spontaneous" includes all the customary and seemingly harmless manipulations of the child in an otherwise normal and spontaneous labor, such as external pressure during uterine contractions (Kristeller's maneuver), extraction of the rest of the body after the head is born, efforts, outside of actual swinging, to incite suspended respiration, etc. Severe visceral injuries, with the exception of one single observation of hepatic rupture recorded by him, according to Hedrén have never been seen in a baby born spontaneously in the strictest sense of the term, i.e., without any aid of any sort. Medicolegally this is a most important fact.

In a case of suspected infanticide the medical expert will have to

take into consideration that it has been irrefutably established by various authentic observations, that a woman trying to deliver herself, especially in a pelvic end presentation, may rupture the liver by too firm a grasp around the infant's waist.

In regard to the rupture of viscera in the lower abdomen, especially of the colon and the urinary bladder, the opinion prevails that the accident occurs only if these organs are abnormally distended and in a pathological condition. This holds true particularly for the bladder.

## LIVER

By far the most common among intra-abdominal birth traumas are the injuries of the liver. Arranged according to their respective frequency they are: small petechial surface hemorrhages; subcapsular hematomas appearing as multiple blebs of varying size; lacerations of the capsule, as a rule, caused by the breaking of a subcapsular hematoma; and actual rupture of the liver.

Petechial hemorrhages are rarely absent in deeply asphyxiated infants. Subcapsular hemorrhages do not seem very rare even after spontaneous labors, but more often are found after operative deliveries, in particular after difficult breech labors.

The superficial lacerations of the capsule must be distinctly differentiated from deep ruptures into the liver tissue. Only the latter lead to a serious or fatal intraperitoneal hemorrhage. Capsule lacerations probably often are postmortal injuries produced in careless handling of the corpse. Parenchymatous liver ruptures are comparatively rare. They are usually produced in artificial deliveries or are the result of severe traumatization of the born child (resuscitation, self-help, infanticide).

Hedrén in his 1000 post mortems discovered one hepatic rupture subsequent to a labor, spontaneous in the strictest meaning of the word. It was a small premature baby quickly expelled in breech presentation without aid of any kind. This authentic observation deserves particular attention because Dittrich, a recognized authority in forensic medicine, had asserted that he had never known a rupture of the liver to occur in a spontaneous labor without some definite external trauma.

The following papers and case reports, selected chiefly from recent literature, may serve as good illustrations of the various

etiological factors recognized in the causation of parturitional rupture of the liver: Brodhead (large child born in breech presentation); Dietrich (Kristeller expression made to aid in a high forceps extraction); Friedman (breech extraction); Geill (Schultze's swinging); Hannes (sharp blow against patient's abdomen on the day before labor); Merner (unintentional blow against the abdomen of an asphyxiated newborn), and Nacke (swinging of a child delivered by cesarean section).

Noteworthy in this connection are observations recorded in forensic literature in which the rupture of the liver seemingly was caused by the fall of the baby in precipitate labors while the mother was standing up. Hedrén mentioned one case of this kind described by Bureau and 2 cases of Strassmann. These writers assumed that the jerk on the cord, while the baby is falling, may injure the liver. This theory has been supported by Koehler, who showed in experiments that under certain mechanical conditions a sharp pull on the cord is sufficient to cause a tear of the suspensory ligament which may extend into the liver tissue.

From the viewpoint of diagnosis it is striking that infants, eventually dying from a ruptured liver, apparently as a rule, seem perfectly normal for approximately the first three days of life. Symptoms of serious illness do not manifest themselves until a considerable amount of extravasated blood has reached the peritoneal cavity. Then death is prone to occur suddenly and unexpectedly. Only a post-mortem examination can in these cases reveal the true cause of death.

## SUPRARENAL GLANDS

The lesions found in these glands consist either in parenchymatous hemorrhages or rupture. Of 8 cases mentioned by Hedrén 4 occurred after spontaneous labors and 4 after instrumental deliveries. Schultze's swinging supposedly is an etiologic factor of great importance. However, in many cases the source of the traumatization remains unknown.

In the case of Weltmann, the normal-sized child was born spontaneously, was not asphyxiated and required no resuscitation. It was well for three days, then developed a marked icterus and died on the eighth day. Autopsy showed a very large hematoma of the adrenal body. The only possible explanation for the severe lesion

could be found in the fact that the membranes had ruptured prematurely thus exposing the infant to strong direct pressure from the contracting uterine wall. In a case of Runyan (mentioned by Friedman) both suprarenals were ruptured in the second born of twins, delivered in a breech presentation. A baby observed by Reuss was born spontaneously, took sick on the third day and died. A large amount of blood was discovered in the abdomen originating from a torn suprarenal body. Reuss emphasized icterus as the predominant symptom of such a seeming hemorrhagic diathesis which becomes manifest in an apparently healthy child a few days after birth.

Minor hemorrhages in the suprarenal glands occur very frequently. Most interesting facts have been brought to light in this respect by the systematic study of these glands by Magnus in 124 infants either stillborn or dying within the first eight days of life. In 8 of them hemorrhages were macroscopically evident in one or both glands. As in all cases so far recorded in literature he also found that in the histological picture the destruction of the medullary portion stands in the foreground, the cortex being either intact or only secondarily changed. A systematic microscopic examination, however, convinced him of the justification of Mattei's claim that entirely normal adrenal bodies in a newborn coming to autopsy are the rare exceptions. Mattei had found but 2 normal out of 39 pairs examined. Previous investigations made by Philipp and by Leconte had yielded practically identical results. Magnus thought that the severer lesions are seen almost only after artificial deliveries, and that the 2 observations of fatal injuries after spontaneous labor, recorded by Doerner, represent a most unusual occurrence. In his belief Hengge's assertion that swinging proves particularly dangerous to the adrenals is not well proved because a newborn whose adrenals have been severely traumatized in birth, very likely will offer the clinical picture of asphyxiation and thus probably will be subjected to swinging. In a most thorough study of the entire problem Lundsgaard deduced from certain findings in 2 personally observed cases, that a serious hemorrhage possibly occurs only from an adrenal gland which is congenitally abnormal.

Various writers have speculated on the possible consequences of nonfatal birth lesions of the adrenal glands on the health of the child later in life.

## SPLEEN

In regard to parturitional rupture of the spleen, to the obstetrician the one fact is of greatest importance that without a single exception in every instance so far recorded, the injured spleen was found to have been distinctly pathological, very large and unusually friable. Hedrén's paper gives a review of all cases reported up to that time. I found only one more record of a case described by Heinrichsdorff. It pertains to a normal-sized child, congenitally luetic, born spontaneously and not asphyxiated. In this instance the physiological trauma of a normal birth proved sufficient to produce in the large and soft spleen a 4-centimeter long tear of the capsule on the anterior and upper surface and a 4½-centimeter long deep cleft posteriorly, the latter responsible for a fatal intra-abdominal hemorrhage.

## PANCREAS

According to Ipsen, the studies of Reubold and of Kratter have proved that small disseminated hemorrhages can often be found in the pancreas of infants dying in a state of profound asphyxiation. Quite different from this condition was a large subcapsular and interstitial hematoma of the pancreas seen by Ipsen in a premature infant. There being no microscopic findings in this case to account for the lesion, he had to consider it due to a severe trauma sustained in birth. Hedrén was unable to find another observation of this sort in literature.

## KIDNEYS

While Spencer asserted that he discovered hemorrhages (most often in the loose cellular tissue of the hilum) in almost 20 per cent of his autopsies on newborn infants, serious injuries of the kidneys in birth apparently are of extreme rarity. Hedrén stated that there was but 1 case of rupture of the kidney on record.

## INTESTINES

Practically all the information available concerning the rupture of intestines at birth is embodied in a paper of Sury on 17 cases collected from literature and an additional personal observation. As

a rule the colon is affected. Genersich's case of laceration of the ileum is unique. In the explanation of this accident most writers have laid stress on an abnormal distention of the bowel. Zillner was one of the first to emphasize this point in the etiology. He thought, and most of subsequent observers agreed with him, that the distended gut is actually ruptured by a trauma from without. Paltauf in a careful microscopical study, however, established for his own cases that the coprosthesis in itself is responsible for the eventual rupture. The accumulation of meconium causes severe stretching of the fibers of the muscular coats of the bowel, leading to necrotic processes in this layer. Therefore, the severe trauma assumed to be essential by other investigators, in Paltauf's opinion is not required for the final rupture. However, various observations recorded in recent literature seem to support Sury's view that it is a pathologically affected and distended loop of intestine which is most likely to be ruptured by a trauma incident to spontaneous labor, or more often sustained in an artificial delivery and during manipulation of the born child. Thus Benn reported a peritonitis, evident within the first day of life, terminating fatally on the fourth day. It was due to the incarceration (unquestionably before birth) of a loop of intestines in a defect of the mesentery. Bullowa and Brennan were forced to perform a laparotomy on a newborn child in whom an intestinal obstruction had been caused by an impaction of inspissated meconium. In a case of Owen and Lake an obstruction was evidenced immediately after birth by the extensive distention of the abdomen. It was found due to a band arising from the mesentery and running down to the bladder—seemingly the remains of the vitelline duct. It seems plausible to assume that in a situation of this sort but a slight and unnoticed trauma or even only the compression of the abdomen during its passage through the birth channel would suffice to rupture the excessively distended loop of bowel.

It is possible that other conditions outside of mere distention may favor laceration of the intestines. Bonnaire, Durante and Ecalle saw a newborn baby die from a perforating duodenal ulcer. In their opinion the underlying cause for such an ulcer may be found in local vascular anomalies together with toxic factors, the situation being aggravated in the newborn by the commonly reduced coagulability of his blood. A localized hematoma is prone to develop which might be ruptured by a slight trauma.

Reuss had assumed that the sudden change from the high intra-uterine pressure to a decidedly lower pressure immediately after expulsion might cause hemorrhages in some of the viscera of the newborn. On this basis, Mayer explained an obscure intestinal hemorrhage in a newborn baby in which no evidences of any pathological processes could be discovered.

Fatal intra-abdominal hemorrhages have been observed from the tearing of omental vessels (2 cases quoted by Hedrén), and of umbilical vessels just within the abdominal wall. Obstetrically interesting in this respect is a case of Gaifami. The child was deeply asphyxiated and died in spite of swinging. Necropsy revealed a large peritoneal hematoma around the umbilical vein. The injury, in his belief, was due to traction from the weight of a clamp which had been left on the cord stump during swinging.

### BLADDER

Only ruptures of congenitally malformed bladders have been reported. A typical instance of an accident of this sort was described in detail, e.g., by Boehi.

### UTERUS

The only mention of an affection of the uterus as result of birth trauma is made in the paper of Spencer. He found fairly often a distinct congestion and in 5 (of 130) cases a hemorrhage either in the endometrium or subperitoneal layer. Presumably these findings represent only the ecchymoses commonly seen in asphyxiation.

### TESTICLES AND SCROTUM

Testicles and scrotum are exposed to a definite risk of traumatization in all breech labors. The edematous swelling, occasionally with small superficial hemorrhages, of the scrotum presenting in the cervix is known to all obstetricians. Rather generally this condition is considered of no particular significance. Spencer, however, found that in careful examinations hemorrhages in the testicles as

the result of exaggerated congestion are frequently discovered (in 19 per cent of his cases) as well as small hematomas in the cord, or under the tunica albuginea and tunica vaginalis.

Simmonds, who studied this problem thoroughly, asserted that testicular hemorrhages are not by any means uncommon and not limited to infants born in breech presentation. Often the presence of such hemorrhages can be ascertained only by a microscopic examination, though in the more pronounced cases they can be readily recognized by the increased size and tenseness of the organ and its characteristic dark reddish-blue color. Usually the hemorrhage also involves the epididymis. Most often the condition is bilateral. If autopsies are made on very young infants the extravasated blood is seen between the convoluted tubules. In children dying later, the epithelium of those tubules, which are embedded in the hematoma, show necrotic changes. The possibility of a permanent, and at least partial, destruction of one or both glands as the result of a trauma sustained in birth, is strongly suggested by the occasional finding, in older children, of a circumscribed fibrous focus within the testicular tissue.

In breech cases testicles are known to have been severely injured by rough and unskilled examination during labor. Stumpf mentions a case, reported by Meurer, in which a scrotum greatly enlarged by a hydrocele, had been mistaken for the unruptured bag of waters and perforated with an instrument.

## FEMALE EXTERNAL GENITALIA

Royster described the repair of a completely lacerated perineum in a girl nine years old, produced at birth by the attending obstetrician. The child's grandfather, a very aged physician, had acted as accoucheur. This type of injury is not so rare as is assumed by Royster. Identical observations have been recorded, e.g., by Sawicki, Neugebauer, and 2 cases by Dittrich. The origin of such an injury is almost typical. The anus is mistaken for an unyielding cervix which in the judgment of the attending physician or midwife requires forcible digital dilation. In most instances in this manner complete perineal lacerations are produced extending far up into the rectum.

Rather uncommon is the etiology of a shallow perineal tear,



seen by Dittrich, in his opinion, the result of traction and rotation of the thigh during version and extraction.

Older obstetrical literature frequently mentioned injuries both of the male and female external genitalia and of the groin produced by the blunt hook of Smellie which used to be extensively employed in the extraction of the presenting breech. This very danger prompted obstetricians to discard the hook. Also the use of a finger for this purpose implies the risk of injuring the region of the groin or fracturing the thigh. Forceps specially constructed for application on the breech have not proved practical. The use of the head forceps on a breech must be deprecated. It is claimed that in this manner in several instances either the pelvis or thighs of infants have been fractured.

#### LITERATURE

- Ahlfeld. *Ztschr. f. Geburtsh. u. Gynäk.*, Stuttg., 1911, 68:131.  
 Benn. *Abst. in Zentralbl. f. d. ges. Gynäk. u. Grenzgeb.*, Berl., 1914, 5:209.  
 Bonnaire, Durante and Ecalles. *Gynécologie*, Par., 1914, 18:161.  
 Boehi. *Arch. f. Gynäk.*, Berl., 1914, 101:700.  
 Brodhead. *Am. J. Obst.*, N. Y., 1917, 75:1067.  
 Bullowa and Brennan. *J. Am. M. Ass.*, Chicago, 1919, 73:1882.  
 Dietrich. *Zentralbl. f. Gynäk.*, Leipz., 1913, 37:1002.  
 Dittrich. *Vierteljahrsschrift für gerichtliche Medizin*, 1895, 9:234.  
 Friedman. *Am. J. Obst.*, N. Y., 1917, 75:1068.  
 Gaifami. *La Gynecologia*, Firenze, 1913, 9:497.  
 Geill. Original in Danish, *abst. in Monatschr. f. Geburtsh. u. Gynäk.*, Berl., 1899, 10:658.  
 Hannes. *Berl. klin. Wchnschr.*, 1914, 51:31.  
 Hedrén. *Vierteljahrsschrift für gerichtliche Medizin*, 1917, 54:230.  
 Hedrén. *Laekaresaellskapets Handlingar*, 1918, 44 (Author's abstract in German).  
 Heinrichsdorff. *Monatschr. f. Geburtsh. u. Gynäk.*, Berl., 1914, 40:417.  
 Hengge. *München. med. Wchnschr.*, 1904, 51:2134.  
 Ipsen. *Zentralbl. f. Gynäk.*, Leipz., 1907, 31:1358.  
 Lesser. *Vierteljahrsschrift für gerichtliche Medizin*, 1910, 39:415.

- Lilla. Original in Italian, abst. in München. med. Wehnschr., 1910, 57:1468.
- Lindner. Vierteljahrsschrift für gerichtliche Medizin, 1882, 36:242.
- Lundsgaard. Virchow's Arch. f. path. Anat., Berl., 1912, 210:164.
- Magnus. Berl. klin. Wehnschr., 1911, 48:1119.
- Mayer. Zentralbl. f. Gynäk., Leipz., 1915, 39:795.
- Merner. Vierteljahrsschrift für gerichtliche Medizin, 1882, 36:226.
- Nacke. Ztschr. f. Geburtsh. u. Gynäk., Stuttg., 1911, 68:713.
- Owen and Lake. Brit. J. Child. Dis., Lond., 1920, 17:115.
- Paltauf. Virchow's Arch. f. path. Anat., Berl., 1888, 111:461.
- Reuss. Wien. klin. Wehnschr., 1912, 25:859.
- Royster. Am. J. Obs., N. Y., 1902, 45:405.
- Schwab. Zentralbl. f. Gynäk., Leipz., 1908, 32:69.
- Simmonds. München. med. Wehnschr., 1910, 57:1367.
- Spencer. Transactions of Obstetrical Society, London, 1891, 33:203.
- Stumpf. Winckel's Handb. der Geburtsh., 3:3, p. 628.
- Sury. Vierteljahrsschrift für gerichtliche Medizin, 1912, 43, Supplement II, 91.
- Weltmann. Wien. klin. Wehnschr., 1911, 24:728.

## CHAPTER XVII

### INJURIES OF THE LOWER EXTREMITIES

Intra-uterine fractures—Fractures of femur—Injuries of hip-joint—Fractures of tibia and fibula—Luxation of knee—Injury of foot—Paralysis of lower extremity.

So-called intra-uterine fractures more often are found to affect the lower than the upper extremities. As already explained in Chapter XIV, they have to be regarded of two kinds: true fractures due to a definite trauma from without, and pseudofractures. The latter, beyond question the commoner type of the two, represent only an angular bending of a long bone of the lower extremity occurring at a time when ossification has not yet begun or is far from completed. According to more recent conceptions the main causative factors of pseudofractures are want of room within the uterus (oligohydramnion, a protruding fibroid, etc.) or amniotic adhesion bands. What seems in the X-ray picture to be a callus and tends to prove an actual fracture, might in some instances represent only a thickening from a circumscribed periostitis. The occasional deficient development of the extremity peripherally to the assumed site of fracture, and presumably the sequela of the injury (see Chapter XIV), by other authors is explained as the result of blood circulation interfered with by the pressure of an amniotic band. There is much evidence available to support the claim of some writers that both bending or fracture during intra-uterine life are dependent upon either an osteogenesis imperfecta or an osteopsathyrosis. These also are the conditions which cause one or many of the bones of the skeleton to break in the course of normal and spontaneous labors. It seems obvious that in the presence of such a pathological fragility of the bones an infant presenting by the breech will have but little chance to escape a fracture of his lower extremities.

**Fractures of the Femur.**—Among the fractures of the lower extremity in the newborn those affecting the femur are the most common. As a rule the bone breaks transversely in its upper half or third, where it is relatively the thinnest.

Version and extraction offer most favorable opportunities for the production of this injury. During version: when the accoucheur incorrectly grasps the thigh instead of the foot; when the one leg is pulled down over the other lying crossways, or when a version is made a long time after the escape of the amniotic fluid and severe traction is required to effect the turning of the fetus. In a breech presentation: when a leg is pulled down while the breech already is partially fixed in the pelvic inlet, or when during manual aid in the delivery of the shoulders and arms the child, improperly, is held by one thigh.

In older medical literature many references can be found to thigh fractures produced with the blunt hook or the flexible fillet, formerly more often than at the present time (at least in this country) employed for the extraction of the arrested breech.

Gauss presented some interesting figures in regard to traumas incident to these methods of breech extractions. Jolly, in 139 operatively terminated breech labors, encountered 5 femoral fractures (3.6 per cent). Ponfick, in 16 breech presentations ended with the hook, saw 1 thigh fracture and 2 deep wounds of the groin requiring sutures; Henkel noted fractures of the femur in 7.7 per cent of the breech extractions made in his clinic. A strikingly high frequency of 22.5 per cent of femoral fractures after breech extractions was reported by Mueller, 18.7 per cent produced by the hook, 3.8 per cent by the sling. Majocchi discovered 6 femoral fractures in 59 breech labors terminated with the hook, and none among those terminated with the forceps. Gauss stressed this latter fact and with his personal experience claimed that the forceps might be most successfully applied to the impacted breech, if only the instrument is applied and the tractions made strictly in accord with a technic described by him in detail. In 9 breech cases terminated instrumentally by him, he had 1 femoral fracture from the blunt hook, another from a forceps, in his belief solely due to a faulty technic. Of 7 fractures reported by Weltmann, 4 occurred after version, 1 in a head presentation, and 2 in breech extractions, in 1 of the 2 cases, accomplished by digital extraction of the arrested breech.

Femoral fractures caused by digital extraction of the breech have been repeatedly reported. Packard stated that he had personal knowledge of several cases of this kind. Abt (according to Weltmann) saw 3 cases in which the thighs had been broken by

digital extraction. Many other single observations could be quoted.

Obstetricians agree that also this method of breech extraction is not void of danger to the fetus, that the risk, however, can be minimized if only one finger, never two, is hooked into the groin, and if during traction proper care is taken that all pressure is directed against the infant's pelvis.

While femoral fractures undisputably are more prone to occur, and indeed are fairly frequent, in artificially terminated breech labors, literature contains many reliable records of thigh fractures sustained in spontaneous breech labors and even in perfectly normal head presentations.

Green found both femurs broken in a small premature child, spontaneously expelled in face presentation. In this case the fractures were caused by the tight coiling of the umbilical cord around both thighs, clearly evidenced by visible pressure marks. The thighs thus had been held in fixed flexion and broke when they were forced into extension during passage through the pelvic canal. (This same case has also been described by Henning in an Inaugural Dissertation, and for this reason in literature appears erroneously as two different observations). Engel (quoted by Jaeger) observed a fracture of both femurs after a breech labor in which the child had been expelled unaided after a few strong contractions. Stumpf mentioned a case seen by Vaneček in which the femur of a leg, that had prolapsed on the side of the presenting head, had broken spontaneously.

The most reliable proof of the possibility of a spontaneous fracture of the femur in the course of labor—a point of great forensic importance—is the striking frequency with which one meets in the reports with the positive assertion that a distinct crack or thud had been heard before the child was actually delivered. Thus, e.g., Stumpf noticed a "loud and distinct crack" in the moment when in a perfectly normal head presentation the trunk passed through the pelvis. This "thud" was heard in a breech case by Gruwell, and is mentioned by others. It was clearly perceived by Piorte, who only much later was able to determine that it had been caused by the detachment of the upper epiphysis of the femur.

As for most of the birth injuries also for femoral fractures at present no reliable information is available concerning their actual frequency. Reports of individual observations for various reasons are not likely to appear in literature. It certainly is a noteworthy

fact that, e.g., in 1893 a short letter by Hubbard to the editor of the *Medical News* (Philadelphia), concerning an observation of this injury, prompted Gruwell and Davidson (also only in form of letters) to place on record several other cases of this sort.

Only recently I personally experienced how easily a femur might be fractured. In a cesarean section, performed at the onset of labor for a contracted pelvis, the median incision in the anterior uterine wall was made too short on account of a fibroid unexpectedly encountered in the lower portion of the wall. The right leg of the large child was readily lifted out and during the somewhat difficult extraction of the breech a distinct snap or crack was felt, the left femur, still within the uterus, had been fractured approximately in its middle. I have no doubt that other operators must have met with the same accident, though I am not aware of any other report of a fracture of the femur during a cesarean section.

As a rule the fracture causes a distinct and unmistakable deformity of the thigh. Nevertheless, among the comparatively few reports I found 2 in which the injury was discovered only later. In a case of Fowler (mentioned by Ballantyne) the fracture was revealed four days post partum by the considerable swelling of the thigh. No injury had been suspected because the child was born spontaneously in a head presentation. In an infant seen by Young (cited by Hubbard) the fracture was not discovered until twelve days after labor when continued crying of the baby finally led to a careful examination.

Roentgenograms almost invariably show some displacement of the fragments (also in my case), and for this reason every case of femoral birth fracture requires proper attention to preclude a shortening or permanent deformity of the affected leg.

No attempt will be made to outline such an appropriate form of therapy in this monograph. As a matter of fact there exists even among expert orthopedists some difference of opinion concerning the respective usefulness of fixation of the fragments by means of splints or a plaster cast, a combination of splint with traction, and mere fixation of the thigh in forced flexion against the infant's abdomen. Definite difficulties are experienced in the management of a femoral fracture in the newborn, because it is practically impossible to protect any permanent bandage or cast from soiling, and because it is essential for proper nutrition that the child can be placed to the

mother's breast. Among recent writers the various methods of treatment are discussed by Blair, Facchin, Le Grand and Reuss.

**Injuries of the Hipjoint.**—Formerly it was the prevalent idea that the most important cause of nontraumatic *hipjoint dislocations* of young children was to be found in various obstetrical manipulations, the injury remaining unrecognized until much later in life and revealed by the first attempts to walk. To-day there exists rather general agreement that the congenital type of hipjoint dislocation is due to a true malformation, consisting essentially in an arrest of development of the acetabulum, and perhaps secondarily in the malformation of the femoral head. The acetabulum remains small and the head cannot lodge in it. According to Keith (Birnbau) this arrest of growth manifests itself as early as the third month of fetal life. The exact time when the actual dislocation takes place is not known. It seems possible that in rare instances it might occur during delivery as the result of traction on the leg. That the dislocation in the overwhelming number of cases, however, existed long before labor is clearly established by certain common radiographic findings: a very small and shallow acetabulum, and a malformed, flat, misplaced femoral head. Only recently (1921) Sippel placed on record a third case of congenital hipjoint dislocation carefully studied in X-ray pictures immediately after birth (previous studies had been made by Joachimsthal and Bader). As in congenital torticollis the deformity seems the result of undue pressure on account of limited space. The apparent prevalence of congenital hipjoint luxation subsequent to breech labors probably can be ascribed to the usually oblique position of the uterus in breech presentations. In the case of Sippel this pronounced obliquity might have been the result of unequal shortening of the round ligaments in a preceding Alexander-Adams operation.

In preoentgen times more often than of late the traumatic *detachment of the upper femoral epiphysis* is mentioned in literature (various authors are quoted by Weltmann, 2 cases have been reported by Foersterling). Some writers, on the other hand, on theoretical grounds have denied the possibility of such an accident in birth. At least in 1 case of Pforte, who discussed this problem very thoroughly, the existence of the injury was definitely established at a later autopsy. Also he quoted several observations of older literature, e.g., a case of Carus' of intra-uterine abruption of this epiphysis supposedly caused by a heavy fall of the mother in the fifth

month of pregnancy. Piorte thought that one may justifiably expect that an injury of this kind unrecognized at birth, in some instances represents the true origin of a seemingly congenital coxa vara. The investigations of Gurlt, Sturrock and Bruns have shown that the traumatic detachment of the epiphysis in children is the result of strong traction with simultaneous rotation or strong abduction. Gurlt's experiments on cadavers of newborn babies seemed to prove that this injury cannot be produced by traction alone.

In performing a version such a dangerous combination of traction with abduction might be unavoidable, but during traction on the already born leg a simultaneous rotation or forced abduction are clearly objectionable maneuvers.

**Fractures of Tibia and Fibula.**—Practically without exceptions these fractures are produced in the management of breech labors. Jaeger quoted an observation of Hoffa. Fracture of both bones had led to the formation of a pseudoarthrosis. The delivery having been reported to have been normal in this child, Hoffa assumed that the leg was fractured while the foot was forcibly pressed during labor against the lateral pelvic wall. Hayken saw a baby born with a fixed angulation of the tibia, possibly the result of a fracture sustained during a fall of the mother in the sixth month of pregnancy. However, in this case various other etiological factors cannot be excluded, since the baby exhibited a syndactylia and hyperdactylia on the same extremity, and furthermore at birth the cord was found tightly coiled around it 4 times. Buengner had collected from literature 10 observations of parturitional fractures of both bones of the foreleg. Twenty-nine reports from literature and 2 additional personal observations formed the basis of an exhaustive consideration of this trauma in a paper by Hayashi and Matsuoka. Unfortunately they were using the term "congenital" and "intrapartum" promiscuously in regard to the origin of the lesion, and therefore, do not offer any clear views concerning the actual etiology of these fractures. Noteworthy is the anatomic finding that the break always seems the result of exaggerated bending, since in no instance a comminuted fracture has been observed. Of still greater value are the results of anatomic studies made by Bruns and Messerer, quoted by Weltmann. There usually are two separate fracture lines, separating forklike from the anterior aspect of the leg backwards. This fact permits a definite explanation of the mechanical factors which directly lead to the fracture. If during manual aid in a breech labor



both legs of the infant, improperly, are grasped in one hand, so that four fingers lie anteriorly and the thumb crossways posteriorly, undue pressure by the thumb will bend the bones forward and eventually cause them to break. The space between the separated fracture lines corresponds exactly to the position of the thumb during this procedure. As a matter of fact in most cases of parturitional fracture of tibia and fibula, recorded in literature, the angle forming between the upper and lower fragments was found pointing backwards. This angle, and with it the deformity of the foreleg, is exaggerated by the traction exerted by the relatively much stronger muscles of the calf attached to the heel.

**Luxation of Knee.**—This is an injury but very rarely seen in the newborn. A case of McGillicudi, often quoted by writers, is so indefinitely described in the original publication that it hardly can be analyzed in its bearing on the problem of etiology or even symptomatology. The child was born in a breech presentation with both limbs reflected on the body. On the left leg the knee was found "dislocated." "The dislocation reduced itself naturally and the knee became all right."

Another observation, reported in detail by Offergeld, offers valuable suggestions. In a breech labor, with the lower portion of the infant spontaneously born and its upper portion still within the pelvis, a striking anomaly in the position of the left foreleg could be noticed. Bent at the knee, it was lying along the lateral aspect of the thigh, parallel to it, the toes looking forward, the big toe being on the outside. Careful examination showed a lateral dislocation of the patella together with the tibia. The ligamentum collaterale fibulare (longitudinale externum) and the ligamentum patellare proprium were intact. A distinct depression indicated that the ligamentum collaterale tibiale was torn. Offergeld assumed that in this case the foreleg was held bent in the knee against the thigh and slightly abducted, a common attitude in pelvic end presentations. If for some reason the foot or the heel on account of limitation of space would become firmly fixed, just such a luxation of the knee would result, when the trunk is forcibly driven down into the pelvis by strong uterine contractions. In general this mechanism would closely resemble the one presumably responsible for an identical injury of the knee occasionally seen in the adult as the result of a forceful impact directly against the foreleg.

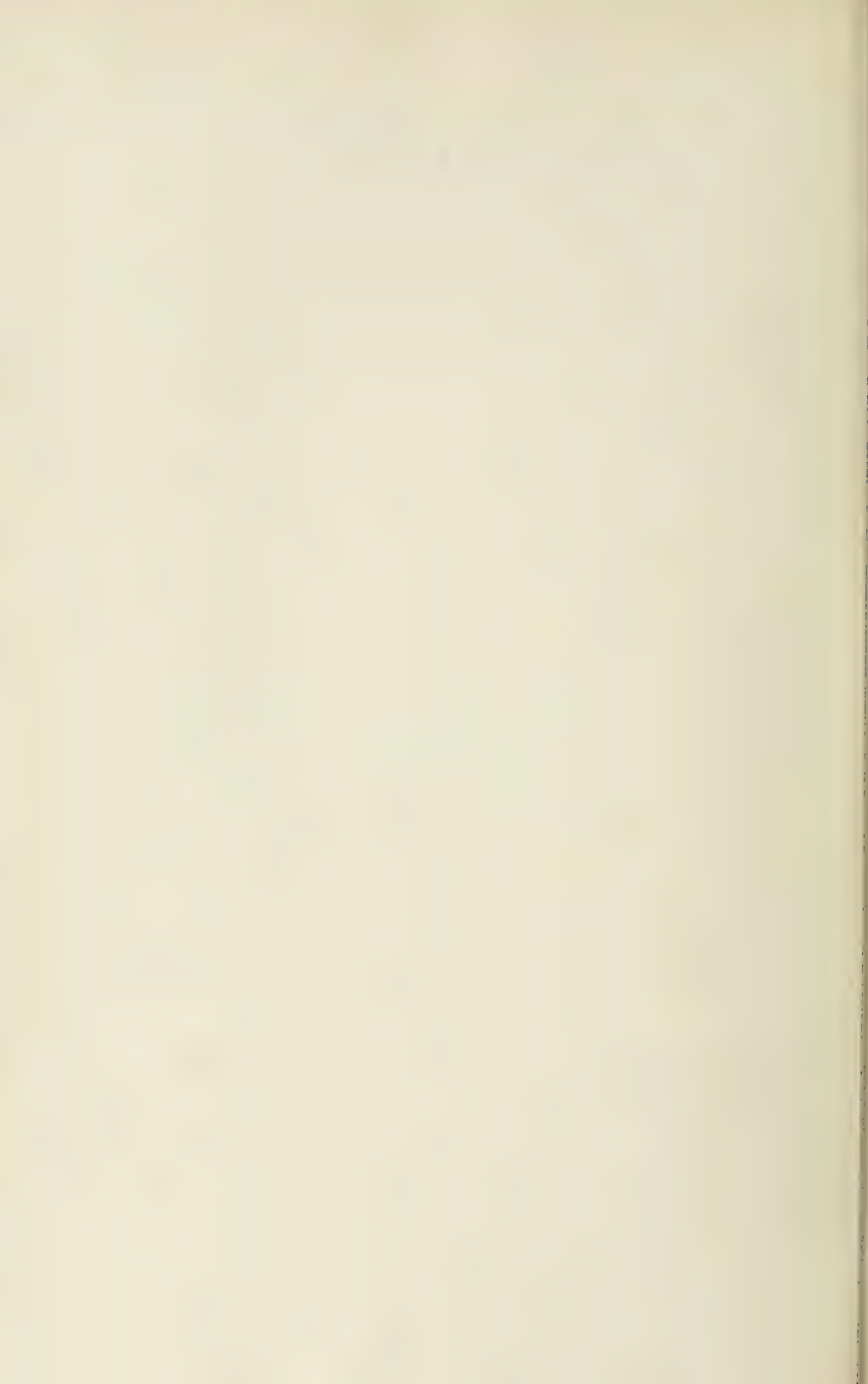
**Injury of the Foot.**—Stratz mentions that he and Treub occasionally use volsella, hooked to the foot, for the purpose of bringing down a leg when a podalic version must be performed through an only partially dilated cervix. In one instance the death of a premature infant, delivered in this manner, seemed to have been hastened by a severe hemorrhage from the wounds inflicted by the tenaculum forceps.

**Paralysis of the Lower Extremity.**—In preceding chapters paralysis of one or both lower extremities has been mentioned as a symptom of intracranial, vertebral or spinal cord injuries during birth. Distinctly different are palsies or merely paretic conditions of muscle groups innervated by a single nerve trunk. Literature contains only a few carefully described or studied observations. Many of them are cited by Reuss. Some writers ascribed such conditions to congenital maldevelopment of nerves or muscles, and others assumed an injury of the lumbar plexus by excessive traction comparable to similar traumatization of the brachial plexus. Öpenheimer reported a paralysis of the femoral nerve seen in a newborn after a breech labor. Bernhardt observed several cases of palsy in the area of distribution of the sciatic nerve after extraction by the foot. A peroneus palsy has been reported by Peltessohn, the result of pressure exerted by an amniotic adhesion band which had left a deep depression across the leg just below the knee. Bruns expressed the opinion that such circumscribed paralyzes in the lower extremity of the newborn infant probably are not uncommon, and that they are overlooked or not reported because they are likely to disappear promptly.

#### LITERATURE

- Ballantyne. Antenatal Pathology. Foetus, 1902, p. 48.  
 Bertkau. München. med. Wchnschr., 1912, 59:1715.  
 Birnbaum. Malformations of the Foetus, Blakiston, 1912.  
 Blair. Surg., Gynec. & Obst., Chicago, 1914, 18:640.  
 Davidson. Med. News, 1893, 62:614.  
 Facchin. Arte Ostet., Milano, 1913, 27:200.  
 Foersterling. Inaugural Dissertation, 1898, abstr. in Jahresb. ü. d. Fortschr. in d. Geburtsh. u. Gynäk., 1899, 12:989.  
 Gauss. Ztschr. f. Geburtsh. u. Gynäk., Stuttg., 1905, 55:502.  
 Green. Ibidem, 1899, 41:522.

- Gruwell. *Med. News*, 1893, 62:531.
- Hayashi and Matsuoka. *Arch. f. klin. Chir.*, Berl., 1912, 98:413.
- Haykens. *Nederl. Tijdschr. v. Geneesk.*, Amst., 1912, 2:14.
- Hubbard. *Med. News*, 1893, 62:323.
- Jaeger. *Gynäk. Rundschau*, Vienna, 1912, 6:511.
- LeGrand. *Gynécologie et Obstétrique*, Paris, 1920, 1:527.
- McGillicudi. *J. Am. M. Ass.*, Chicago, 1892, 19:107.
- Offergeld. *Deutsche med. Wchnschr.*, Berl., 1900, 35:1703.
- Packard. *Keating's Cyclopedia of the Diseases of Children*.
- Peltesohn. *Zentralbl. f. Gynäk.*, Leipz., 1912, 36:1100.
- Pforte. *Gynäk. Rundschau*, Vienna, 1908, 2:285.
- Reuss. *Krankheiten des Neugeborenen*, Springer, Berl., 1914, p. 166.
- Sippel. *München. med. Wchnschr.*, 1921, 68:1221.
- Stratz. *Zentralbl. f. Gynäk.*, Leipz., 1900, 24:739.
- Stumpf. *Winckel's Handb. der Geburtsh.*, 3:3, p. 512.
- Weltmann. *Inaugural Dissertation*, Breslau, 1917.



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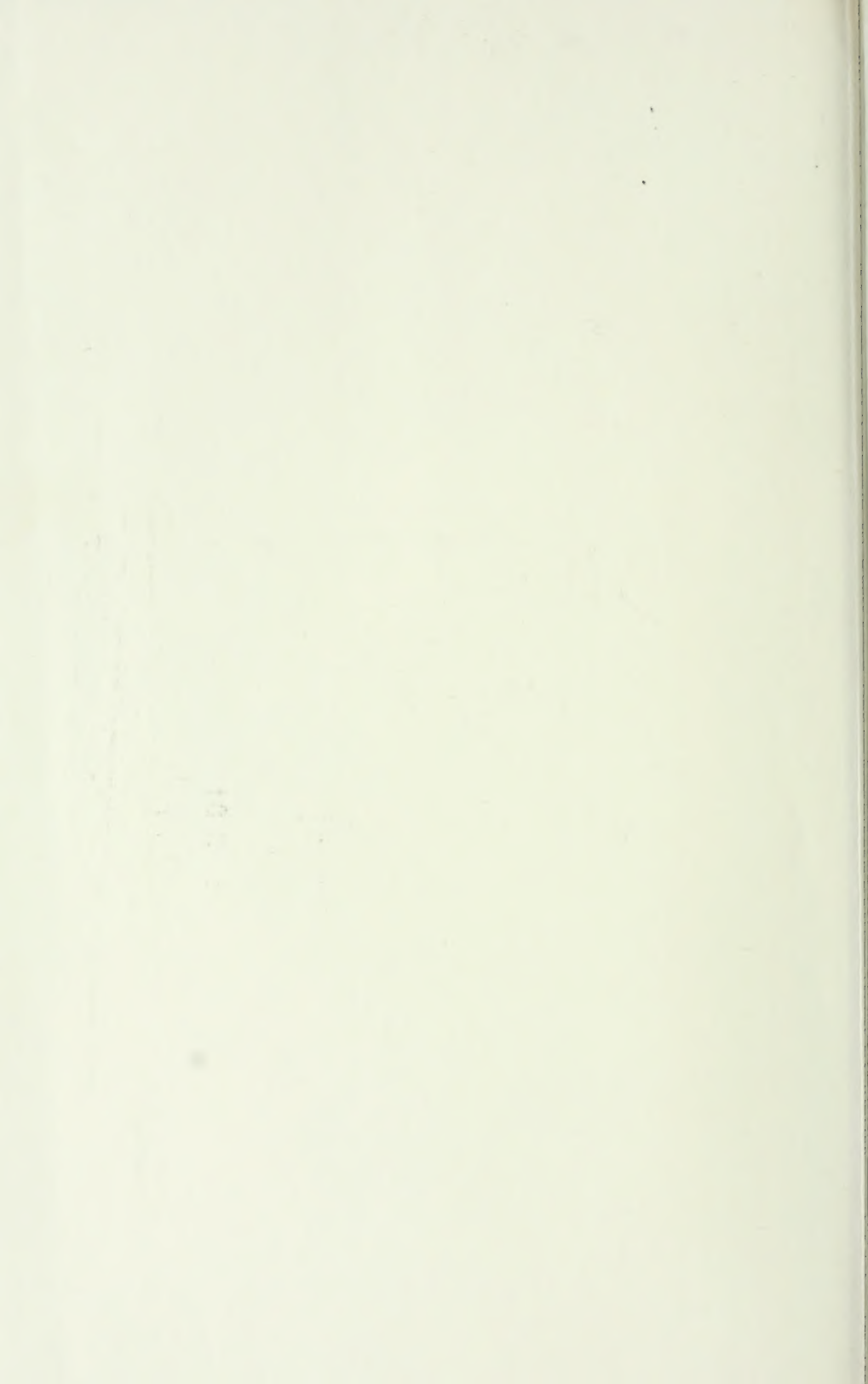
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