Birth Trauma and Perinatal Brain Damage

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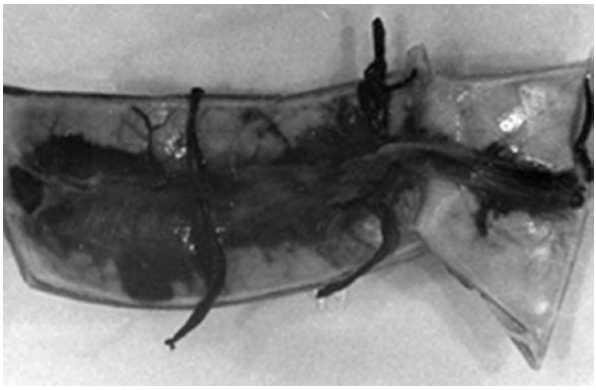

Fig. 7.29 A superior sagittal sinus with parts of parietal bones (view from below). Congested bridging veins detached from the arachnoid membrane originate from the sinus on both sides

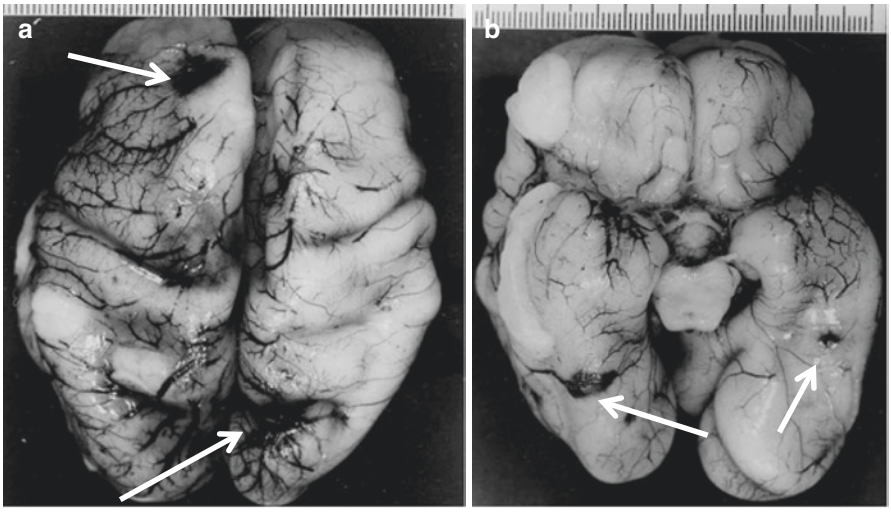

Fig. 7.30 Finely spotted SAH in the sites of origin of the bridging veins (arrows) in a neonate weighing 1100 g, who lived for 35 h. (**a**) View from above, (**b**) view from below

7.10 Compression of the Skull, Brain and Increased Intracranial Pressure

With the head configuration following its compression in the parturient canal, the skullcap bones are displaced, the fetal head changes its shape, the brain is compressed with its parts displaced under the dura mater due to the brain flexibility, and "displacements of the brain mass" occur [26]. Bridging veins that older infants and adults do not have (surface veins end directly in sinuses and do not form "bridges" between a cerebral hemisphere and sinus) make possible these displacements of the brain with the head configuration without any ruptures of veins or cerebrovascular disturbances. Normally there is a subdural fluid

(effusion) under the dura mater, which ensures sliding of the cerebral hemisphere covered with the arachnoid membrane under the dura mater. The pressure of the skullcap bones is directly transferred to the brain surface, and, with excessive head configuration, the reserves for displacements of the brain mass are exhausted when the brain itself and its membranes and vessels are compressed, and the circulation of blood and liquor is impaired. We can define this condition as *cerebral compression*. What are the signs of this compression though? Ruptures of the cerebellar tentorium, falx, and veins would only suggest a possibility of compression of the brain. Cerebral compression and increased intracranial pressure (ICP) can occur with subdural hemorrhage caused by these ruptures. But this "compression" does not correspond to the compression of the brain in the process of delivery. *The compression of the brain in labor can occur without any ruptures and hemorrhages, causing hypoxia and ischemia of the brain tissue*. The condition of such fetuses is often clinically evaluated in error as "intrauterine hypoxia" or "asphyxia"; although oxygen saturation is not disturbed, all internal organs receive sufficient oxygen. Only the brain suffers from ischemia caused by the birth injury – its compression.

Gunther [27] identifies explicitly this kind of asphyxia as "traumatic asphyxia," which he explains precisely by substantial compression of the brain in labor, cerebrovascular disturbances, and hypoxia of brain tissue.

With an intrauterine pressure of 40 mmHg at the end of delivery period 1, the contracting myometrium presses on the presenting part of the fetus with a force of 4.1 kg and with an intrauterine pressure of 140 mmHg with a force of 14.5 kg [28].

Compression of the skull results in *increased intracranial pressure* (ICP), venous congestion, and compression of the veins, sinuses, arteries, and brain matter. There is some evidence in the literature of a close correlation between the value of intracranial pressure and the development of cerebral injuries, in particular, hemorrhages, as well as the infant survival rate. No neonate with an ICP of over 13 mmHg would survive [29]. At the same time, in case of premature infants, even a slight compression of the head with a tape when putting on a photo-therapeutic eye mask causes an increased ICP and may have an impact on the pathogenesis of intracranial hemorrhages [30]. The increased ICP is often accompanied by an increase in the pulsation index, indicating a decrease in the diastolic flow in the cerebral arteries, which is typical of venous congestion. Not only compression of the head in the parturient canal (with the configuration) but also vascular microcirculation disorders, edematous hemorrhagic phenomena in the brain tissue, damage to the hematoencephalic barrier, secretory-resorptive disorders, etc. may cause an increase in the ICP. Experimental studies with measured compression of the heads of dog fetuses [31, 32] showed that an increase in ICP results in bradycardia, lower volumetric blood flow and oxygen partial pressure in the pallium, the suppressed rate of oxidative phosphorylation in mitochondria, etc. The ICP of 18 mmHg and higher is fatal for a fetus. Therefore, an increase in the intracranial pressure causes hypoxic damage to the brain tissue. There is evidence that with an ICP of up to 55 mmHg the fetal heartbeat is stable, but if the ICP increases over this value, the heart rate decreases and bradycardia occurs [33].

The cerebral compression, which is often not taken into account as a sign of a birth injury, is observed in the autopsy of the dead mainly in mature, large fetuses and neonates. Signs of cerebral compression are found in 56.3% of dead fetuses and neonates delivered with obstetric cavity forceps and in 40% of dead infants delivered with output forceps [8]. Compression often causes significant overlapping of the parietal bones over the frontal and occipital bones. In the areas of these overlappings along the coronal and lambdoid sutures, the edges of the subjacent bones going into the cranial cavity formed characteristic *sulci of compression* on the surface of the hemispheres or the so-called steps (Figs. 7.31 and 7.32). There might be no ruptures of the cerebellar tentorium, falx, and veins, and only the existing sulci of compression can help to substantiate the diagnosis of a birth injury. Subarachnoid and intracerebral hemorrhages can be detected along the lines of compression. Cerebellar hemorrhages may occur in the compressed occipital lobe of the brain.

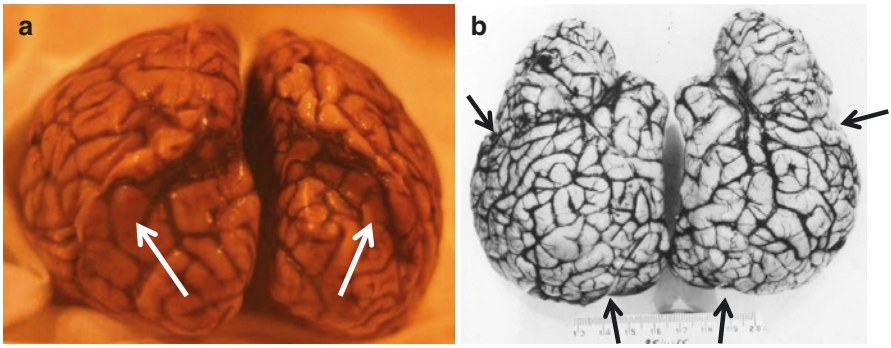

Fig. 7.31 Sulci of cerebral compression (arrows). (**a**) A sulcus of occipital lobe compression corresponding to the bone overlapping along the lambdoid suture. (**b**) Cerebral compression in an intranatally dead fetus with a mass of 3100 g extracted with obstetric forceps. The arrows show the sulci of compression corresponding to the coronal and lambdoid sutures with overlapping of the parietal bones over the frontal and occipital bones

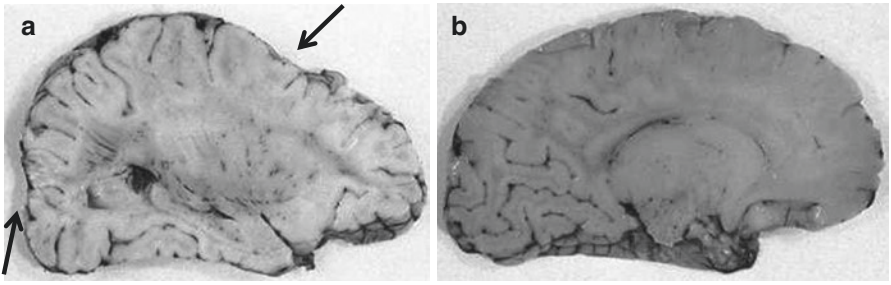

Fig. 7.32 (**a**) The view of the cerebral hemisphere in the sagittal section with cerebral compression (arrows). The frontal lobe is on the right, the occipital one is on the left. (**b**) A cross section of the brain in the sagittal section without any signs of compression

Sulci of compression may be invisible in case of rough extraction of the brain out of the skull and its turning on the dissection table. Premature infants under 32 weeks of gestational age have no signs of cerebral compression in the form of corresponding sulci. This is due to the fact that the skull grows faster than the size and mass of the brain increases. Extremely premature infants always have some gap between the inner surface of the cranial bones and pia mater during cephalotomy, so the lower scissor jaw does not damage the meninges. In term and post-term children, opening the skull with scissors and removing the bony plates of the parietal bones is usually accompanied by damages to the brain and its membranes. Therefore, bone displacements in mature infants cause pressure on the brain surface. Bone displacements in premature infants are accompanied by stretching of the tentorium and falx and bridging veins and only occasionally by cerebral compression. Displacements of the brain mass with cerebral compression in premature infants often lead to subpial hemorrhages on the surface of the gyri due to the separation of the pia mater from the molecular stratum of the pallium [7].

The cerebral compression occurs with excessive head configuration and is accompanied by its axial dislocation with characteristic signs of cerebellum tonsil compression in the great foramen. So we can detect sulci of compression around the cerebellum tonsils and sometimes also sulci on the superior surface of cerebellar hemispheres (resulted from the pressure by the edge of the tentorium) or macular subarachnoid and subpial hemorrhages along these sulci. The compression causes ischemia and hypoxia of the brain tissue, but it is clinically manifested by the symptoms characteristic of "fetal asphyxia."

The cerebral compression is accompanied by narrowing of the superior sagittal and transverse sinuses, compression of the superficial cerebral veins, stretching of the bridging veins and the vein of Galen, pressure by the edges of the tentorium onto the superior surface of the cerebellar hemispheres, elongation of the brain in the direction of the birth axis according to the head configuration, elongation of the brainstem, an excessive bend of the spinal bulb in relation to the cervical cord in combination with the compression of the cerebellum, elongation of the arterial circle of Willis, narrowing of the great foramen, etc. [1, 26]. The venous drain in the sinuses and veins gets impaired, intracranial pressure gradually increases, etc.

Not only disturbed uterine-placental and funicular circulation but also the excessive head configuration with cerebral compression (birth injury) resulting in hypoxic-ischemic encephalopathy can cause intrauterine fetal hypoxia. The ICP increases, the venous drain is impaired, the brain matter is compressed, and its ischemia develops. The available data indicate an interlinkage of hypoxic and traumatic factors, as well as the significance of increased ICP in the pathogenesis of cerebrovascular disturbances.

Subarachnoid and intracerebral hemorrhages can be detected on the r in the compressed occipital lobe of the brain (Fig. 7.33).

Sulci of compression may be invisible in case of rough extraction of the brain out of the skull and its turning on the dissection table. Premature infants of the second and third degree have no signs of cerebral compression in the form of corresponding sulci. This is due to the fact that the skull grows faster than the mass of the brain increases. Extremely premature infants always have some gap between the inner surface of the cranial bones and pia mater during cephalotomy, so the lower scissor jaw does not

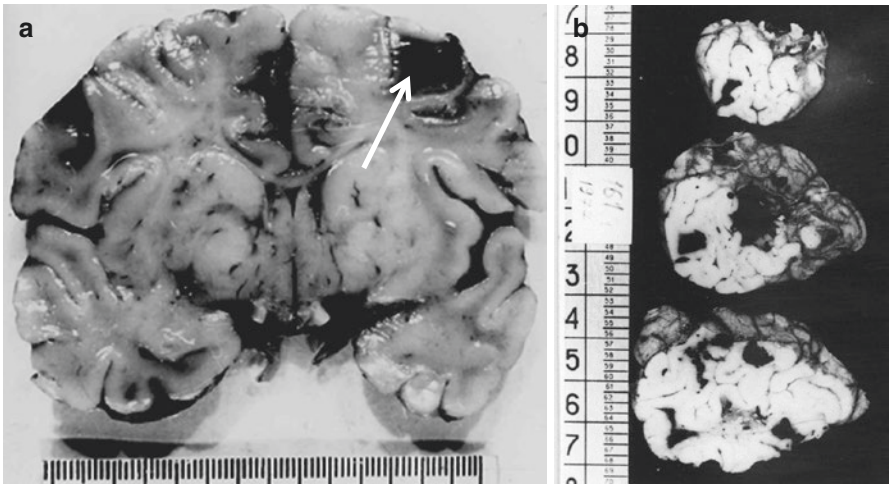

Fig. 7.33 (**a**) ICH in the right brain corresponding to the coronal suture along the line of cerebral compression (arrow). (**b**) ICH in the occipital lobe with compression along the lambdoid suture

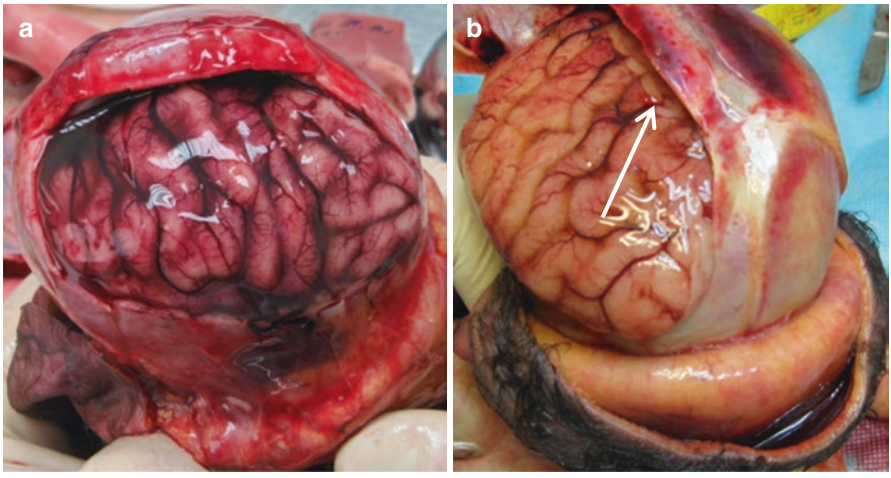

Fig. 7.34 (**a**) The right brain of a premature infant weighing 1500 g, accumulations of liquor in the subdural space. (**b**) The right brain of an infant weighing 3650 g, who died from a birth injury. We can see a line of damage to the brain and meninges by the scissors jaw (arrow). The brain bulges out of the gap formed in the skull, the gyri are smoothed

damage the meninges. As already indicated, in connection with the fact that the bones of the skullcap in full-term and post-term fetuses and newborns are directly adjacent to the convective surfaces of the brain, then when the skull is opened, the membranes and brain are damaged (Fig. 7.34b). Therefore, the expression of bone displacements in mature infants cause pressure on the brain surface. Bone displacements in extremely premature infants are accompanied first of all by stretching of the tentorium and falx and bridging veins and only occasionally by cerebral compression.

The cross overlapping of bones observed mainly in premature infants prevents their excessive displacement (Fig. 5.3). This bone displacement is a natural protection for the extremely vulnerable brain of premature infants. The higher the degree prematurity is, the bigger is the water content in the brain matter and the more vulnerable the brain is to physical impacts. With a low degree of prematurity (the first), pressure on the brain surface causes displacements of the pia mater on the apexes of the gyri and subpial hemorrhages (see below).

Compression of the skull and brain results in stretching of the cerebral falx and cerebellar tentorium, which may rupture (Fig. 7.35).

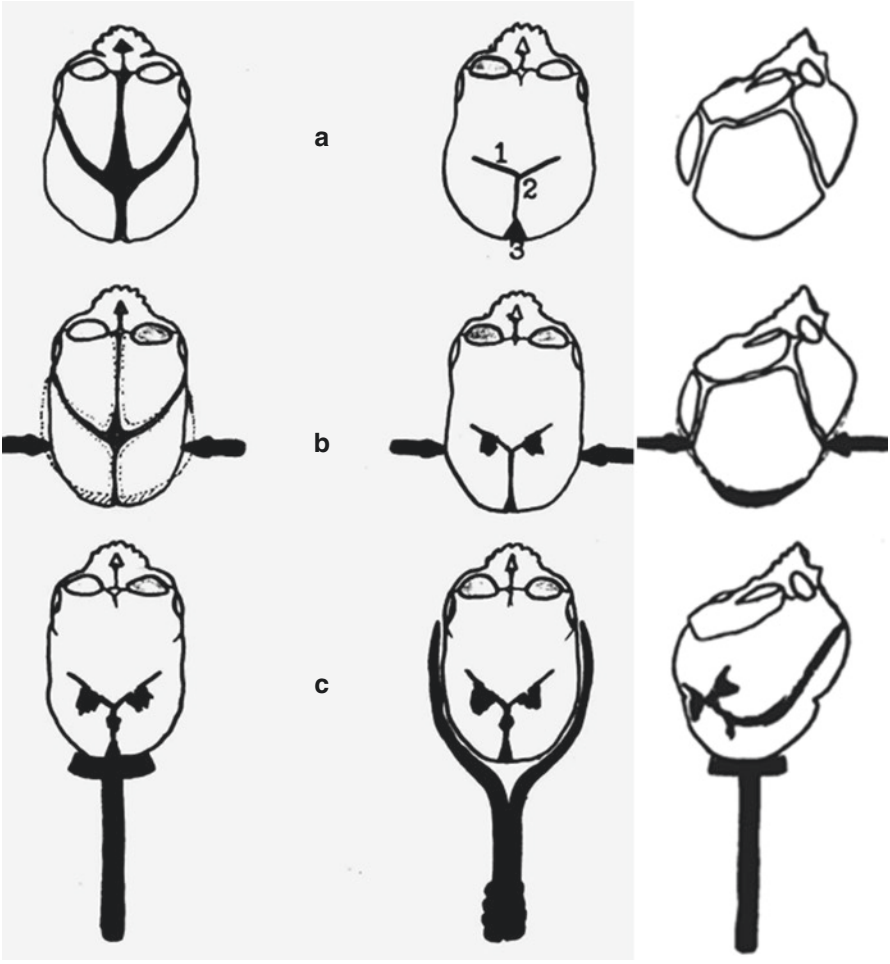

Fig. 7.35 On the left – changes in the skull, cerebral falx (2), and cerebellar tentorium (1) caused by biparietal head compression and delivery interventions. On the right – frontal-occipital head compression (the skull is in the sagittal plane). (**a**) Before fitting of the head; (**b**) with the head compression; (**c**) with vacuum extraction and forceps

The cerebral compression occurs with excessive head configuration and is accompanied by its axial dislocation with characteristic signs of cerebellum tonsil compression in the great foramen. So we can detect sulci of compression around the cerebellum tonsils and sometimes also sulci on the superior surface of cerebellar hemispheres (resulted from the pressure by the edge of the tentorium) or macular subarachnoid and subpial hemorrhages along these sulci. The compression causes ischemia and hypoxia of the brain tissue, but it is clinically manifested by the symptoms characteristic of "fetal asphyxia" (Fig. 7.36).

The cerebral compression is accompanied by narrowing of the superior sagittal and transverse sinuses [34], compression of the superficial cerebral veins, stretching of the bridging veins and the vein of Galen, pressure by the edges of the tentorium onto the superior surface of the cerebellar hemispheres, elongation of the brain in the direction of the birth axis according to the head configuration, elongation of the brainstem, an excessive bend of the spinal bulb in relation to the cervical cord in combination with the compression of the cerebellum, elongation of the arterial circle of Willis, narrowing of the great foramen, etc. The venous drain in the sinuses and veins gets impaired, intracranial pressure increases, etc. It is proved that the front-rear compression of the skull in labor is accompanied by a decrease in circulation through the superior sagittal sinus (up to its complete stop) and changes in the fetal cardiac function – bradycardia [26]. Blood through the anastomoses is drained into the deep cerebral veins, which may contribute to the occurrence of SEH and IVH. Therefore, the cerebral compression can be accompanied by a broad range of hemorrhagic and ischemic CNS. There is evidence that with the skull compression, the ICP value can exceed the systolic blood pressure, which, naturally, results in ischemia of the brain tissue. A microscopic examination of the dead infants reveals ischemic lesions of both cortical neurons and the brainstem nuclei. These lesions are usually referred to as hypoxic-ischemic encephalopathy. Therefore, we can conclude that compression the skull, the brain and the excessive configuration of the fetal head in childbirth is one of the causes of hypoxic-ischemic encephalopathy.

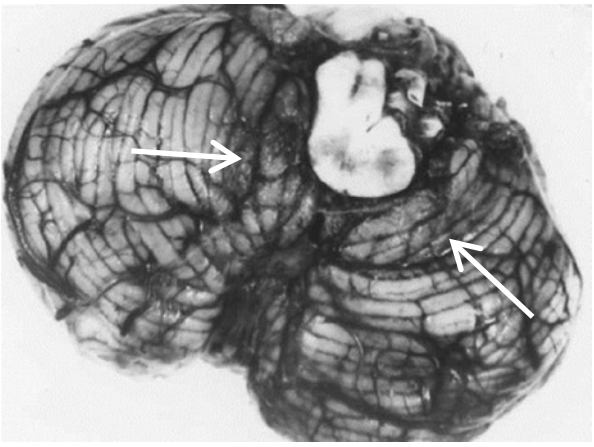

Fig. 7.36 Penetration of the cerebellum tonsils into the great foramen and formation of a compression sulcus (shown with arrows) in a neonate with a mass of 4080 g delivered with cavity forceps, who lived for 9 h, with cerebral compression. The spinal bulb is distorted

I examined six intranatally dead fetuses and two neonates, who lived 17 and 52 h, with signs of cerebral compression. All the fetuses and neonates were mature with birth weights ranging from 3150 to 4000 g; they were delivered with occipital and anterior parietal (one case) presentations and suffered from intrauterine asphyxia. There was poor uterine contraction strength in four cases and an accelerated delivery in one case. Cavity and outlet forceps were used for the delivery in three cases and vacuum extraction in one case. The morphological examination of the afterbirths did not reveal any changes that could explain the cause of intrauterine asphyxia. Massive aspiration of amniotic fluid was detected in the lungs in six cases, and there were signs characteristic of a shock lung, as well as primary atelectasis, in two cases. The signs of a birth injury to the skull (ruptures of the tentorium, cerebral falx, and subdural hemorrhages) were found in three cases and a birth injury to the liver in one case. All the fetuses and neonates had excessive head configurations with significant overlappings of the parietal bones over the frontal and occipital bones. Moreover, in the areas of these overlappings along the coronal and lambdoid sutures, the edges of the subjacent bones going into the cranial cavity formed characteristic sulci of compression or so-called steps on the surface of the hemispheres. They were particularly expressed between the occipital and parietal lobes. The shape of the brain changed accordingly. There were macular leptomeningeal (four cases) and intracerebral (one case) hemorrhages along the sulci of compression. Encephalotomy in the areas of these sulci revealed expressed venous congestion with some punctate hemorrhages. The pallium is pale and clearly delineated from the distinctly plethoric subcortical white matter. Such changes are called a "ribbon effect," which occurs with hypoxic brain damage (Fig. 14.1).

There were also minor subarachnoid hemorrhages in the hemispheres and cerebellar vermis (in three cases). However, subarachnoid hemorrhages in the cerebral hemispheres and cerebellum, as well as a minor cerebellar hemorrhage, could hardly be considered the cause of death.

A microscopic examination in the pia mater and the pallium, especially in the area of compression sulci, detected discirculatory changes in the form of venous plethora, edema, stasis, several hyaline thrombi, and perivascular hemorrhages. There were ischemic and occasionally severe changes in the cortical neurons, hippocampus, optic thalamus, tegmentum of pons, nuclei of medulla, inferior olives, cerebellum (Purkinje cells and dentate nuclei), caudate nuclei, and quadrigeminal tubercles. Karyorrhexis characteristic of post-hypoxic necrosis of the maturing brain was observed in some neurons. Ischemic changes in epy cortical neurons were more expressed in the areas of the brain compression sulci than in other areas.

Surviving infants with these post-hypoxic brain lesions accompanied by necrosis of the nervous tissue have astroglial gliosis of the white matter, microcysts in various regions, dead and calcified neurons, ponto-subicular necrosis, cerebellar infarction, multicystic encephalomalacia, and other lesions that are briefly described in the section on heart attacks and brain necrosis.

Thus, ischemic brain lesions, most often in stem structures, were observed in all the mature fetuses and neonates with signs of cerebral compression due to the excessive head configuration. These lesions can be considered the most significant

for the explanation of intrauterine asphyxia, for aspiration of amniotic fluid, and, consequently, for establishing the cause of death. While in three cases the competing causes of death were subdural hemorrhages (although in two cases they were minor and did not significantly affect the increase in ICP and in the one case it was hemoperitoneum due to the liver rupture), the remaining four cases had ischemic lesions of the brain as the main and only cause of death. Consequently, ischemic lesions of the brain as a result of the head compression and increase in ICP can both be combined with a birth injury to the skull and be an independent lesion.

Thus, not only disturbed uterine-placental and funicular circulation but also the excessive head configuration can cause intrauterine fetal asphyxia. The ICP increases, the venous drain is impaired, the brain matter is compressed, and its ischemia develops. The available data indicate an interlinkage of hypoxic and traumatic factors, as well as the significance of increased ICP in the pathogenesis of cerebrovascular disturbances.

There is evidence that in the case of a birth injury and compression from contact with the almost incompressible tricorn cerebrospinal fluid, there is always trauma and ischemia of the periventricular portion of the white matter [35]. This, in particular, can explain PVL foci and telencephalic astrocytic gliosis in surviving neonates. In one of the observations I consulted, an infant weighing more than 5000 g, who underwent a complicated delivery (the fetus was "squeezed out") with cephalic presentation, did not breathe and had no heartbeat but was reanimated and lived for 18 days. We can assume that in this case, the so-called intrauterine asphyxia was due to cerebral compression, since there were no pathological changes in the afterbirth. In addition to the cortical and subcortical lesions, total astrogliosis of the white matter of great cerebral hemispheres with initial cyst formation (the initial stage of multicystic encephalomalacia) was found in the neonate's brain. There was extensive neuron loss with remaining calcified neurons, signs of their lysis and karyorrhexis, shadow cells, and ischemic lesions observed in the cerebral cortex. Neuronal deposition was observed in the nuclei of the medulla, dentate nuclei of the cerebellum, and other structures.

To establish the mechanisms of traumatic injuries, I compressed the heads of fetuses and neonates postmortem in various planes. There were also "inspection openings" cut in the cranial bones, and parts of the brain hindering observation in the right direction were removed. I found out (Fig. 7.35) that *head compression in the large transverse (biparietal) dimension*, which occurs with a typical application of forceps, caused the *following changes*:

1. Increased frontal-occipital circumference of the head
2. Increased vertical and large transverse dimension of the head
3. Stretching of the cerebral falx and cerebellar tentorium.
4. Bone displacement along the sagittal suture
5. Narrowing of the superior sagittal sinus along the entire length (narrowing is increased with "pseudo-overlapping," i.e., when one of the parietal bones protrudes)
6. Stretching of the bridging veins ending in sinuses and the vein of Galen

Head compression in the frontal-occipital (direct) dimension results in:

1. Increased biparietal dimension of the head
2. Increased vertical dimension
3. Stretching of the cerebral falx and cerebellar tentorium.
4. Displacement of the cranial bones along the lambdoid and coronal sutures
5. Cerebral compression
6. Compression of the superior sagittal sinus at the level of the lambdoid suture by the edge of the squama occipitalis displaced under the parietal bones
7. Stretching of the bridging veins and the vein of Galen

Head compression in oblique diameters, which occurs with atypical application of forceps, causes the following:

1. Increased vertical and the opposite transverse dimensions of the head
2. Stretching of the cerebral falx and cerebellar tentorium, with more stretching of the tentorium part corresponding to the compressed portion of the occipital bone
3. Asymmetric and irregular displacement of the skullcap bones
4. Stretching of the bridging veins and the vein of Galen.

Therefore, the head compression in any of these planes is accompanied by the increased vertical dimension of the head, displacement of the cranial bones, stretching of the cerebral falx and tentorium, and stretching of the bridging veins. The natural delivery suggests simultaneous compression of the head both in transverse and direct dimensions, the degree and nature of which depend on its location in the parturient canal. Consequently, with additional compression of the head, for example, by obstetric forceps, there is usually no possibility of an increase in the frontal-occipital or transverse dimensions, but the large transverse and vertical dimensions may increase. There is also stretching of the may increase and tentorium, and with excessive head compression, the latter ruptures first, typically along the free border.

Thus, with the head compression, the intracranial pressure rises, bradycardia occurs, the volumetric blood flow and oxygen partial pressure in the cortex decrease, the rate of oxidative phosphorylation in mitochondria is suppressed, etc. The result might be a cardiac failure and death. With the skull compression, a *hypoxia of the brain* occurs, which should be differentiated from *fetal asphyxia* caused by disturbed uterine-placental and funicular circulation. The deceleration of the fetal cardiac activity does not always reflect the state of its "asphyxia," although this term is most widely used in modern literature for the overall assessment of its suffering. Bradycardia in the fetus occurs with compression of the head or umbilical cord, as well as with acidosis, arterial hypertension, compression of the lower vein, and for other reasons. In order to accurately assess the condition of the fetus, we have to consider many factors: changes in the cardiotocogram (*it is head compression that causes early deceleration*), pH of the amniotic fluid, ultrasonic data of the fetus and placenta, fetus weight, condition of the maternal passages, position of the head,

nature and degree of displacement of the skullcap bones, and others. Early deceleration is a reaction to short-term cerebral ischemia caused by the compression of the fetal head during the contractions. However, not all late decelerations and various heart rhythm disturbances in the fetus are associated with placental insufficiency or, for example, compression of the umbilical cord.

The accurate assessment of the fetus is important not only to prevent the overdiagnosis of asphyxia in the fetus and neonate by obstetricians, pediatricians, and pathologists but also to address the issue of delivery tactics and delivery interventions. In case of true asphyxia caused by disturbed uterine-placental and funicular circulation, application of obstetric forceps may be preferable. If the so-called asphyxia is caused by an excessive head configuration with cerebral compression (hypoxia of the brain with normal oxygen saturation), then a Cesarean section is recommended. This condition of the fetus is more correctly attributed not to asphyxia but to the birth injury of the skull with cerebral compression.

In general, we can distinguish *two variants of lesions* caused by compression of the skull and brain: (1) those associated with the disturbed venous drain from the brain (compression of sinuses and veins, thrombus formation, the bent vein of Galen, increased venous pressure, etc.) and (2) those caused by the disturbed influx of oxygenated arterial blood (compression of various arteries, spasm, arterial hypotension). The first variant (more frequently observed in premature infants) causes SEH, IVH, LMH, CH, periventricular hemorrhagic infarcts, and hemorrhagic cortical infarcts. The second variant (more frequently observed in mature infants) results in hypoxic cortical and subcortical necrosis with a "ribbon effect," ponto-subicular necrosis, cerebellar infarction, PVL, subcortical leukomalacia, multicystic encephalomalacia, and status marmoratus. With the skull compression in premature infants, the venous drain is primarily disturbed, while in case of mature and large fetuses, the superficial arterial network is also compressed, and the arterial blood flow is blocked. Therefore, cerebral compression is accompanied by various forms of cerebrovascular disturbances, which are discussed below.

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