Fetal and Neonatal Trauma in Dystocic Labor and Delivery

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Vasily Vlasyuk

56.1 Introduction

Dystocia arises from a variety of reasons related to the mother and the fetus. Dystocia causes birth traumatic complications both in the mother (from rectovaginal fistula to rupture of the uterus and death of the mother (Fig. 56.1)) and in the fetus (from various injuries to birth trauma (BT) and death of the fetus). With dystocia, surgical delivery is often performed, which can also be complicated by birth damage. Cephalopelvic disproportion, prolonged or dysfunctional labor, malposition (especially occiput posterior position), malpresentations, excessive uterine contractions, vacuum extraction or forceps delivery, difficult delivery, and other conditions are accompanied by significant physical effects on the fetal head and lead to its pronounced configuration.

Speaking about the BT of the fetus and the newborn, one should distinguish between the concepts: (1) BT as a traumatic (compression) injury that has one or another significance during the child's underlying illness; (2) BT as a disease (nosological form), when a general reaction occurs in the child's body to the resulting traumatic injury; and (3) compression hypoxia due to circulatory disorders and cerebral hypoxia. The action of physical force on the fetal head is mediated by its configuration (molding).

56.2 Configuration (Molding) of the Skull and Birth Trauma

The dystocia, disproportion between the fetal head and the mother's pelvis, arising from incorrect presentation and position of the fetus, a contracted pelvis, and other conditions is directly related to the configuration of the head—its adaptation to the size and shape of the birth canal. There is also the concept of "configurability" [1]—the ability of the skull to undergo configuration. This property depends on the density and elasticity of the bones of the skullcap, gestational age, and other factors. The pressure on the fetal head during labor can be 120–500 mmHg [2, 3]. The pressure from the side of the uterine wall is maximum in the area (zone, belt) of contact of the fetal head in the birth canal and amounts to 200 mmHg or more [4]. At the same time, intracranial pressure rises to increased 30–50 mmHg [5] and sometimes higher, which can lead to birth injuries and impaired cerebral circulation. The skull plays a protective role against brain damage. The configuration also protects the mother's birth canal from traumatic injuries. However, the skull should not only protect the brain but also should not impede the progress of the head along the birth canal of the mother, what happens thanks to the configuration process.

Configuration is an evolutionarily formed process that ensures the adaptation of the size and shape of the head to the birth canal of the mother and also prevents birth injury to the mother and fetus. The configuration promotes the advancement of the head in the birth canal. During configuration, the fronto-occipital and bitemporeal sizes of the head are usually reduced, and the vertical size of the head usually increases. A decrease in the fronto-occipital diameter of the head occurs when the bones enter along the lambdoid and coronal sutures (can decrease to 1 cm [6]); a decrease in the transverse diameter of the head occurs when the bones enter along the sagittal suture. The suboccipito-bregmatic diameter is shortened and the occipito-mental diameter lengthens [7–9]. When studying the configuration, coefficients and indices are extracted from the data on skull measurements [10, 11]. After the birth of the baby, the head is restored and reconfigured. If the compression force of the head exceeds the compensatory-adaptive capabilities of the head, then this leads to birth trauma. Excessive configuration is accompanied by ruptures, hemorrhages, and other cerebrovascular disorders. So, one should distinguish between (1) physiological and (2) pathological configurations [12]. The pathological configuration is accompanied by birth mechanical

V. Vlasyuk (✉)
Department of Forensic Medicine, S. M. Kirov Military Medical Academy, St Petersburg, Russia

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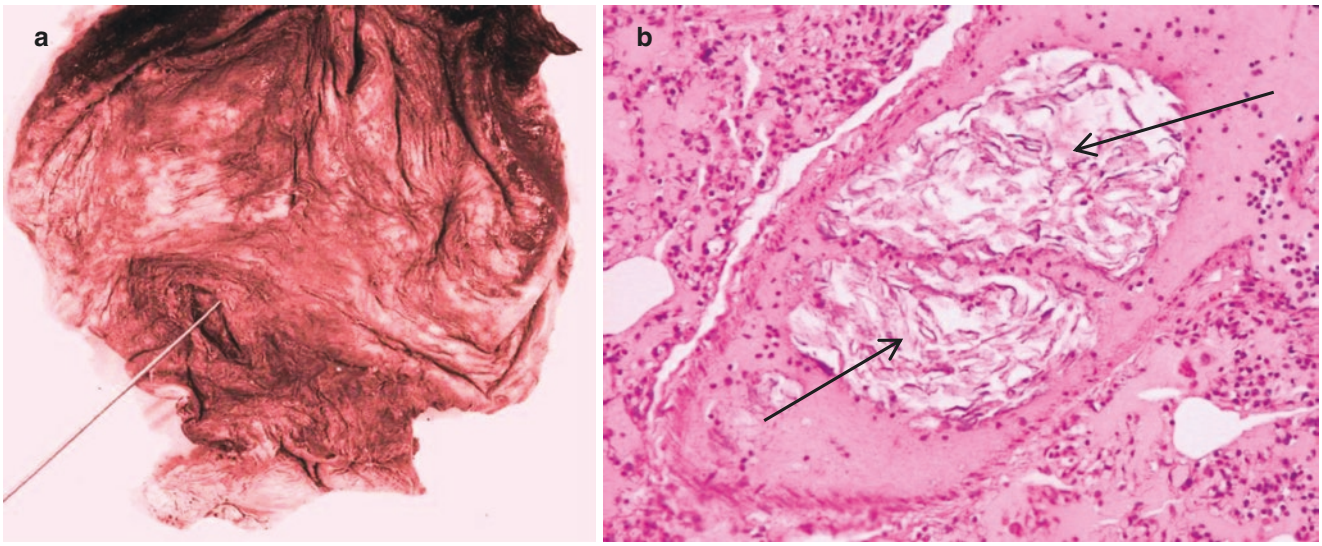

Fig. 56.1 Maternal death of 28 years in childbirth due to uterine rupture and amniotic fluid embolism (primary uterine inertia, stimulation with oxytocin): (**a**) uterine rupture in the lower segment; (**b**) amniotic fluid (horny scales) in the lumen of the artery of the lungs (arrows). Hematoxylin and eosin (H&E) stain, ×110

damage, and the physiological configuration prevents birth damage.

The leading configuration mechanism is the displacement of bones relative to each other, the overlapping of bones. Bone overlapping can be *partial* (overlapping along the part of the suture), *complete* (throughout the suture), and *criss-crossed* (e.g., in one part of the lambdoid suture, the parietal bone comes under the occipital bone, and in the other part of the lambdoid suture, the parietal bone goes over the occipital bone). In addition, *pseudo-overlapping* should be distinguished when one of the bones protrudes with respect to the opposite one by 0.3 cm or more. The nature of bone overlapping depends on the presentation and position of the fetus, as well as on gestational age. For example, with brow presentation, the frontal bone goes over the parietal bones, and not vice versa. The degree of bone overlapping depends on the strength of the uterine contractions, on the size of the intrauterine pressure, and on the acting forces on the fetal head.

Three degrees of configuration can be distinguished [12]—(1) light, (2) moderate, and (3) expressed: 1 degree, bones overlapping along one of the sutures; 2 degrees, bones overlapping within 2–3 sutures; and 3 degrees, bones overlapping at 4–5 sutures.

Clinical and experimental studies have shown that head compression leads to deceleration of cardiac activity [2, 13, 14]. Other studies have shown that deceleration of cardiac activity is associated with compression of the vessels of the umbilical cord, inferior vena cava, and even the iliac vein [15]. Summing up these data, it can be concluded that various circulatory disorders significant for the fetus lead to deceleration of cardiac activity. Indeed, with severe head compression, large veins and sinuses are compressed, cerebral blood flow is reduced, and blood outflow through the jugular vein and blood flow through the superior vena cava are disrupted.

Clinical studies have shown that with the second and third degrees of the configuration of the head, early deceleration is observed: with the second degree, the appearance of early decelerations, and with the third degree, the appearance of early and sporadic decelerations [16]. Light and moderate configurations do not lead to pathological changes, but expressed (pathological) degree leads to compression hypoxia of the fetal brain [17] and birth trauma.

There is another way to highlight the degrees of configuration of the head [18, 19]: one degree (+1), the edges of the skull bones move in the area of the joints, bones touching; two degrees (+2), bones overlapping but easily separated with digital pressure; and grade 3 (+3), bones overlapping and not separable with digital pressure. The absence of bone displacements is considered as a zero stage. These data are recommended to be entered in the partogram. Unfortunately, this method of determining the degree of configuration does not take into account how many seams there is a bone overlapping.

The **pathological configuration** is divided (Fig. 56.2) into (1) excessive, (2) rapid, and (3) asymmetrical [12].

With an *excessive configuration*, the tensile strength of various structures of the skull and brain becomes such that various tears and hemorrhages occur (with severe contractions and violent labor, during labor stimulation, contracted pelvis, application of obstetric forceps, etc.). The tension force of the falx and tentorium cerebelli (TC) begins to exceed their strength, which leads to rupture of structures. First of all, the TC is tearing, as it is less durable than falx.

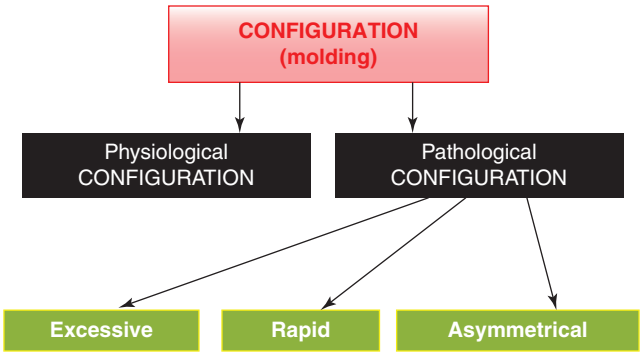

Fig. 56.2 Types of configurations

Before ruptures, intradural hemorrhages can occur. A rupture of the TC leads to subdural hemorrhage and often to the death of the child. With strong pressure on the parietal bones, the cerebral hemispheres are significantly displaced; bridging veins are stretched. First, the inflows of the bridging veins rupture, which leads to subarachnoid and subpial hemorrhages, and then the veins themselves rupture, which leads to subdural hemorrhages. With significant bone overlapping, there is a strong pressure on the brain primarily in the areas of overlapping (sutures), the formation of impressions in it in the form of steps, rupture of blood vessels, and subarachnoid and intracerebral hemorrhages. With an excessive configuration, the superior sagittal sinus is compressed; the outflow of blood from the superficial veins of the head is disrupted. Other changes characteristic of BT also occur.

A *rapid configuration* occurs during rapid parturition and precipitated labor, oxytocin stimulation, the use of obstetric forceps or a vacuum extractor, with pelvic presentation, and with the desire to quickly complete the birth in the interests of the mother or fetus. In these cases, various compensatory-adaptive mechanisms do not have time to develop, the blood circulation in various structures does not have time to rebuild, the fetus does not have time to change the position of the head, etc. Structures can withstand gradually developing tension, but break when the same force acts for a short period of time.

An *asymmetrical configuration* often occurs when the head is inserted asynclitically. Small asynclitism (physiological) is characteristic of normal childbirth and is associated with small lateral movements (declination) of the head in the birth canal (deviation sagittal suture and 1.5 cm from the central axis of the birth canal). With a contracted pelvis, with a cephalopelvic disproportion, with stimulation of childbirth, etc., declination of the head becomes pathological (pathological asynclitism). In this case, the tension force of the two halves of the TC becomes different and focuses on one half, which leads to a rupture. If the head were inserted synclitically, then with the same degree of configuration, the tension force would be distributed evenly over the two halves of the TC and no rupture would occur. It is with the asymmetric configuration of the head that many cases of birth injury are associated, since *ruptures of the TC are most often unilateral*.

It is extremely important in childbirth to determine the nature of the configuration of the skull, which can affect obstetric tactics and prevent BT. Neurosonography can help control the nature and degree of configuration of the fetal head.

56.3 Adaptations in the Head to Protect the Brain During Configuration

Displacements of the skull bones during head configuration do not exhaust all existing adaptive mechanisms. Nature created ingenious adaptations in the head to protect the brain during configuration aimed at protecting the fetal brain from damage during the configuration of the skull. Indeed, with the configuration, the shape of the skull changes; the TC and falx are tensed; the brain is subjected to pressure, moves in the cranial cavity, and can change shape; vessels and sinuses are compressed, etc. Only the fetal brain can survive without damage in a process such as childbirth, which is accompanied by compression of the head of significant strength.

Adaptations in the head to protect the brain during configuration are as follows:

1. The presence of a subdural fluid (effusion) in the subdural space (Fig. 56.3a, 56.4a), which provides equalization of pressure on the surface of the brain and glides of the cerebral hemispheres along the dura mater during their movement; the amount of fluid increases with meningitis (Fig. 56.3b) and with atrophic changes in the brain.
2. The absence of arachnoid villi and cerebrospinal fluid outflow into the sinuses, characteristic of older children and adults, which would prevent gliding between the arachnoid and dura mater.
3. The presence of long bridging veins, which ensure the outflow of blood from the surface of the brain to the sinuses during movements of the cerebral hemispheres (Fig. 56.4b).
4. The presence of a criss-crossed type of bone entry that prevents excessive displacement of the bones of the skullcap [12, 20].
5. Plasticity (flexibility) of the brain, providing significant changes in its shape (due to weak myelination, high water content, immaturity, etc.).
6. The presence of long anastomosing veins, which ensure the outflow of venous blood from the cortex and superficial cerebral veins into the system of internal cerebral veins (with narrowing of the superior sagittal sinus, with pressure of the bones of the skull on the hemispheres of the brain).

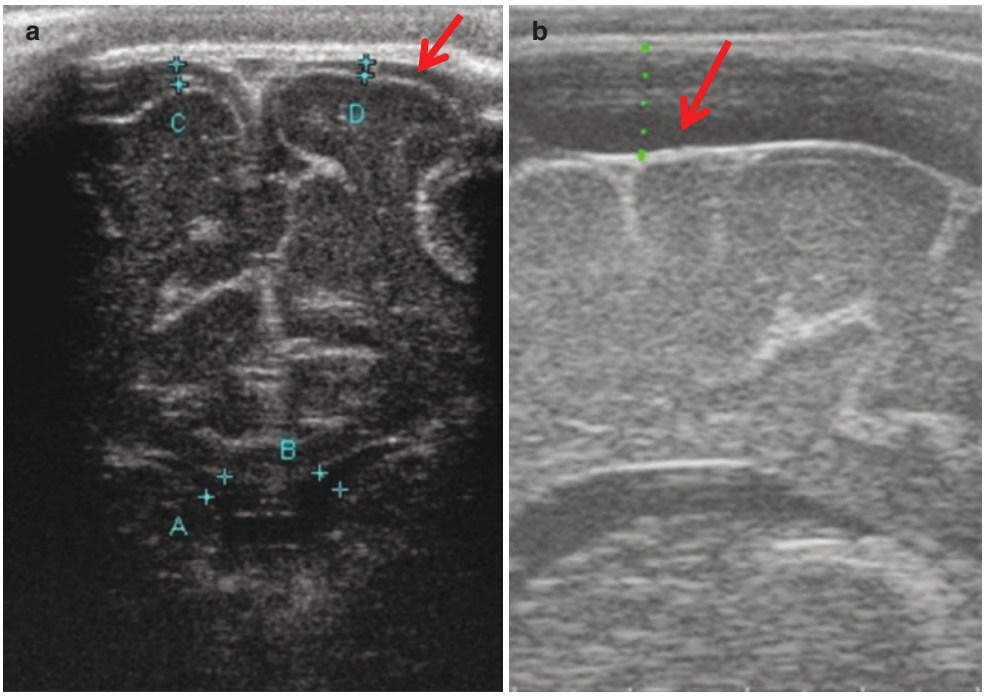

Fig. 56.3 Subdural space (arrows): (**a**) in the newborn is normal, frontal scanning; (**b**) with purulent meningitis in the child for 5 months. Ultrasound diagnostic

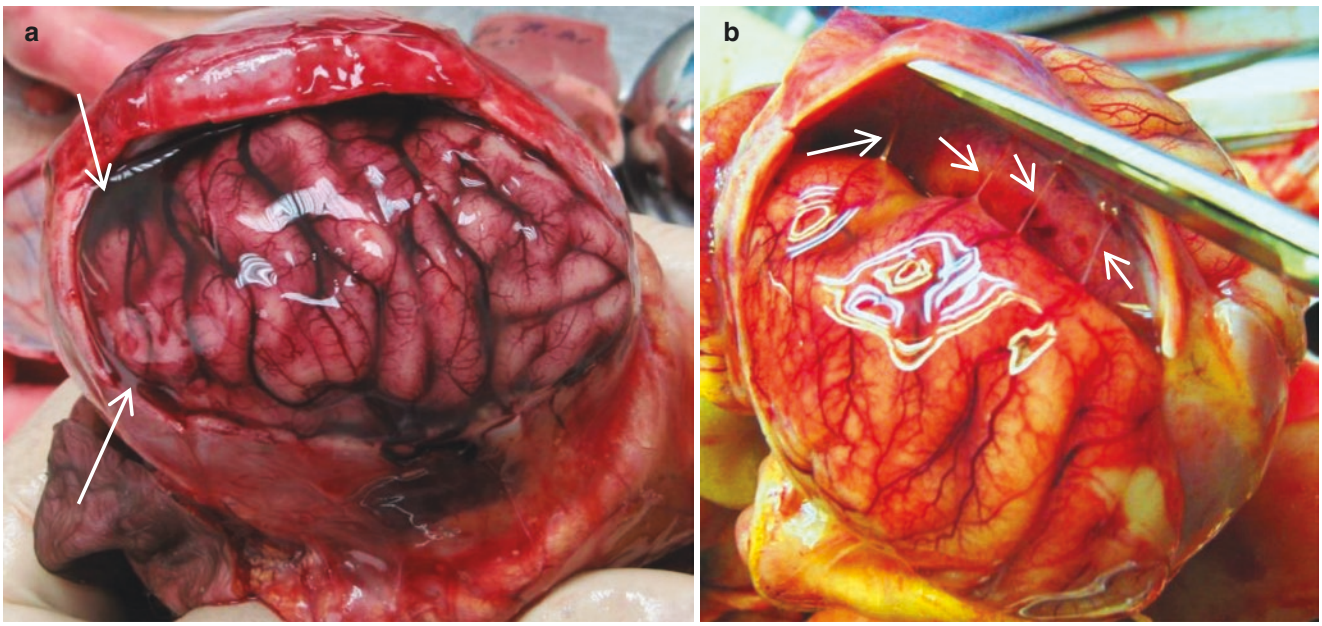

Fig. 56.4 Adaptations to protect the brain from damage in newborns: (**a**) effusion (fluid) in the subdural space (arrow) above the arachnoid membrane; (**b**) long bridging veins flowing into the superior sagittal sinus (arrows)

7. The movement of blood from the external cerebral veins and periosteum veins to the epidural cerebral veins and then to the sinuses.
8. The presence of well-developed epidural venous plexuses of the spinal cord, ensuring the outflow of blood from the brain into the lower and upper vena cava; an increase in venous outflow from the brain through the vascular plexuses of the spinal canal.

With the configuration, the falx and TC are stretched, preventing the excessive configuration of the skull, increasing the vertical size of the head, and compressing the brain with the bones of the skull.

These adaptations prevent the development of cerebrovascular accidents during compression of the head and its configuration. Thanks to these adaptive changes, a child can be born without hypoxia during a prolonged or rapid birth,

with cephalopelvic disproportion, with the use of obstetric forceps, and other complications of childbirth. However, all adaptive changes have their limits, beyond the boundaries of which there are birth traumatic injuries and hypoxia of brain tissue.

56.4 Birth Traumatic Injuries of the Fetus and Newborn and Traumatic Injuries of the Tentorium Cerebelli

Birth traumatic injuries on the part of the fetal head are caput succedaneum; subaponeurotic hemorrhage (Fig. 56.5a), most common during fetal vacuum extraction; region of periosteal congestion (RPC) of the skullcap (Fig. 56.7); cephalohematoma (Fig. 56.5b); fractures of the skull bones (Fig. 56.5a); epidural hemorrhage; subdural hemorrhage (Fig. 56.6a); intradural hemorrhage in the TC (Fig. 56.8a) and falx; ruptures of the TC (Figs. 56.8b, 56.9a, b), falx, and bridging veins; areas of brain compression (Fig. 56.6b); some subarachnoid and intracerebral hemorrhages; etc. described in the literature [12].

Such birth injury as the region of periosteal congestion (RPC), first described in 1985 [21], insufficient attention is paid. RPC is the area of the periosteum of the fetal skullcap, located below the area (zone, belt) of contact of the fetal head in the birth canal. Both in the event of a caput succedaneum and in the occurrence of an RPC, the leading cause is disorder of the outflow of blood from the adjacent region of the head due to compression of the tissues and blood vessels at the level of the area (zone, belt) of contact of the fetal head in the birth canal. Cephalohematoma also occurs in the RPC region due to massive hemorrhages in the periosteum, followed by its detachment. RPC exists more than 10 days after each birth with vertex presentation. The hemorrhages do not quickly resolve. The harder the birth and longer the second period, the more pronounced the RPC. In the vessels of the periosteum, pronounced venous congestion and numerous small focal hemorrhages are determined. Therefore, this region has a dark red color and is clearly delimited from the surrounding periosteum (Fig. 56.7). RPC is the "imprint" of labor, presentation of the head. Studying RPC, it is possible to determine the presentation of the head, the wire point of the head, and the degree of asynclitism [12]. In the study of RPC, it is possible to evaluate the birth, the degree of flexion, and the extension of the fetal head. Ultrasound studies of RPC are not known to me.

Of particular importance in the diagnosis of BT are tentorium cerebelli (TC) injuries, since they are most common, occur under the influence of skull compression, and allow us to prove the compression nature of other brain injuries. Therefore, TC can be called a *mirror of birth trauma.* Beneke [22] first pointed out the leading role of TC ruptures in the occurrence of birth injury to the skull.

Birth traumatic TC injuries (Figs. 56.8, 56.9) are of two types: (1) intradural hemorrhages and (2) ruptures (tears). TC tears usually occur against hemorrhages in the TC. TC is a duplicate of the dura mater, between the leaves of which are arteries and veins that flow into the direct and transverse sinuses. During the compression of the skull, TC is stretched while the veins flowing in it are compressed, the outflow of blood is disrupted, and intradural hemorrhages occur. Then,

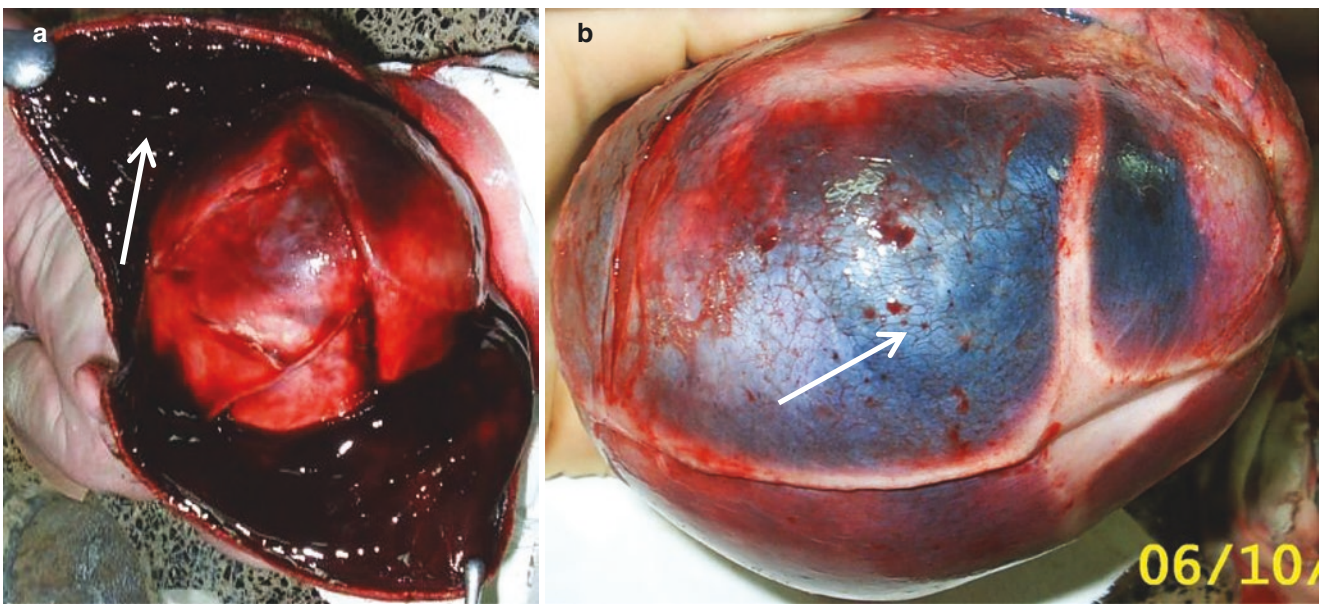

Fig. 56.5 Birth injuries: (**a**) subaponeurotic hemorrhage and fracture of the right parietal bone during vacuum extraction of the fetus; (**b**) cephalohematoma of the left parietal bone in the initial phase

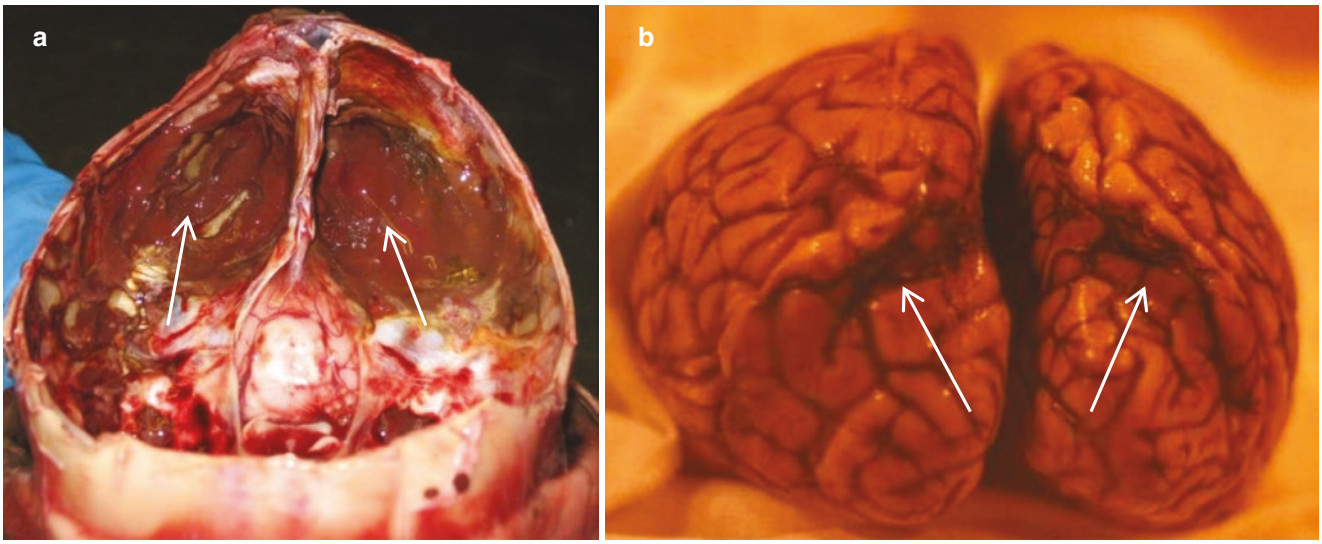

Fig. 56.6 Birth injuries: (**a**) subdural supratentorial hemorrhage; (**b**) groove of compression of the tissue of the occipital lobe of the brain, corresponding to the area of overlapping of bones along the lambdoid suture

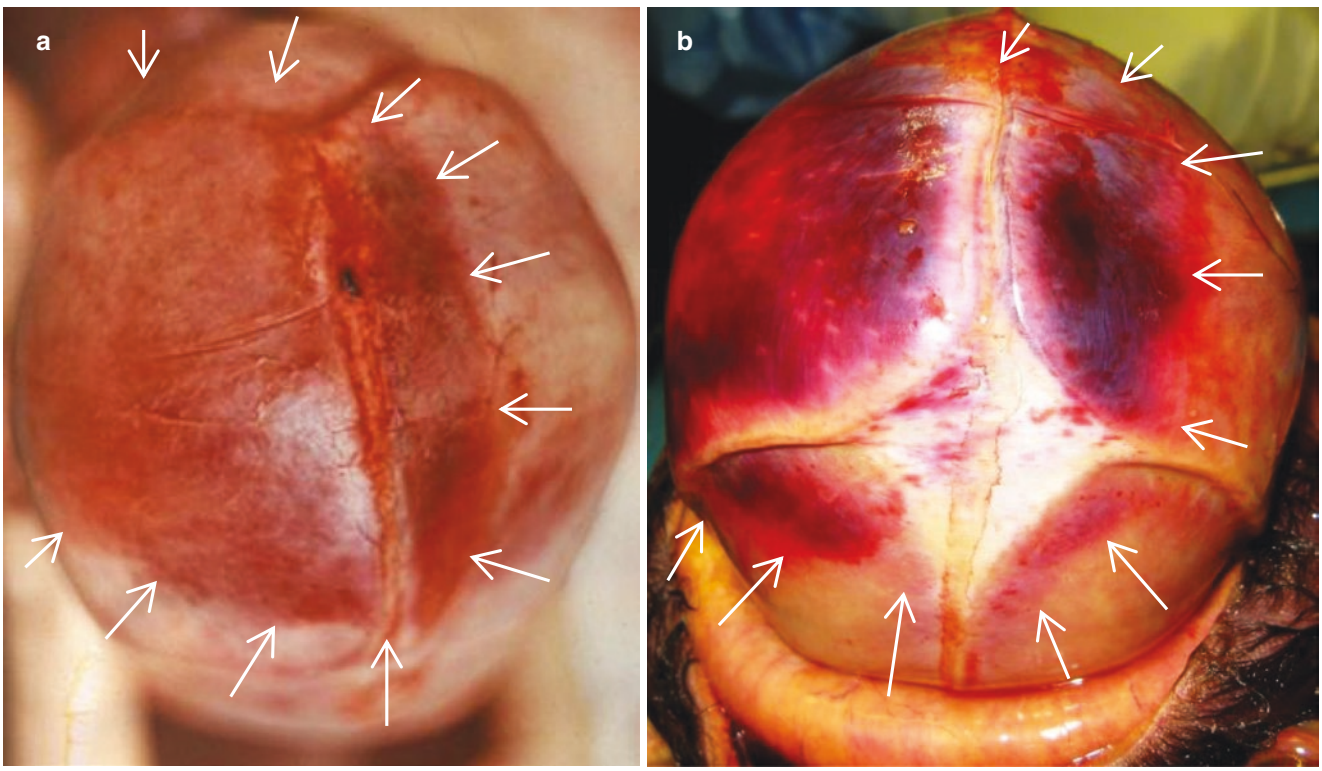

Fig. 56.7 RPC in the area of the skullcap in newborns (edges are marked by arrows): (**a**) RPC is shifted to the right parietal bone, which indicates asynclitism; (**b**) RPC is shifted to the right and to the frontal bone, which indicates extension of the head (anteroposterior insertion, asynclitism)

in the period between contractions of the uterus, blood circulation in the TC is restored. If the pull force of the basting exceeds its strength, then a rupture occurs, which leads to subdural hemorrhage. Sometimes there are small tears of one leaf of a TC (Fig. 56.9a) that do not lead to subdural hemorrhage, or the resulting hemorrhage is insignificant, quickly stops, and has no thanatogenetic significance. If the child dies, the detection of intradural hemorrhage in the TC indicates the presence of birth traumatic injury. This helps to justify the occurrence of other brain injuries by compression of the skull and BT in childbirth. Ultrasound diagnostics of intradural hemorrhages in newborns can also help in the clin-

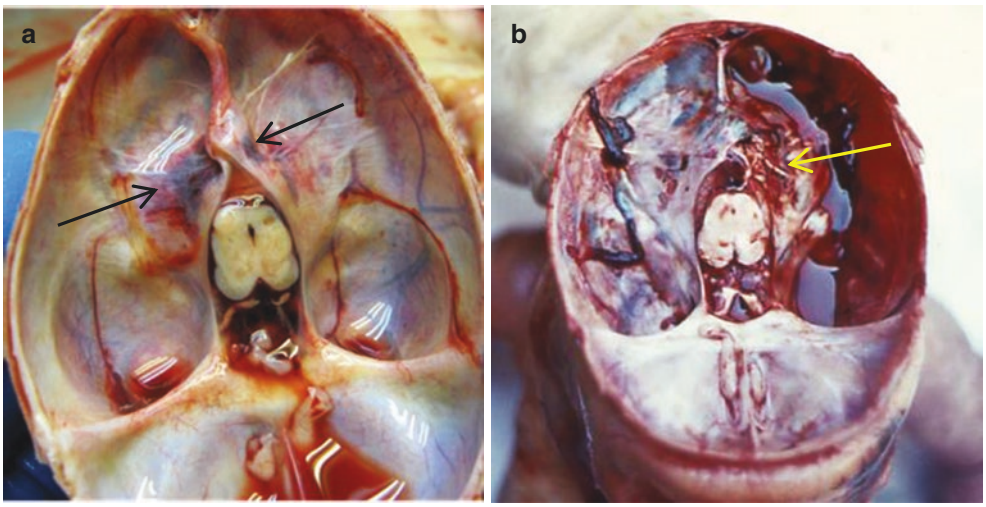

Fig. 56.8 TC injuries: (**a**) intradural hemorrhages in both halves of TC, large on the right (arrow); (**b**) rupture in the left half of the TC (arrow); in the right half, there is a small rupture in the upper leaf of TC

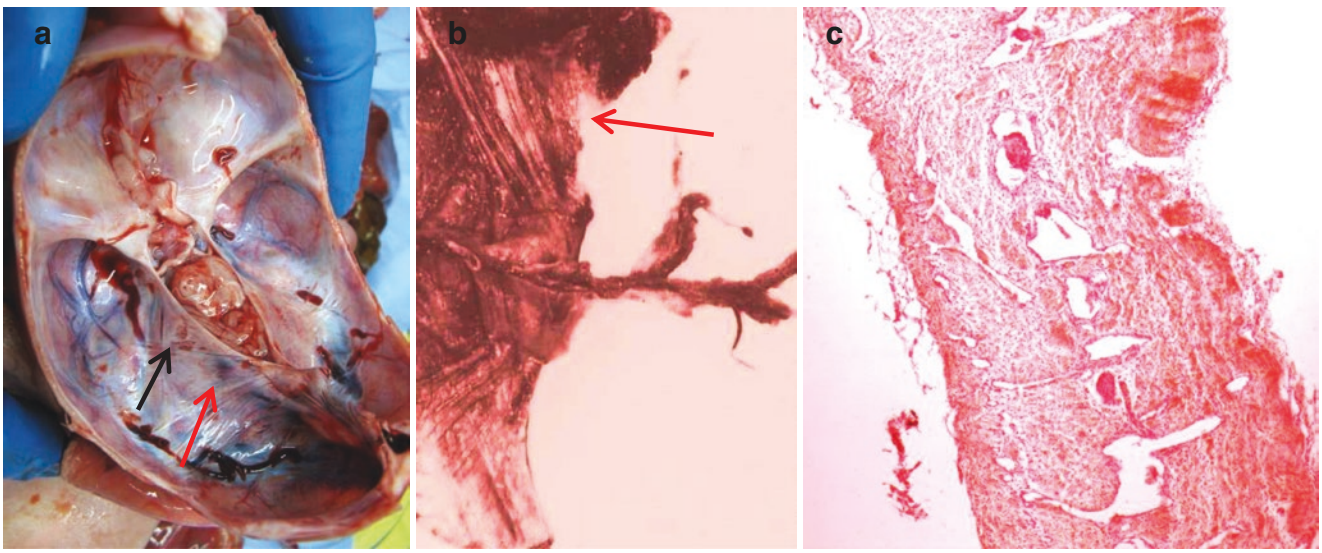

Fig. 56.9 TC in newborns: (**a**) intradural hemorrhage (red arrow) and rupture of the upper leaf (black arrow) in the left half of the TC; (**b**) the edge rupture of the left half of the TC (arrow); ahead, great cerebral vein with tributaries; (**c**) a cross section of the TC; in the center, a large number of vessels without contents. H&E, ×40

ical diagnosis of brain lesions and in substantiating the clinical diagnosis of BT.

When studying the TC tears on sectional material, it was found that with vertex presentation, they are more often unilateral, and with pelvic presentation, they are bilateral [12]. In case of vertex presentation, tears (or tears of a larger size with bilateral lesions) are more often located in the left half of the TC [12, 23, 24]. TC tears are much more common in pelvic presentation compared with head presentation and more often observed in full-term infants compared to premature infants and in children with a larger mass and length [12, 25].

It should be noted that among the most dangerous birth defects for the fetus, the most frequent are TC ruptures, followed by ruptures of the falx and bridging veins, fractures of the skull bones, etc.

56.5 The Importance of Asynclitism in the Occurrence of Birth Traumatic Injuries

Asynclitism is often combined with dystocia during childbirth, accompanied by an increase in the incidence of newborns and an increase in the frequency of cesarean section [26]. A comparison of the locations of RPC and TC tears revealed two patterns [12]. It turned out that (the **first regularity**) with a central location of the RPC, predominantly bilateral uniform discontinuities of the TC arise, and with asymmetric, corresponding to asynclitic insertion of the head, unilateral discontinuities or prevailing on one side (89.3%; $p < 0.01$). A **second regularity** was also found: a one-sided TC rupture or a larger TC rupture is localized on

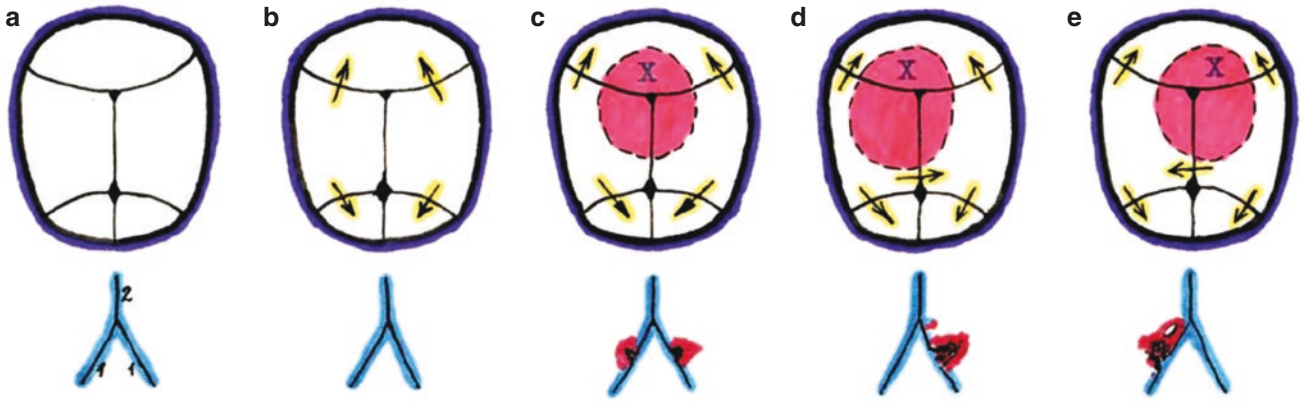

Fig. 56.10 Correlation between the location of the RPC and the localization of injuries (rupture, hemorrhage) of TC: (**a**) skull (above) and the TC (1) with part of the falx (2) before childbirth; (**b**) the beginning of the configuration of the head; the entry of the parietal bones on the occipital and frontal; (**c**) RPC approximately in the center of the parieto-occipital region with synclitic insertion of the head; bilateral uniform tears of TC; (**d**) RPC displaced to the right parietal bone; left-sided tear of TC; (**e**) RPC shifted to the left parietal bone; right-sided tear of TC

the side opposite to the to the RPC movement side (Fig. 56.10). If the RPC is shifted to the right parietal bone, the left half of the TC ruptures, and if the RPC is shifted to the left parietal bone, the right half of the TC ruptures. The same pattern is also confirmed in relation to intradural hemorrhages in TC.

The revealed patterns indicate the role of asynclitic insertion of the head in the occurrence of injuries to the TC. Indeed, the displacement of the RPC to the side of the sagittal suture indicates an asynclitic insertion of the head. The predominance of unilateral TC ruptures indicates a high frequency of asynclitic insertion of the head, which is considered by obstetricians as physiological asynclitism and is not accepted in the obstetric diagnosis. At the same time, this is a fact of obstetrician underestimation of asynclitism in the development of pathological changes in the fetus.

With asynclitic insertion of the head and compression of the skull, the TC tension force propagating from the side of the falx is unevenly distributed on two halves and focuses on the side opposite to the direction of the RPC displacement. On this side, intradural hemorrhages and ruptures occur. At the first position and anterior asynclism, the RPC is shifted to the right parietal bone, and the damage will be located in the left half of the TC. Since the first position and anterior asynclitism are most common, therefore, the most often found left-sided TC ruptures. It can be assumed that with synclitic insertion of the head and the same degree of configuration of the head, tears might not occur, since the tension force of the TC was distributed evenly on both halves and would not reach the degree of their rupture.

Since the shift of the RPC to one of the parietal bones indicates an asynclitic insertion of the head (in all observations, obstetricians mistakenly regarded it as "physiological asynclitism"), it can be concluded that asynclitic insertion of the head leads to unilateral or predominantly unilateral lesions of the TC (hemorrhage, rupture). *Any degree of asynclitism leads to an uneven tension in the TC and increases the risk of damages.*

56.6 Differences Between Birth Trauma and Compression Hypoxia

For a long time among pathologists and clinicians, discussions have been held about the differences between birth trauma (BT) and asphyxia. What injuries in newborns are attributed to BT and which to hypoxia (asphyxia)? The outstanding American pediatric pathologist Potter pointed out the importance of separating hypoxic and traumatic lesions [27], but so far these issues have not been resolved. Currently, the diagnosis of hypoxic-ischemic encephalopathy (HIE) is widespread. At the same time, there are distinguished "craniocerebral compression ischemic encephalopathy" [8, 14], which includes brain damage caused by compression of the skull during childbirth (when the head is configured, as well as when using obstetric forceps, a vacuum extractor, and other reasons). One of the prominent Russian pediatric pathologists Dergachev used the term "traumatic asphyxia" and considered fetal compression as one of the causes of intrauterine hypoxia [28]. The concept of "fetal head compression syndrome" is singled out [29]. It is believed that BT can be mechanical and hypoxic. Gutner [30] considered circulatory hypoxia to be the leading and most frequent hypoxia of the fetus, which included brain damage caused by circulatory disorders associated with compression of the fetal head during childbirth, venous congestion, and increased intracranial pressure. All this indicates the existence of a problem of differential diagnosis of hypoxic and

traumatic lesions. The question arises: Should all injuries caused by compression of the skull (mechanical forces) be attributed to BT? Can compression cause fetal hypoxia?

Unfortunately, it is widely believed that intrauterine hypoxia is caused by "disorders of the uteroplacental and placental-fetal circulation." Therefore, it is in the placenta and umbilical cord often trying to find the leading cause of fetal hypoxia. Although many pathologists note that after a thorough examination of the placenta, the cause of hypoxia often "remains unidentified". This approach is understandable, but not accurate. The placenta and the fetus represent a single mother-fetus system; therefore, under the action of any cause of hypoxia, placental-fetal circulation can be disturbed. However, if you approach from the perspective of searching for the primary (main) cause, then you can distinguish five types of causes and their corresponding types of hypoxia, which are based on individual elements of the mother-fetus system: (1) maternal, (2) uteroplacental, (3) umbilical cord, (4) fetal, and (5) brain's compression [17]. Compression hypoxia is caused by compression of the skull in the absence of the pathology of the placenta and with normal oxygen supply to the fetal blood at the initial stage of cerebral hypoxia [13].

In the literature, there is an understanding that there are injuries to the brain of the fetus and newborn caused by the configuration of the head and compression of the skull in childbirth, leading to venous congestion and hypoxia of the brain. Can this condition be attributed to birth trauma, since it is caused by the action of mechanical forces during childbirth? Is it correct to attribute this condition to hypoxia, since the child does not have considerable mechanical damages of the skull and brain? To answer the questions, it is necessary to identify the differences between BT and hypoxia.

The configuration can be physiological and pathological. The configuration can become pathological when using obstetric forceps and a vacuum extractor. The pathological configuration leads to both BT and compression hypoxia (CH) of the brain.

The causes of CH are rapid parturition, prolonged labor, dystocia, discoordinated labor, incorrect presentation of the head, breech delivery, excessive and asymmetrical configuration of the head, cephalopelvic disproportion, the use of obstetric forceps and vacuum extractor, obstetric benefits, and pressure on the fetus. The main part of the listed reasons is given in the International Classification of Diseases and Causes of Death of the tenth revision under the heading P03 "Fetus and newborn injuries due to other complications of childbirth and delivery."

The results of a study of the causes of death indicate the existence of birth trauma (1); hypoxia (2) due to disorders of the uteroplacental circulation, pathology of the umbilical cord, and diseases of the mother; as well as condition (3), in which children have slight signs of mechanical (compression) injury in childbirth, which are combined with signs of severe hypoxic lesions of brain neurons; this condition can be referred to as "compression hypoxia" [17].

With compression of the fetal head, three successively developing stages can be distinguished: (1) the stage of the physiological configuration of the head, (2) the stage of CH, and (3) the stage of cranial BT. At each stage, functional and morphological changes are different. In the first stage, compensatory-adaptive mechanisms come into effect, in the second stage, changes characteristic of CH occur, and in the third stage, BT occurs. The transition of one stage to another occurs with an increase in the action of physical factors, an increase in the compression of the skull, and an increase in the configuration of the head. However, staging can be disrupted or occur very quickly when the fetal head immediately or quickly begins to experience excessive physical effects, compensatory-adaptive mechanisms and CH do not have time to develop, and ruptures of structures and subdural hemorrhages immediately occur. BT is formed. It should be borne in mind that skull BT can occur against the background of various types of intrapartum hypoxia.

What happens with a pathological configuration of the head? There are tears and disturbances in cerebral circulation. Two phases can be distinguished: (1) without significant pressure of the skull bones on the surface of the brain and (2) with the presence of pressure on the inner surface of the bones covered with the dura mater on the hemisphere of the brain.

First phase. **Moderate head configuration** → stretching of the TC, falx, and bridging veins → impaired blood flow to the sinuses and subdural veins → narrowing of the sinuses → tension of the large cerebral vein → increased venous pressure in the internal cerebral veins and sinuses → impaired venous outflow from the brain → hypoxia and acidosis of the brain tissue brain

Second phase. **Expressed configuration** of the head → pressure of the inner surface of the cranial bones on the arachnoid membrane of the brain → change in the shape of the brain → displacement of the arachnoid along the dura mater → tension of the bridging veins, compression of the superficial cerebral and basal veins → sinus compression → tension of the Galen vein → obstruction of the outflow of blood into the sinuses and along the large vein of the brain → venous congestion in cerebral hemispheres, subcortical nuclei, cerebellum, and brainstem → hypoxia and acidosis brain tissue

A further increase in the degree of configuration and the development of pathological variants lead to various ruptures, compression of the brain tissue by the edges of the bones, and intracranial hemorrhages and, consequently, to the skull BT. First of all, there are ruptures of the TC, and then there are ruptures of the bridging veins and the falx.

Since both BT and CH are caused by the action of mechanical forces, it is necessary to distinguish between these states. In order to clearly distinguish between birth trauma and hypoxia, it is necessary to distinguish such a type of hypoxia as "compression hypoxia" and to clearly know the lesions characteristic of this brain damage.

Mechanical injuries, related to **compression hypoxia** of the skull and brain, are [12]:

1. Intradural hemorrhages in the TC and falx
2. The RPC and spotted epidural hemorrhages
3. Spotted subpial hemorrhages at the apex of the convolutions (Fig. 56.11a), caused by rupture of small vessels and capillaries between the choroid and the molecular layer of the cortex (the mechanism of their occurrence was described in detail by Schmidt [31])
4. Subarachnoid hemorrhages in areas of the cerebral hemispheres where bridging veins connect with the arachnoid membrane due to ruptured inflows of bridging veins (Fig. 56.11b) (one cannot agree that bridging veins rupture in the falx area [32])
5. Small focal intracerebral and leptomeningeal hemorrhages in the areas of pressure on the brain (Fig. 56.6b) of the overlapping bones (mainly in the areas of lambdoid and coronal sutures)
6. Intraventricular hemorrhages due to rupture of blood-filled capillaries of the vascular plexuses of the lateral ventricles of the brain; some subependymal hemorrhages
7. Hemorrhages in the region of the quadrangular lobes of the cerebellum, due to the pressure of the edges of the TC on the cerebellum during dislocation of the brain and some others

In the process of childbirth, under the influence of mechanical forces and compression of the skull, two types of compression lesions (conditions, diseases) can occur: BT and CH. They differ in the nature of the resulting damage to the skull and brain. In BT, pronounced mechanical damage dominates in the form of ruptures of TC, falx, bridging veins, fractures, large focal subarachnoid and intracerebral hemorrhages, subdural hemorrhages, etc. In CH, hypoxic damage to the brain caused by impaired blood circulation, venous congestion, increased intracranial pressure, compression of the blood vessels of the brain, and others dominates.

Thus, two different nosological forms of brain damage in children should be differentiated—BT and CH. There are characteristic morphological changes with these nosologies. Sonography specialists may try to diagnose these changes in living children; pathologists determine the morphological changes in the dead. In order to prevent CH and BT, it is necessary to diagnose the degree of head configuration in childbirth, monitor the course of childbirth, and control so that the physiological configuration does not become pathological.

56.7 Traumatic Injuries During Surgical Delivery

Surgical delivery is carried out strictly according to the indications in the interests of the mother and the fetus. There may be complications with each surgery. However, more often there are favorable outcomes of the operations undertaken, the rescue of children from fetal death and severe BT. It is necessary to know the condition of the fetus and its

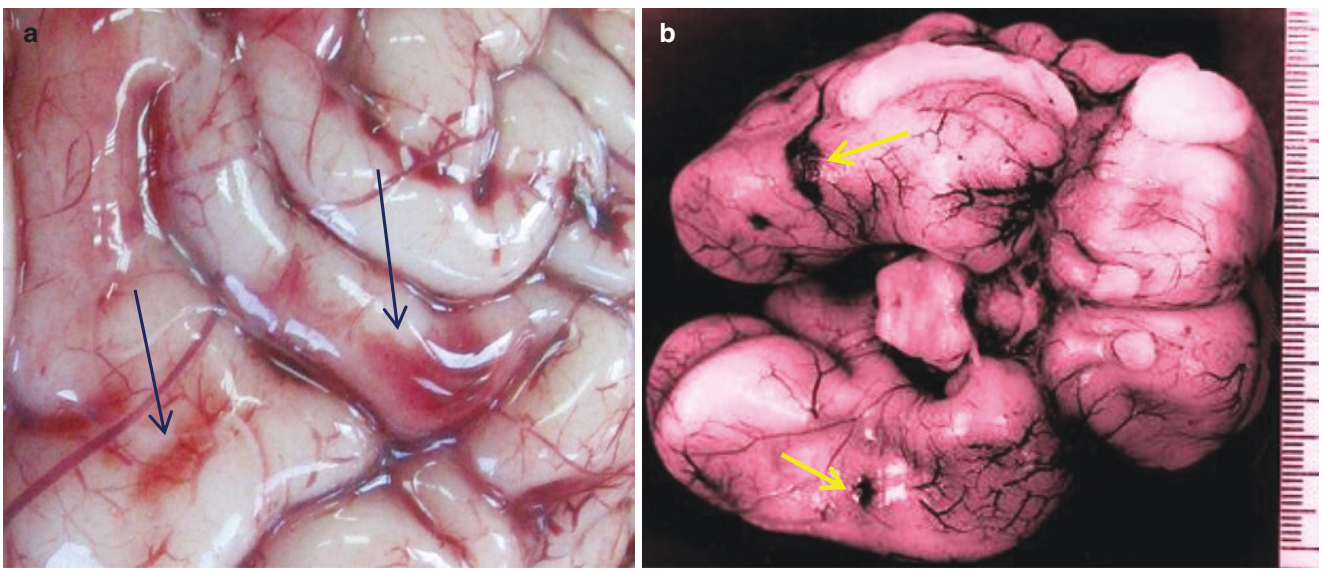

Fig. 56.10 Subpial and subarachnoid hemorrhages with CH: (**a**) spotted subpial hemorrhages on the tops of the convolutions (arrows); (**b**) spotted subarachnoid hemorrhage in the area of the outflow of bridging veins (arrows)

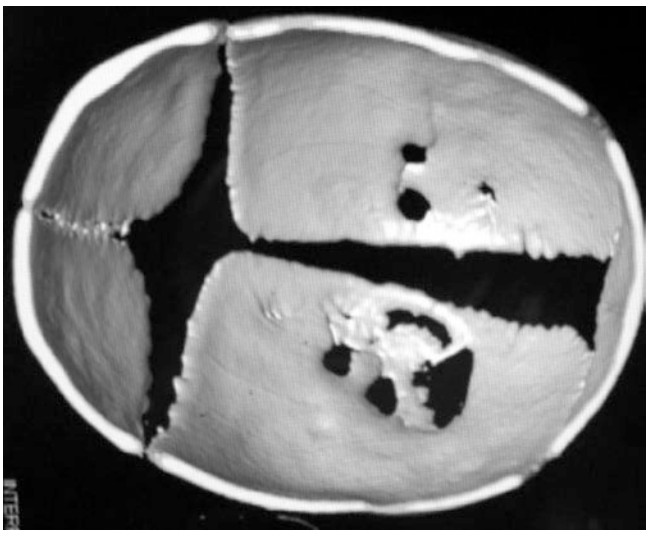

Fig. 56.12 Lacunar skull of a newborn (MRI), extracted using a vacuum extractor, which determines birth defects and a fracture of the right parietal bone. After birth, signs of brain damage and a delay in psychomotor development were revealed. A judicial investigation was conducted into the mother's complaint about the harm to the child's health

head and the condition of the mother and master all methods of operative delivery. To know the methods is to know the indications and contraindications, as well as possible complications.

Given the pathogenesis of TC ruptures, I consider it inappropriate to use a vacuum extractor with an asynclitically inserted head. With asynclitism, the use of obstetric forceps is also inappropriate if the operation is not aimed at correcting asynclitism and saving the mother with the rapid completion of labor. The use of forceps and a vacuum extractor for pathological asynclitism will contribute to the onset of TC rupture and subdural hemorrhage.

The use of a vacuum extractor is contraindicated in cases of skull anomalies. Therefore, it is important to know the condition of the skull before deciding to apply surgery (Fig. 56.12).

56.8 Conclusions

The chapter presents morphological data and theoretical foundations for BT resulting from dystocia, complications of childbirth, and the use of benefits and obstetric operations. It was shown that under the action of mechanical forces, birth injuries, BT, and compression hypoxia of the brain, complicated by compression encephalopathy, occur. The morphological manifestations of birth traumatic injuries are briefly described, and the emphasis is on explaining the occurrence of RPC and tentorium cerebelli lesions. The head adaptations that prevent the development of a BT are described; first of all, this is the configuration of the head. Undoubtedly, sonography is the leading method for the diagnosis of all pathological conditions of the fetal head in childbirth. This method should help the obstetrician evaluate the configuration of the skullcap, as well as help the neonatologist in the diagnosis of existing traumatic injuries in the newborn. Sonography is a diagnostic method that is closest to structure and morphology—fundamental knowledge. Many features of sonography have not yet been realized and require their implementation.

The above materials indicate the great importance of intrapartum ultrasonography for the prevention of a variety of birth injuries, birth trauma, and compression hypoxia, which lead to compression hypoxic-ischemic encephalopathy, disability, cerebral palsy, and fatal outcomes.

The intrapartum ultrasonography improves the fetal head malposition and malrotation diagnosis in comparison with the traditional digital vaginal examination [33]. In fact, in dystocia labor, digital vaginal examination in the presence of caput succedaneum can be difficult, especially in asynclitism, and conversely sonographic diagnosis is easy [34, 35]. The sonographic asynclitism diagnosis is done by transabdominal and translabial sonography [36, 37].

The most common sign for asynclitism diagnosis by transabdominal ultrasonography is the squint sign (or Malvasi sign), or single orbit sign, because the fetal head is twisted [38, 39]. The signs for asynclitism diagnosis by translabial sonography are the midline to pubis in posterior asynclitism and midline to sacrum in anterior asynclitism, and also the sunset thalami and sunset cerebellum signs [40]. Childbirth should not be the art of an obstetrician, which is based on intuition and experience. Childbirth is primarily an obstetric practice based on knowledge. Such knowledge is provided by sonographic (the simplest) and other methods of neuroimaging studies. Knowledge makes it possible to save children from formidable complications in very difficult situations.

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