CURRENT OPINION

Re-evaluation

Compression of the fetal brain

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Knowledge is more than equivalent to force.[33]
Samuel Johnson

COMPRESSION of the fetal head during labor or delivery may result in brain damage which is manifested by perinatal death or epilepsy, mental retardation, or cerebral palsy. In recent years the incidence of such complications has been reduced by the development of techniques to predict dystocia (x-ray pelvimetry) and by the abandonment of certain obstetric maneuvers (version and extraction, high forceps) in favor of cesarean section. Unfortunately, there is still a great deal of room for improving our infant salvage as is evidenced by the too frequent autopsy findings of brain trauma in the newborn.

It seems plausible that any advance in this field is dependent upon a clear assessment of the causes and mechanisms of brain injuries due to compression. This paper will review these aspects of cerebral compression, consider its effects on the fetal cardiovascular system, and survey possible methods for restricting it to permissible limits of safety.

Sources of compression trauma

Compression of the fetal skull may result from two sources: endogenous: the bony pelvis, the myometrium, the cervix, and the perineum; and exogenous: the accoucheur's hand, forceps, or vacuum extractor.

Mechanisms of compression trauma

Compression of the fetal skull may produce brain damage by one of three mechanisms:

1. The increased pressure is transmitted inside the calvarium where it may overcome the intravascular blood pressure resulting in arrest of the cerebral circulation. The ensuing development of anoxia and asphyxia may damage not only the brain cells, but also the blood vessel walls, making them liable to rupture when exposed to hypertension.

2. It will be recalled that the brain is covered by two protective envelopes, the dura (with fibrous tentorium cerebelli and falx cerebri) and the calvaria. The dura represents a framework which fixes the brain to the skull bones and supports the cerebral blood vessels. The anterior margins of the tentorium cerebelli and the inferior portions of the falx cerebri are characterized by thickened bands of connective tissue, "stress bands," which represent protection against stretching.[29] Such stretching occurs whenever the mobile and separated skull bones are distorted due to cerebral compression. The resultant pull and stretch of the tentorium or the falx may cause them to tear. If the laceration extends into the venous sinuses,

bleeding into the confined subdural space occurs and the pressure of the accumulating blood may damage vital centers.

3. Severe compression on the fetal head may cause a fracture of a skull bone with laceration or direct injury to the underlying brain tissue.

Forceps and cerebral compression

There is little doubt that forceps represent the most conspicuous source of cerebral compression. Indeed, during the eighteenth and ninteenth centuries, the use of forceps to decrease the diameters of the fetal skull was an accepted method of management for cephalopelvic disproportion. It was not unusual to compress the fetal skull to such an extent that its transverse diameter was shortened by as much as 3.5 cm. Indeed, Milne Murray in 1887 designed a scale for application to the forceps from which the decrease in head diameter with compression could be determined exactly.[49] Application of such mechanical guides to instrumental delivery were popular during this era. Several obstetricians[15, 18, 34, 58] modified their forceps so that the amount of traction exerted could be directly determined at the instant of each pull; "average pulls" had a span of 30 to 42 pounds and "severe pulls," a range of 80 to 286 pounds. The latter force (286 pounds) seems incredible, although it was obtained by the combined efforts of two men with their feet propped in supports pulling a fetal head through a contracted pelvis.[34]*

The use of forceps to deliver the aftercoming head may require especially strong traction since in breech births the head has not been favorably molded to the pelvic architecture and dimensions by several hours of labor. In addition, the aftercoming head is subjected to sudden compression and is therefore at a mechanical disadvantage as compared to the forecoming head. Other factors such as deflexion and an incompletely dilated cervix also contribute to the necessity for strong forceps traction. The resultant increase in cerebral compression is a major reason for the intracranial trauma associated with breech deliveries.

Pioneering efforts to investigate the relationships of forceps traction and intracranial pressure changes were instituted in 1874 when Fehling made a few preliminary studies of cerebral compression in dogs and dead infants.[22] By measuring the changes in cerebrospinal fluid pressure he showed that there was an elevation in intracranial pressure in response to squeezing the head with forceps. More extensive studies of a similar nature were carried out by Baxter in 1946. He found that the intracranial pressure rose slowly with traction until the pull reached 50 pounds. He observed that a pull of 50 to 70 pounds was associated with a rapid rise in intracranial pressure.[3]

In 1887, Acconci studied this problem by inserting a manometer through a trephine hole in the parietal bone next to the uninjured dura mater of 17 dead infants. He then tested various types of forceps and reported that the least compressive force (as indicated by the smallest increase in intracranial pressure) was achieved with a modified version of the Simpson forceps.[1] In 1907, Seitz investigated the response of intracranial pressure to varying amounts of forceps traction by means of small balloons placed inside the cranium. When forceps were applied he found that the intracranial pressure was 15 to 25 mm. Hg. As traction was exerted the intracranial pressure rose progressively and with a "strong pull" it reached a level of 85 to 95 mm. Hg. Seitz quotes speculations by Helfer that the fetus would exhibit no symptoms while its intracranial pressure was below 75 mm. Hg, since this level was still below that of the fetal blood pressure (75 to 100 mm. Hg).[62] These workers evidently were not aware of the thesis that the fetal blood pressure would rise in response to elevation of intracranial pressure. This principle was stressed in the subsequent studies of Gurewitsch and Vogel.

*It should be mentioned that these studies were prophesied some 20 years before they took place! In the controversial novel of 1847, *Tristam Shandy*, Dr. Slop is accused of using "a pressure of 470 pounds of avoirdupois weight" to effect a forceps delivery.[65]

who used an intracranial manometer, dead newborn infants, and a model of a pelvis. They supported the hypothesis that the stress of forceps compression triggered a reflex increase in the fetal blood pressure to protect against cerebral anoxia and asphyxia.[25]

This concept of a hypertensive response to cerebral compression had been introduced in 1866 by Leyden.[42] It was later re-emphasized by the noted neurosurgeon, Harvey Cushing. In animal experiments, he demonstrated that induction of a rise in the intracranial pressure was immediately followed by an increase in the systemic blood pressure.[17] Cushing's experiments have been confirmed[26] and recent reports by Kety and his associates[40, 63] have stressed the thesis that the hypertensive response to elevations of intracranial pressure represents an effort by the body to maintain cerebral blood flow.

This information is provocative for it provides both an explanation and a rationale for the well-known bradycardial response of the fetus during forceps delivery: Compression of the head causes a rise in the intracranial blood pressure and, secondarily, the systemic blood pressure. The latter, according to Marey's law, would be associated with a deceleration of the heart rate.[7]

Cerebral compression and fetal bradycardia

The association of cerebral compression and bradycardia in the human fetus was first recognized in 1822 when de Kergaradec noted that the fetal heart rate often slowed during a uterine contraction.[36] Subsequent years saw his observations repeatedly confirmed and the emergence of two schools of thought as to the mechanism for this bradycardia. The most popular theory held that the fetal heart was slowed due to compression of the baby's brain against the pelvis and cervix by the contracting uterus. The minority opinion contended that the bradycardia was due to anoxia from myometrial compression of the placenta. This latter view has been weakened by the reports that the percentage saturation and partial pressure of oxygen in the blood of the intervillous space is *not* reduced during a uterine contraction.[52] Additional evidence against the placental compression belief was the demonstration that during a contraction a twin in breech position had no bradycardia, although its sibling, with vertex engaged, had a slowing of the heart rate.[30] Further confirmation is provided by the studies which report that the fetal heart rate of a baby presenting by breech does not slow with uterine contractions.[6, 30]

The first advocate of the theory that bradycardia was due to cerebral compression was the elder Nägele. In 1838, he reported that he could produce bradycardia in the fetus by manually squeezing its head through the abdominal wall of the mother.[50]* Since Nägele's time there have been numerous articles substantiating the association of cerebral compression and slowing of the heart rate of both the fetus[21, 24, 32, 54, 67, 69, 72] and the newborn infant.[8, 24, 52, 68] Indeed, in labors where the head was unengaged, the appearance of fetal bradycardia was often taken as an indication that the head had become engaged; this association was labeled the *Eintritts-effekt* (entrance-effect) by Gauss in 1905 and was still a recognized clinical sign many years later.[19]

It is of interest to review the probable sequence of events which transpire during the activation of this bradycardial reflex by head compression. When the brain is squeezed, certain vagal centers may be activated either through specific volume and pressure receptors or through the accumulation of anoxia and asphyxia due to circulatory arrest. (Although this latter possibility seems weakened by the very rapid myocardial response to cerebral compression [2 to 3 seconds],[38] it has been shown that compression of the skull of a fetal guinea pig is followed immediately by a lowered avail-

*Nägele astutely suggested this maneuver as a means for diagnosing the presenting fetal pole as breech or vertex. Nearly a century later, Kautsky reported the same observation and his name has subsequently been attached to this technique "Kautsky's phenomenon."[35] Since his name was already preserved in eponyms (his perforator, his rule for calculation of the estimated date of confinement, and his obliquely contracted pelvis), perhaps Nägele would not have considered this oversight an injustice.

ability of metabolically active oxygen in the brain tissue.)[46] These changes may stimulate the cardioinhibitory vagal center directly, or indirectly through specific vasomotor centers. When the latter are stimulated they cause peripheral vasoconstriction. The resultant rise in blood pressure affects receptors in the aortic arch which then send impulses by afferent vagal fibers to the cardioinhibitory center in the floor of the fourth ventricle. It exerts its bradycardial response by sending impulses through the vagus nerve to ganglion cells in the myocardium of the auricles. From this site the signal passes along postganglionic fibers to the sinoauricular and auricular-ventricular nodes where it evokes the release of acetylcholine. This substance changes the membrane permeability of the myocardial cells, resulting in alterations of the ionic concentrations of sodium and potassium. As a result, diastole is lengthened and the heart rate slows.

The amount of brain compression required to produce bradycardia has been studied. In animals, the fetal heart slows with cerebral compression.[26, 27, 46, 59] Although some workers have not detected such bradycardia in fetal lambs,[56] others have noted that it appears when pressure on the head of the lamb exceeds 50 mm. Hg.[70]

Rech in 1933 investigated the reaction of the human fetal heart to various degrees of skull compression. He observed that a "gentle squeeze" of the head usually caused a fall of 20 beats per minute whereas a "hard squeeze" elicited a fall of 40 beats per minute. Rech quotes the earlier work of Sauerbruch, who found that a pressure of 50 to 60 mm. Hg on the fetal head caused bradycardia, whereas elevation of this pressure to 80 to 100 mm. Hg produced tachycardia.[54] In a recent study, Chung and Hon, using pessaries of various sizes, noted that bradycardia usually results when the skull compression reaches an intensity of 30 to 40 mm. Hg.[14]

The above results are interesting when they are correlated with studies of fetal heart rate and uterine contractions. Caldeyro-Barcia reported that fetal bradycardia usually develops when the amniotic fluid pressure reaches an intensity of 40 mm. Hg.[12] At this pressure the fetal head would be supporting a weight of 11 pounds (assuming the head to be spherical and having a diameter of 10 cm. and using the formula: area × weight of 40 mm. Hg). From such evidence, it may be surmised that 11 pounds represents the threshold value above which a compressive force on the fetal head will generally induce bradycardia. In accord with this suggestion is the fact that a forceps pull of as little as 10 pounds is commonly followed by a slowing of the baby's pulse below 100.[38] Since most labor contractions exceed an amniotic pressure of 40 mm. Hg[12, 43] and nearly all forceps pulls exceed 10 pounds,[45, 71] one would predict that fetal bradycardia should be a common occurrence during these events. Indeed, this is a familiar clinical observation and is not considered an indication of fetal distress. As a matter of speculation, slowing of the heart rate may well be a physiologic change, reflecting the cardiovascular effort to maintain normal perfusion of the brain during the period of compression. It has been suggested that the lowered heart rate will allow increased diastolic filling and alteration of blood flow through the foramen ovale and ductus arteriosus. This would permit a much larger fraction of umbilical venous or caval return into the left ventricle and subsequent augmentation of cerebral blood flow.[2] If this thesis is valid, efforts to abolish such a response by the administration of atropine[9, 28, 31] may not be in the best interests of fetal well-being.

"Safe" brain compression

In the effort to reduce cerebral trauma from compression, the detection of signs heralding imminent danger to the fetal brain would be invaluable. Unfortunately, if such signs occur, their existence has not yet been discovered, probably because techniques for monitoring parameters other than the fetal heart rate have not yet been developed. Although cardiotachometers are gaining in popularity, the information they provide may

not be helpful. The appearance of fetal bradycardia per se does not provide a valid indication of dangerous cerebral compression since, as mentioned above, even a mild sequeeze of the baby's head will cause the heart rate to slow. It is possible that either the degree of the bradycardia or the tardy resumption of a normal rate between compressions may indicate trouble; studies of these and other changes in comparison with the baby's ultimate outcome are presently in progress.

The absence of clinical signs which could warn of impending cerebral trauma emphasizes the need to consider measures which might prevent or reduce excessive cerebral compression. The renaissance of the vacuum extractor can be attributed in great part to its apparent safety in regard to cerebral compression. Rosa has mathematically calculated that with equal amounts of traction, the cerebral compression with this device is one-twentieth that created by forceps. Moreover, the instrument requires less traction force for delivery than would be necessary with forceps.[45] In addition, the principle inherent in the vacuum system provides an insurance against excessive traction. Under the standard conditions for its use (negative pressure of 0.7 Kg. per square centimeter), the cup will detach from the fetal scalp when the traction force exceeds 35 pounds.[11, 20, 38] These apparent advantages of the vacuum extractor may be offset by the unavoidable scalp trauma which is associated with the use of this instrument.

The aspect of forceps delivery which merits our attention is the measurement of the amount of force to which the fetal head is subjected. Obstetricians-in-training who ask the question "how hard should I pull," rarely receive an objective answer. Advice ranges from "pull only with one hand, though as hard as is necessary" to "pull until the delivery room table starts to move." Opinions vary widely even among experienced obstetricians as to what constitutes a "difficult forceps delivery" and it is a valid observation that judgment on how hard to pull is often acquired at the expense of traumatic deliveries. Birth is a mechanical process in which force overcomes resistance and scientific measurements of the forces involved would seem imperative if optimum fetal results are to be expected. Although it has been possible for a long time to modify forceps so that obstetricians would know exactly how much force is being exerted on the fetal head,[34, 71] these methods have not gained wide application.

Nevertheless, it is likely that with modern electronic refinements, some type of "metric forceps"[23, 39] will be standard procedure in the future. The question of what amount of pull is safe and what amount is hazardous deserves serious consideration. The answer, though unknown as yet, may be approached through a combination of experimental investigations and past experience. One theoretical approach might be considered teleologic in nature: it may be that the maximum amount of safe forceps pull is an amount equal to that maximal force of thrust which is exerted on the head of a term baby during spontaneous delivery in a primigravid woman. According to the studies of Lindgren,[43] the maximal "normal" amniotic pressure during the second stage of labor approximates 120 mm. Hg, a force of 33 pounds. These estimates are similar to those quoted by Reid[55] which range from 25 to 33 pounds of force on the fetal vertex. The teleologic point of view therefore would restrict the forceps pull to a maximum of 33 pounds. It is of interest that this figure is very close to that force which is used in routine low forceps deliveries—namely, 27 to 34 pounds.[45, 71]

However, experience has shown that teleologic reasoning is not always consistent with accepted obstetric practice. One may speculate that countless instrument deliveries have resulted in birth of "normal" infants, despite pulls in excess of 33 pounds. This speculation is based on findings that approximately 20 per cent of forceps deliveries exceed 35 pounds of maximum traction;[38] this practice could never have gained such long-term acceptance if it had been associated with any perceptible increase in cerebral damage.

Thus, any proposed maximal limit of safe forceps traction should be greater than 33 pounds.

From the retrospective point of view, it would seem wise to limit the traction force on the fetal skull to that incurred in "atraumatic" forceps deliveries which have resulted in presumably "normal" infants. Numerous reports[15, 16, 34, 41, 58, 61, 64, 66, 71] and our own experience[37, 38] suggest that a traction force of 50 pounds might be considered the upper range of pull with regard to fetal (and maternal) safety.* It is germane to inquire how much greater compression is exerted by a pull of 50 pounds than would occur during a spontaneous delivery. The latter is associated with a maximal cranial pressure of approximately 1,000 Gm. per square centimeter.[43, 44] In contrast, the compression from a 50 pound traction alone is variously reported as 3,180,[57] 3,600,[16] and 4,500[39] Gm. per square centimeter. Evidently there is a sizable margin of safety in regard to permissible cerebral compression. The thesis that this margin of safety may be surpassed with a forceps pull greater than 50 pounds has been fortified by observations at our own hospital in the past year. Of 20 deliveries with forceps traction ranging from 55 to 80 pounds, 9 of the infants had complications such as skin bruises or lacerations, Apgar scores below 5, transient facial nerve palsies, and conjunctival hemorrhages. Extended follow-up of these infants may uncover further complications.

Even though 50 pounds seems to be a reasonable upper limit of forceps traction, it is associated with certain qualifications. One concerns the phrase "normal infants": its validity is tenuous since the diagnosis is based on a high Apgar rating, normal findings at physical examination, and an uneventful course. Other qualifications to the 50 pound limit are the following: it implies a correct forceps selection, a perfect application, an optimal coefficient of friction and a traction executed in the axis of least resistance. The fulfillment of all these criteria demands experience, skill, and good fortune. It is optimistic to anticipate that these three entities are present in every birth by forceps. Moreover the compressive effects of obstetric instruments will be influenced by such variable factors as the pliability of the membranous bones of the skull,[4] the amount of molding already present,[48] the precompressive state of the cerebral tissues in regard to oxygenation,[40] and the general metabolic and physiologic state of the fetus and its maternal host.[10] These unpredictable aspects provide serious obstacles to any dogmatic conclusions as to permissible limits of fetal safety. They also emphasize our need for basic knowledge of the miracle of birth and the importance of prophylaxis against excessive compression in obstetrics.

One other method of reducing cerebral compression deserves brief mention, namely, the time-honored obstetric technique of pulling in conjunction with a uterine contraction, and a bearing-down effort or fundal pressure. There is little doubt that such adjunctive measures can supplement and reduce the necessary extractive force. For example, uterine contractions may provide 11 to 22 pounds (40 to 80 mm. Hg amniotic pressure[13, 43]), a Valsalva maneuver can contribute up to 30 pounds,[47] and fundal pressure can exert a force of 15 to 20 pounds.[43] It perhaps is unnecessary to recall that cerebral compression can also be reduced by lowering the perineal resistance with an early and adequate episiotomy and conduction anesthesia.

In conclusion, although the evolution of medicine has been remarkable in the past 100 years, little progress has been achieved in avoidance of excessive cerebral compression. To this end we suggest that forceps be monitored for traction and that there be an upper limit of 50 pounds of pull. We present such measures in an effort to introduce a

*Even so, it is a fact that many "normal" babies have been delivered by pulls in excess of 50 pounds. At our own institution approximately 5 per cent of all forceps deliveries exceed this figure.[38] Scott and Gadd report that one of every four of their deliveries by Kjelland forceps exceeded 50 pounds of pull.[61] Baxter (who is of the opinion that 70 pounds is the maximum for safety) has delivered several babies with forces between 90 and 100 pounds; his follow-up of these children has not uncovered unusual mental or physical abnormalities.[5]

modicum of scientific objectivity into a sphere replete with subjectivity. Countless lives have been saved by the skillful use of forceps; countless lives have been lost or harmed by excessive cerebral compression and its undesirable sequelae. Birth injury to the central nervous system of newborn infants is one of the most frequent and deleterious of diseases, and perhaps the most neglected condition in human pathology.[60] Let us recall the advice offered by two experienced obstetricians, one hundred years apart:

In 1844, Dr. John Ramsbotham said: "Cautiously and tenderly must this iron instrument be used! We must recollect that no sensation can be imparted to the operator's hand of any injury that may be done to the woman; and we must remember that one injudicious thrust, one forcible attempt at introduction, one violent effort in extraction may bruise, may lacerate, may destroy! Bear in mind the principle on which it ought to be employed, recollecting that the metallic blades have no feeling, and cannot communicate to our perceptions a knowledge of any mischief we may inflict."[53]

And, in 1944, Dr. John Parks wrote:

"Only at the time of birth may one's life destiny be directed between two unyielding blades of steel. When thus used, forceps become the first form of child guidance."[51]

Summary

1. The causes, mechanisms, and dangers of cerebral compression have been reviewed.

2. The association of pressure on the fetal head and the reflex bradycardia it induces at a threshold of 11 pounds have been discussed.

3. Measures to regulate the amount of cerebral compression exerted by forceps have been suggested.

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