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Mechanism of Neonatal Brachial Plexus Injuries

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Abstract

This article is based on a literary review pertaining to the etiology and pathological mechanism of brachial plexus damage sustained by neonates at birth. The study attempts to identify characteristic environmental, chronologic, epidemiologic and clinical features of arrest of the shoulders at birth in order to establish their compatibilities with two competing concepts about the cause and mechanism of Erb's and Klumpke's palsies in newborn infants:

A) Excessive traction exerted by the professional who assists the mother during the birthing process.

B) Spontaneous injury 'in utero' caused by myometrial activity during gestation and/or labor.

Of the pertinent literary data the following ones appear to be of importance within the context of this analysis:

1. Since fluid fills the amniotic cavity during pregnancy, according to Pascal's law of fluids no part of the fetal body can be subjected to excessive pressure as long as the membranes are intact.

2. Uterine contractions spread from the fundus towards the cervix gradually, thus creating an expulsive force. The musculature generates no traction force; the mechanism responsible for evulsion and rupture injuries that manifest clinically as Erb's and Klumpke's palsies.

3. Because almost one-third of all births occur by cesarean section in contemporary practice, brachial plexus palsy should be frequent following abdominal deliveries if the spontaneous in utero damage hypothesis was correct. However, Erb's and Klumpke's palsies after cesarean section are extremely rare.

4. Forceps and ventouse deliveries increase the risk of brachial plexus damage up to 10-fold. This could not be the case if a high proportion of these injuries were unrelated to vaginal birth.

5. Shoulder dystocia and its associated injuries are 5 to 15-times more frequent in the USA than in England, Italy, Ireland, Israel or Hongkong (China). Because the reproductive process is largely identical among all races, a difference of such magnitude is unlikely to be caused by a factor which is intrinsic to human pregnancies.

6. During the last 50 years the rate of arrest of the shoulders and the typical injuries associated with it increased 5–10-fold in America. Such a sudden change is unlikely to be attributable to some inherited predisposition in a stable population.

7. Before 1975 non-interference with the birthing process was standard requirement in the USA. Then the policy changed and physicians were advised to extract the child promptly after the delivery of the head. Increase of rate of shoulder dystocia shortly after the introduction of the new policy suggests a cause-effect relationship between the two events.

8. Non-interference with the birthing process has been the recommended routine in Great Britain since the early 20th Century. The rates of shoulder dystocia in the USA and Great Britain were equally low before the 1970's. Its occurrence has remained stable in the Britain but increased steeply in this country after obstetricians had changed the method of delivery from conservative to active.

The above summarized observations have practical implications in obstetrics. These are discussed with emphasis upon the fact that some of the new developments in perinatology, perceived as part of the progress initially, have proved counterproductive in the long run.

Keywords: Shoulder dystocia; Erb's palsy; Two-step delivery; Brachial plexus damage

Introduction

For more than a century Erb's and Klumpke's palsies have been attributed to forceful traction applied by the accoucheur during delivery. A new theory was presented two decades ago. Its proponents suggested that one-half or more of these afflictions result from spontaneous myometrial activity in the course of gestation or during labor [1-3]. It has also been opined that brachial plexus palsies that occur in utero spontaneously are unpreventable [4]. It needs mention that the long established explanation of the pathogenesis of these injuries is still favored by many obstetricians and most neurologists [5-7]. The dispute about the causation is not purely academic. If brachial plexus damage generally is secondary to traction, many injuries can be prevented either by resisting the temptation of extracting the body by force or by elective cesarean section when the fetus appears unduly large. Obviously, this controversy has important clinical implications, thus physicians can ill afford to make an error. Traditionally, neonatal brachial plexus injuries have been associated with arrest of the shoulders of the fetus at the outlet of the birth canal after the delivery of the head; a phenomenon generally referred to as "shoulder dystocia". A variety of factors predispose for this dangerous complication of the birthing process (Table 1). In terms of importance they vary on a broad range but three of them, namely maternal diabetes, large fetal size and use of extraction instruments for delivery are widely recognized as conducive to arrest of the shoulders. Since in any one case usually several of the listed factors appear in the background the relative importance of the remaining ones often remains obscure. As an example, oxytocin often is used for induction or stimulation of labor when the mother suffers from diabetes or preeclampsia; conditions that predispose for shoulder dystocia themselves. When administered by an inexperienced

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Preconceptional	Prenatal	Intrapartum
Family history of diabetes	Borderline glucose tolerance	Induction of labor
Personal history of diabetes	Gestational diabetes	Protracted 1st stage of labor
Small maternal stature	LGA* fetus	Prolonged 2nd stage of labor
Maternal obesity	Excessive (>35 lbs) weight gain	Stimulation of labor with oxytocin
Previous LGA* newborn	Preeclampsia	Conduction anesthesia
Primigravida >35 years old	Postdatism or postmaturity	Delivery by forceps
Previous shoulder dystocia		Delivery by vacuum extractor
Inadequate maternal pelvis		Manual extraction of fetus

*Large for gestational age.

Table 1: Risk factors for shoulder dystocia.

practitioner, it may be given in a high dose to overcome uterine dysfunction that had been caused by the drug itself. The inclusion of manual extraction of the fetus in this listing is an unusual feature of this article which is unlikely to be found elsewhere. However, for reasons to be explained later the author believes that not only does the currently fashionable procedure deserves to be listed as a risk factor but equals in magnitude the three prominent ones previously mentioned.

While the earlier presented definition of shoulder dystocia appears to reflect a clear consensus about this diagnosis, in actual fact it is a poorly defined clinical entity. The unresolved question is how soon the shoulders of the fetus are supposed to follow the emergence of the head from the birth canal. Opinions in this regard range from 5-10 seconds to 6-8 minutes. This divergence of opinions means that at this time it may need some search to find two physicians who are in full agreement about the time when the diagnosis of shoulder dystocia becomes applicable. Thus, whereas there is no reason to question the validity of statistics indicating that 0.5 to 2 out of 1000 neonates suffer lasting brachial plexus injury at birth, it is less certain that about 1 out of 7 cases of shoulder dystocia results in such damage [6].

Apart from the fact that they are incapacitating, brachial plexus birth injuries are often associated with brain damage. Clinical problems of this kind rarely offer themselves to evaluation by prospective, controlled, double blind studies. On this account, the validity of theories concerning their causation needs to be tested on the basis of their compatibilities with facts that surround, characterize or derive from the pathological entities involved. This being the case, the subject of the current review is the question of whether the 'in utero' damage hypothesis of Klumpke's and Erb's palsies is consistent with the clinical features of this dangerous obstetrical complication.

Physiology of Labor

Although every fiber of the myometrium is a potential pacemaker [8], normally uterine activity is generated at the region of the utero-tubal junctions. During pregnancy these areas incite intermittent and infrequent contractions that do not involve the entire organ and do not cause therefore significant cervical dilation. At the onset of labor one of these pacemakers becomes dominant. It begins to trigger regular and frequent contractions that spread slowly from the fundus towards the cervix and involve eventually the entire uterine musculature. Prominent investigators emphasized that as long as the membranes remain intact, in keeping with Pascal's law of fluids, the pressure generated by contractions is distributed evenly inside the amniotic membranes [9,10]. They cannot impose therefore more pressure upon the shoulders than on any other part of the body between the top of the head and the tip of the little toes of the fetus. Localized pressure injury can only occur when the membranes rupture.

From the above outlined considerations it follows that if Erb's palsies occur 'in utero', many of them can be prevented by avoiding a frequently used and almost as frequently abused intervention, namely artificial rupture of the membranes. Consequently, the belief that such injuries are categorically unpreventable is obviously incorrect.

The adverse effects upon the fetus of loss of amniotic fluid have been described in detail by Caldeyro-Barcia, a man widely considered the father of modern perinatology [11]. Because spontaneous rupture of the membranes usually occurs during the 2nd stage of labor [12], rarely would the uterus have sufficient time to impose damaging force upon the fetus if the membranes were left intact during labor. Apart from protecting the membranes, other preventive measures could also be explored, such as amnio-infusion or pharmacological expansion of the amniotic fluid volume with administration of prolactin in case of low Amniotic Fluid Index (AFI). Early delivery by induction of labor or elective cesarean section could also be effective. Naturally, these measures would only merit consideration if objective evidence supported the hypothesis of spontaneous brachial plexus palsy 'in utero'.

The force generated by the uterus is expulsive in nature because the stimuli that induce contractions originate at the fundus and then progress slowly towards the cervix. This pressure being distributed upon the surface of the fetal body evenly creates a squeezing effect which brings about its ejection from the womb eventually. However, it does not generate traction force conducive to disruption or evulsion of the nerves in the brachial plexus. Yet, evidence of the latter is found virtually invariably by magnetic resonance examination and during explorative surgery in cases of brachial plexus damage.

Arrest of the shoulders is rare among preterm neonates for two reasons: A) The small size of the fetus. B) The relatively large diameter of the fetal head as compared to the shoulder girdle and the abdominal circumference. At and after term the growth of the shoulders outstrips that of the head, thus the passage of the latter does not guarantee that the available space is sufficient for the shoulders. It is customary to set specific borderlines for vaginal versus abdominal deliveries based on fetal size for didactic purposes. It must be remembered however, that the validity of these recommendations are modified in any particular case by some of the factors listed in Table 1. Besides, official guidelines are simply the common denominators of past disputes and starting points of new ones. They do not always carry therefore the unanimous approval of experts.

The American College of Obstetricians and Gynecologists (ACOG) has circulated among its members a video tape which depicted a birth where no traction was used, yet there was evidence of impaired brachial plexus function in the neonate. However, the loss of function was only temporary in this case. Because the care of affected neonates is promptly taken over by other specialists, the obstetrical literature seldom tries to differentiate between various kinds of degrees of birth

injuries. Thus, "neuropraxia", caused by hemorrhage and edema of the nerve sheet in the absence of injury to the axons may not be readily differentiated from brachial plexus "palsy" which is associated with disruption of the axons. Similar misinterpretation may derive in cases of nerve injury caused by bony structures, a phenomenon referred to as "pseudoparalysis" [6].

Brachial Plexus Injury during Cesarean Section

If many Erb's and Klumpke's palsies develop spontaneously 'in utero', they should be seen often after abdominal deliveries. Since the rate of cesarean sections increased from about 5% in the 1950's to over 30% by first decade of the 21th Century, associated brachial plexus injuries should be found frequently nowadays. Clinical experience shows that this is not the case. After excluding breech deliveries from their material, Ubach et al. [13] found that every one of 102 babies who needed surgery on account of brachial plexus palsy had been delivered by the vaginal route. In the material utilized by the author's group out of about 300 cases of brachial plexus damage only one was associated with abdominal delivery [14,15]. Based on the operative report, this injury also must have been caused by forceful traction since extensive adhesions left behind after a previous cesarean section made removal of the fetus from uterus extremely difficult [16].

Frequent indication for surgical delivery is protracted labor. This complication often entails hypertonic uterine activity caused by obstruction or by the oxytocin stimulation administered to overcome it. If the 'in utero' injury hypothesis of Erb's palsy is valid, prolonged arrest of labor - which seldom occurs in contemporary practice prior to spontaneous or artificial rupture of the membranes- should frequently cause brachial plexus injury. Yet, hypertonic uterine activity is not conducive to Erb's palsy provided delivery is conducted by the abdominal route.

Very large fetuses that are prone to brachial plexus damage often are delivered by cesarean section either electively or following arrest of the labor. Experience shows that abdominal delivery virtually eliminates the risk of lasting plexus injury [17,18].

Brachial plexus injury following cesarean section is a literary rarity [16]. This fact permits the conclusion that Erb's palsy unrelated to vaginal birth is exceptionally uncommon and has little clinical significance.

Instrumental Deliveries and Brachial Plexus Injuries

Two observant investigators noted that the incidence of arrest of the shoulders at birth increases markedly in connection with mid-forceps extractions [19]. Based on reviews of malpractice claims, Brimacombe et al. found that more than one-third of all brachial plexus injuries that led to litigation had been preceded by forceps or vacuum extractions [20]. In their material the risk of fetal injury was ten-fold higher following instrumental than after unassisted deliveries. Fetal damage was more likely to be associated with mid-pelvic than with low and outlet extractions.

If a major proportion of the investigated fetuses in this group had suffered injury 'in utero', the method of delivery would have had little effect upon the rate of Erb's palsies. The contrary evidence indicates therefore that the damage virtually invariably occurred during and was related causatively to the birthing process.

When considering the mechanism of damage in connection with instrumental deliveries, it has to be remembered that traction is routinely applied in synchrony with contractions. On this account, delivery of the head usually occurs at the end of the corresponding contraction. Current recommendations encourage physicians to extract the fetal body promptly [21]. Therefore babies delivered by forceps or ventouse are almost invariably subjected to manual traction also without the support of uterine force. This and other facts earlier discussed are consistent with the interpretation, that the typical causative force of brachial plexus palsies is manual traction applied during the extraction of the body [22].

Geographic Variations in the Incidence of Shoulder Dystocia

The frequency of brachial plexus palsies varies on a broad range in various environments. One would not expect such inconsistency if arrest of the shoulders was simply an undesirable but natural aspect of the reproductive process. Major differences would be unlikely to occur between populations with similar living standards and comparable ethnic compositions. The fact of the matter is however, that whereas in the USA the incidence of arrest of the shoulders usually ranges between 1% and 3%, rates as low as 0.2% have been reported from Great Britain, Ireland, Hong Kong, Israel and Italy; the latter in connection with a research project where the "2-step delivery" method was tested [23-29]. It is unlikely that a dangerous complication could be 5 to 15-times more frequent in one population than in others in the absence of some factor unrelated to the natural course of labor and delivery.

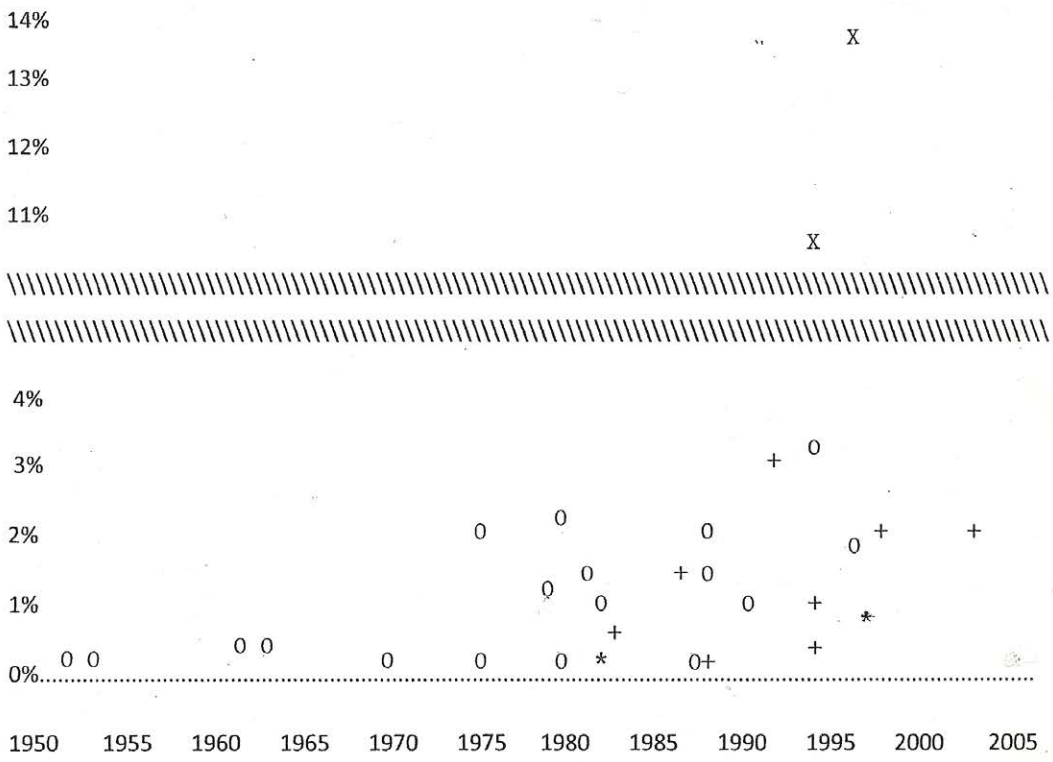
It is thought provoking that Hong Kong was under British influence until recently. The equally favorable statistics in these two geographic areas inhabited by different ethnic populations and separated by thousands of miles suggests the role of a common denominator which dates back to the years when these two groups of people were still connected in terms of culture, economy and education.

Chronologic Fluctuations in the Rate of Shoulder Dystocia

In a recent publication, advocates of the 'in utero' sustained Erb's palsy theory stated that the rate of shoulder dystocia had been constant during the preceding decades [4]. For their statistics they liberally borrowed data from foreign countries where the rate of arrest of the shoulders had been exceptionally low. Even this generous loan from abroad failed to obscure, however the disappointing results reported by most American centers.

As Figure 1 indicates, a computer search conducted by this writer provided vastly different picture of the incidence of shoulder dystocia in this country than what the quoted article had attempted to paint. The illustration indicates that the rate of shoulder dystocia was only at the range of 0.2% - 0.4% between the 1950's and the early 1970's. However, at that time the rate of this complication began to escalate. The increase was continuous and exponential during the last four decades. In one particular service, where strict research protocol demanded prompt extraction of the child after the expulsion of the head, an epidemic increase of arrest of the shoulders occurred [30,31]. In contrast, no unfavorable change was experienced in those services that in keeping with the advice of American textbooks printed before 1975 and European ones published recently adhered to the two-step delivery policy [12,32,33-41].

In the British Islands delivery without the use of traction was the required routine throughout the last century. Thus, the rate of shoulder dystocia remained low there up to these days [25,42,43]. The same is still true for Hong Kong more than a decade after its separation from the British Commonwealth [26].



0 = Percentage of arrest of the shoulders per 100 births.
+ = Percentages were calculated in relation to 100 vaginal births.
* = Deliveries were routinely managed by the "2-step delivery" method (30), without use of traction.
X = Head-to-body delivery times were minimized by prompt use of traction after expulsion of the head.

The illustration is based upon 30 published reports. The authors' names are listed in the reference list under numbers 2, 9, 24, and 31 to 51. The numbers do not match because some articles contained multiple reports. The periods of observation varied between 1 and 12 years in various studies. The illustration refers to the time when about 50% of the relevant birthing events had already occurred. Note low incidence of arrest of the shoulders before the mid-1970's and rapid increase thereafter. Further note continued low rates of shoulder dystocia in services that favored the "2-step delivery" method (*) and exponential increase in one service where attempt was made to reduce the head-to-body delivery interval to a minimum (X).

Figure 1: Incidence of Shoulder Dystocia in the United States between 1950 and 2005.

Effects of Practice Patterns upon Shoulder Dystocia

During the years covered by this review the practice of traditional midwifery became transformed into modern perinatology. Emanuel Friedman's research enabled physicians to avoid or correct protracted and for the fetus stressful labor patterns [44]. Electronic fetal monitoring and tokography allowed doctors to understand and control uterine activity patterns and the fetal condition during pregnancy and labor. Conduction anesthesia administered sporadically by doctors in the 1960's came under the control of expert anesthesiologists. The use of ultrasound turned evaluations of fetal growth, well-being, anatomy and gender into standard procedures.

Impressive progress in neonatal care made it possible for obstetricians to address clinical problems aggressively. In this process cesarean section rates reached 30% at the turn of the century. By that time elective forceps operations were largely abandoned and indications for difficult ones were restricted. In this process, vacuum extractor replaced forceps in the practices of most specialists. Arrested and protracted labors were concluded by cesarean section increasingly often. Many unduly large fetuses were identified before term and delivered by the abdominal route. Difficult instrumental extractions became abandoned. As a result, perinatal mortality rates gradually declined until the late 1990's and then leveled out. Most changes were conducive to elimination or reduction of factors known to be predisposing to shoulder dystocia. Therefore, it is a paradoxical aspect of modern obstetrics that shoulder dystocia and the fetal injuries associated with it reached epidemic proportions after most of their predisposing factors had been either eliminated or markedly reduced.

As a byproduct of the above described developments, at the turn of the 21th century almost one-half of all malpractice actions initiated against obstetricians related to brachial plexus palsies. This development created a professional atmosphere which induced many competent specialists to restrict their practices to gynecology or choose early retirement.

Traditionally, innovations in obstetrics have been introduced with considerable publicity. However, one seemingly minor change entered clinical practice unannounced. During most of the past century American textbooks advised physicians to allow babies to be born spontaneously in the absence of indication for intervention [12,34-38]. Then the picture abruptly changed. After 1975 textbooks of obstetrics instructed doctors to extract the fetus from the birth canal following the expulsion of the head without delay [45-54]. None of them mentioned that preceding editions had recommended the very opposite, nor did they clarify why the proposed intervention was necessary. In new books and new editions of old ones these statements were repeated almost verbatim without explanation or reference to supporting literature.

Clarification of the reason behind the change of directives concerning the method of delivery required extensive review of the medical literature of the 1970's. The result of this search was surprising. Utilizing the at that time novel fetal scalp blood pH determination technique, Wood at al. reported in 1973 that in a group of neonates who had been born without complication the capillary blood pH fell at a rate of 0.04 to 0.14 units / minute after the delivery of the head [55,56]. They noted however, that the births had been uncomplicated, the conditions of the babies excellent and their Apgar scores high.

The cited technique was new, difficult to perform and yet inaccurate. Thus, these articles generated little attention in Great Britain where they were published. On the other hand they induced editors of American textbooks to recommend extraction of the fetus from the birth canal at the first opportunity.

It was against this background that between 1975 and 2005 the rate of arrest of the shoulder increased 5 to 15-fold in the United States [2,9,12,15,24,30-32,57-73]. In contrast, its incidence remained low in the British Islands where physicians continued delivering babies without intervention [25,42,43].

Quarter of a century later the research project of Wood at al. was repeated in well-equipped laboratories by investigators who had long experience with the technique. It transpired that the neonatal scalp blood pH only declined at a rate of 0.0078 to 0.011 units per minute; a change of no clinical significance [55,72,74-76]. Nonetheless, immediate extraction of the fetus from the birth canal remained the recommended management in America according to most, even if not all relevant publications [21,54,38,77].

Some investigators have taken notice of the fact that the ingenious but technically faulty research of Wood et al. had led specialists into wrong direction. Edith Gurewitch on the ground of an investigation which involved over 200 patients demonstrated that the fetal capillary pH does not drop significantly unless the head-to-body delivery interval exceeds 8 minutes [78]. Approaching the subject from the angle of mechanical engineering, Allen demonstrated that the use of strong traction force during delivery is conducive to brachial plexus damage [79]. His research confirmed the earlier findings of French investigators that had been based upon experiments performed on fetal cadavers [80]. Allen also showed that when extraction of the fetus entailed difficulty, unintentionally and unconsciously doctors doubled the force of their traction. Based on a literary review Gurewitch and Allen concluded that excessive traction force applied at birth is the cause of most brachial plexus injuries [81].

Proof of the safety of the "two-step delivery" method was provided by Locatelli et al. [75]. In a study which involved almost 800 mothers they found that in the absence of intervention the average interval between delivery of the head and that of the rest of the body was only 88 seconds. During this time the fetal scalp blood pH only declined by about 0.001/ unit.

Discussion

Due to lack of consensus about the method of the delivery, shoulder dystocia has no widely accepted criteria. If the body is not expelled within 30 seconds after the head obstetricians in the USA try to extract the body and consider the diagnosis established if do not succeed promptly. Doctors who favor two-step delivery attach the diagnosis of shoulder dystocia only to those cases where the fetus remains still undelivered at the end of the next contraction.

Different interpretations invite different actions and different actions lead to different consequences. One may be tempted to think that the differing rates of shoulder dystocia in various practices are simply reflections of the fact that what some obstetricians consider arrest of the shoulders others prefer to call "two-step-delivery". It could also be deduced on this basis that in the absence of common denominator the reported differences in the rates of shoulder dystocia have little significance. Unfortunately, this is not the case. According to reliable statistics the rate of "shoulder dystocia" related fetal damage in the United States is as high as 10% [23]. It follows from this information that arrest of the shoulders that physicians invite by unwarranted interventions is not less conducive to brachial plexus injuries than those brought about by Mother Nature.

The physiology of childbirth provides explanation for the above outlined facts. Interruption of the delivery process after the expulsion of the head signals that the pregnant uterus (a far stronger group of muscles than any man's biceps) has been unable to overcome with one single effort bony and/or soft tissue resistance to the passage of two large body parts, namely the head and shoulders. When doctors try to extract the body with "gentle" traction after the dissipation of uterine systole, they attempt to achieve something that the uterus had been unable to accomplish despite its superior strength and efficient squeezing mechanism. Upon encountering resistance in this process, the doctor is correct when concludes that the shoulders became arrested. However, this arrest was man-induced and could have been avoided.

When the vacuum extractor was introduced into practice several decades ago, it was stipulated that traction must be used synchronously with uterine contractions in order to minimize the risk of fetal damage from either force [82]. Why this expediency that often delays the births even of distressed fetuses is ignored when perfectly healthy babies are delivered, is a puzzle which is difficult to fathom.

Authors of textbooks who changed their instructions concerning the method of delivery had little reason to anticipate that the new approach would have an adverse effect. Their relevant advice emphasized that the force used for the extraction of the fetus should be gentle. They could reasonably assume therefore, that in case of difficulty physicians would abandon their efforts and fall back to the technique they had learned from previous editions of the same books. It required the insight of an engineer to demonstrate that interpretation of the term "gentle" varies on a broad range and includes even sheer force when emotions

rise in response to assumed or real danger signs [79,81]. These authors may have also overlooked that the term gentle cannot be quantitated. Besides, human memory is short and new information tends to sink old experience into oblivion. Lenin, the spiritual leader of the bloody Russian Revolution displayed more wisdom in this regard than what most of us possess. When one of his scrupulous comrades warned him that his contemplated policy statement prepared for "Pravda" would contradict another that he had published only a few days earlier, he answered with cool equanimity:

-"Nobody reads yesterday's newspaper."

Nobody reads yesterday's textbook either. Thus the fact that the management of the birthing process had been changed overnight was overlooked or quickly forgotten by contemporary practitioners.

Physicians take pride in their work and are disinclined to admit if some of their actions prove to be less than perfect. At times the reason for such reluctance may be other than fear of losing patients' respect. Four decades ago when the change from conservative to interventionist management of the birthing process was discussed during a grand round the writer heard the following comment from the mouth of a well-respected obstetrician:

-"How could I justify my fees, if I stood by idly when my patient gave birth to her child?"

He thought that the service should be commensurate with the fee rather than the fee with the service.

Established activity patterns are habit forming. This being the case, it is not easy for physicians to change their conducts of practice. However, since helping into this world a disabled child is a painful experience for both the mother and the physician and because escalating malpractice claims will continue to darken obstetricians' horizons if the need for change is ignored, obviously a decision will have to be made eventually.

Conclusions

In light of the above summarized facts there is no justification for persevering with a ritual which has proved to be conducive to birth injuries. Acceptance of this technique did not rest upon evidence provided by controlled prospective or even retrospective studies. It represents one of the well-intended errors that have numerous precedents in medical history. Its abandonment should not be tied therefore to experiments that are bound to be costly both in terms of dollars and those of human lives.

Interference by the physician with the birthing process is reflection of generations' old misconception which gained undeserved acceptance by the profession and the general public alike, namely that the physician "delivers" the child. Ever since "homo sapiens" had abandoned the community of his fellow primates, babies have been delivered by their mothers, sometimes with, sometimes without assistance. Not unlike in other aspects of medicine, active involvement of a doctor only becomes needed when the childbearing process deviates from the path established by Providence. This principle, first established millennia ago by Hippocrates was embraced by Roman physicians and remains valid in the 21st Century also: "Primum non nocere!"

New ideas and search for new avenues have brought about amazing progress in the science and practice of medicine. Nonetheless, theoretically attractive ideas have frequently led to dead-end roads. When this happens, there is no alternative to turning back and taking another route. The old British method of conducting delivery may not be the last word in the science of perinatology. However, it is the only safe way to follow at the present time. Future investigations should rest on this firm basis rather than on the quicksand of "active management" of labor and delivery. Unfortunate children and unlucky physicians have paid high prices for the error of the 1970's.

Had aggressive management of the birthing process been set up in 1975 as a research project with the British Islands serving as control, there would be good reason to abandon it at this time. The advantages of the conservative approach to the management of the birthing process have been demonstrated by now beyond reasonable doubt. To draw due attention to this fact has been the purpose of this study.

References

1. Gherman RB, Ouzounian JG, Goodwin TM (1999) Brachial plexus palsy: an in utero injury? Am J Obstet Gynecol 180: 1303-1307.
2. Nocon JJ, McKenzie DK, Thomas LJ, Hansell RS (1993) Shoulder dystocia: an analysis of risks and obstetric maneuvers. Am J Obstet Gynecol 168: 1732-1737.
3. Gherman RB (2005) Shoulder dystocia: prevention and management. Obstet Gynecol Clin North Am 32: 297-305.
4. Gherman RB, Chauhan S, Ouzounian JG, Lerner H, Gonik B, et al. (2006) Shoulder dystocia: the unpreventable obstetric emergency with empiric management guidelines. Am J Obstet Gynecol 195: 657-672.
5. Menkes JH (1990) Textbook of Neurology (4thedn), Lea & Febiger, Philadelphia.
6. Volpe JJ (1995) Neurology of the Newborn (3rdedn), WB Saunders, Philadelphia.
7. Pondaag W, Malessy MJ, van Dijk JG, Thomeer RT (2004) Natural history of obstetric brachial plexus palsy: a systematic review. Dev Med Child Neurol 46: 138-144.
8. Csapo A (1981) Force of labor. In: Iffy L, Kaminetzky HA (Eds) Principles and Practice of Obstetrics & Perinatology, New York.
9. Caldeyro-Barcia R, Schwarcz R, Belizan JM, Matell M, Nieto F, et al. (1974) Adverse perinatal effects of early amniotomy during labor. In: Gluck L (Ed.) Modern Perinatal Medicine, Chicago, Year Book Medical Publishers Inc.
10. Goodlin RC (1989) Intrapartum uterine activity: evaluation of an intrauterine pressure transducer. Obstet Gynecol 74: 283-284.
11. Schwartz R, Diaz AG, Belizan JM, Fescina R, Caldeyro-Barcia R (1977) Influence of amniotomy and maternal position on labor. In: Castelazo-Ayala L, Mac Gregor C (Eds.) Proceedings of the VIII World Congress of Gynecology and Obstetrics, Amsterdam-Oxford, Excerpta Medica, 377-391.
12. Eastman NJ, Hellman LM (1961) Williams Obstetrics (12thedn), New York, Appleton-Century-Crofts, Inc.
13. Ubachs JM, Slooff AC, Peeters LL (1995) Obstetric antecedents of surgically treated obstetric brachial plexus injuries. Br J Obstet Gynaecol 102: 813-817.
14. Iffy L, Brimacombe M, Apuzzio JJ, Varadi V, Portuondo N, et al. (2008) The risk of shoulder dystocia related permanent fetal injury in relation to birth weight. Eur J Obstet Gynecol Reprod Biol 136: 53-60.
15. Iffy L, Capo JD, Apuzzio JJ, Varadi V, Brimacombe M, et al. (2009) Conduct of delivery, shoulder dystocia and neurological birth injuries. Hung J Obstet Gynecol 72: 211-219.
16. Iffy L, Pantages P (2005) Erb's palsy after delivery by Cesarean section. (A medico-legal key to a vexing problem.). Med Law 24: 655-661.
17. Cha HH, Kim JY, Choi SJ, Oh SY, Roh CR, et al. (2012) Can a customized standard for large for gestational age identify women at risk of operative delivery and shoulder dystocia? J Perinat Med 40: 483-488.
18. Overland EA, Vatten LJ, Eskild A (2012) Risk of shoulder dystocia: associations with parity and offspring birthweight. A population study of 1 914 544 deliveries. Acta Obstet Gynecol Scand 91: 483-488.
19. Benedetti TJ, Gabbe SG (1978) Shoulder dystocia. A complication of fetal macrosomia and prolonged second stage of labor with midpelvic delivery. Obstet Gynecol 52: 526-529.
20. Brimacombe M, Iffy L, Apuzzio JJ, Varadi V, Nagy B, et al. (2008) Shoulder

dystocia related fetal neurological injuries: the predisposing roles of forceps and ventouse extractions. Arch Gynecol Obstet 277: 415-422.

21. American Academy of Pediatrics and American College of Obstetricians and Gynecologists (2002) Guidelines for prenatal care (5thedn), Washington.
22. Beer E, Mangiante G, Pecorari D (2006) Distocia Delle Spalle. Roma, Edizioni Internazionali.
23. ACOG Practice Bulletin (1997) Shoulder dystocia. No. 7. Washington.
24. Bofill JA, Rust OA, Devidas M, Roberts WE, Morrison JC, et al. (1997) Shoulder dystocia and operative vaginal delivery. J Matern Fetal Med 6: 220-224.
25. Smith RB, Lane C, Pearson JF (1994) Shoulder dystocia: what happens at the next delivery? Br J Obstet Gynaecol 101: 713-715.
26. Cheng YK, Lao TT, Sahota DS, Leung VK, Leung TY (2013) Use of birth weight threshold for macrosomia to identify fetuses at risk of shoulder dystocia among Chinese populations. Int J Gynaecol Obstet 120: 249-253.
27. Kees S, Margalit V, Schiff E, Mashiach S, Carp HJ (2001) Features of shoulder dystocia in a busy obstetric unit. J Reprod Med 46: 583-588.
28. Lurie S, Levy R, Ben-Arie A, Hagay Z (1995) Shoulder dystocia: could it be deduced from the labor partogram? Am J Perinatol 12: 61-62.
29. Strobelt N, Locatelli A, Casarico G, Ferrini S, Bonassera M, et al. (2006) Head-to-body interval time: what is the normal range? Am J Obstet Gynecol 195: 110-114.
30. Spong CY, Beall M, Rodrigues D, Ross MG (1995) An objective definition of shoulder dystocia: prolonged head-to-body delivery intervals and/or the use of ancillary obstetric maneuvers. Obstet Gynecol 86: 433-436.
31. Beall MH, Spong C, McKay J, Ross MG (1998) Objective definition of shoulder dystocia: a prospective evaluation. Am J Obstet Gynecol 179: 934-937.
32. Iffy L (1987) Discussion of the presentation of TL Gross at al. Am J Obstet Gynecol 156: 1416.
33. Mehta SH, Blackwell SC, Bujold E, Sokol RJ (2006) What factors are associated with neonatal injury following shoulder dystocia? J Perinatol 26: 85-88.
34. Page EW, Villee CA, Villee DB (1972) Human Reproduction: Essentials of Reproductive and Perinatal Medicine Philadelphia, WB Saunders.
35. Greenhill JP (1955) Obstetrics (11thedn), Philadelphia, WB Saunders.
36. Beck HC, Rosenthal AH (1958) Obstetrical Practice (7thedn), Baltimore, Williams & Wilkins.
37. Bryant RD, Danforth DN (1971) Conduct of normal labor. In: Danforth DN (Eds) Textbook of Obstetrics and Gynecology, (2nd Edn.), Harper & Row, New York.
38. Bottoms SF, Sokol RJ (1981) Mechanism and conduct of labor. In: Iffy L, Kaminetzky HA (Eds.) Principles and Practice of Obstetrics & Perinatology, Wiley, New York.
39. Rosevear SK, Stirrat GM (1996) Handbook of Obstetric Management. Oxford, Blackwell Science.
40. Myles M (1985) Textbook for Midwives (10th Edn.), Churchill-Livingstone, Edinburgh, 313-314.
41. Kovacs L, Pal A (2007) Elettani vajudas es szules. In: Papp Z. (Edn.) A Szuleszet-Nogyogyaszat Tankonyve. Semmelweis Publ., Budapest, 249-272.
42. Royal College of Obstetricians and Gynaecologists (2005) Shoulder dystocia. Guideline 42. London, 304.
43. Evans-Jones G, Kay SP, Weindling AM, Cranny G, Ward A, et al. (2003) Congenital brachial palsy: incidence, causes, and outcome in the United Kingdom and Republic of Ireland. Arch Dis Child Fetal Neonatal Ed 88: F185-189.
44. Friedman EA (1984) Monitorizacion del proceso del parto. In: Iffy L, Charles D (Eds.) Perinatologia Operatoria, Editorial Medica Panamericana, Buenos Aires, 499-510.
45. Ledger WJ (1983) Labor and delivery. In: Willson JR, Carrington ER, Ledger WJ (Eds.) Obstetrics and Gynecology. The C.V. Mosby Co, St. Louis, 380-414.
46. Romney SL, McGray G, Merril JA, Quilligan RJ, Stander R (1975) Gynecology and Obstetrics. The Health Care of Women. McGrew-Hill, New York, 661.
47. Zuspan FP, Quilligan EJ (1982) Practical Manual of Obstetrical Care. The CV Mosby Co., St. Louis, 256.
48. Gabbe SG, Niebyl JR, Simpson JL, Spellacy WN (1991) Obstetrics (2nd Edn.), Churchill-Livingstone, New York, 355.
49. Creasy RK, Resnik R (1989) Maternal-Fetal Medicine: Principles and Practice, (2nd Edn.), WB Saunders, Philadelphia, 515.
50. Pritchard JA, MacDonald PC, Gant NF (1985) Williams Obstetrics, (7thedn), Connecticut, Appleton-Century-Crofts, Norwalk, 340.
51. Cunningham FG, MacDonald PC, Gant NF, Levano KJ, Gilstrap LC III (1993) Williams Obstetrics (19th Edn.), Connecticut, Appleton & Lange, Norwalk, 381-383.
52. Zlatnik FJ (1994) Normal labor and delivery. In: Scott JR, DiSaia PJ, Hammond CB, Spellacy WN (Eds.) Danforth's Obstetrics and Gynecology. (7thedn), JB Lippincott Co., Philadelphia, 105-128.
53. O'Grady JP, Burkman RT (1998) Obstetric Syndromes & Conditions. The Parthenon Publishing Group, New York.
54. Cunningham FG, Leveno HJ, Bloom SL, Hauth JC, Gilstrap LC III, et al. (2005) Williams Obstetrics, (23rd Edn.), McGrew-Hill, New York, 514.
55. Wood C, Ng KH, Hounslow D, Benning H (1973) The influence of differences of birth times upon fetal condition in normal deliveries. J Obstet Gynaecol Br Commonw 80: 289-294.
56. Wood C, Ng KH, Hounslow D, Benning H (1973) Time--an important variable in normal delivery. J Obstet Gynaecol Br Commonw 80: 295-300.
57. Swartz DP (1960) Shoulder girdle dystocia in vertex delivery: clinical study and review. Obstet Gynecol 15: 194-206.
58. Schwartz BC, Dixon DM (1958) Shoulder dystocia. Obstet Gynecol 11: 468-471.
59. Foad SL, Mehlman CT, Ying J (2008) The epidemiology of neonatal brachial plexus palsy in the United States. J Bone Joint Surg Am 90: 1258-1264.
60. Seigworth GR (1966) Shoulder dystocia. Review of 5 years' experience. Obstet Gynecol 28: 764-767.
61. Parks DG, Ziel HK (1978) Macrosomia. A proposed indication for primary cesarean section. Obstet Gynecol 52: 407-409.
62. Dandolu V, Lawrence L, Gaughan JP, Grotegut C, Harmanli OH, et al. (2005) Trends in the rate of shoulder dystocia over two decades. J Matern Fetal Neonatal Med 18: 305-310.
63. Hopwood HG Jr (1982) Shoulder dystocia: fifteen years' experience in a community hospital. Am J Obstet Gynecol 144: 162-166.
64. Acker DB, Sachs BP, Friedman EA (1985) Risk factors for shoulder dystocia. Obstet Gynecol 66: 762-768.
65. Gross TL, Sokol RJ, Williams T, Thompson K (1987) Shoulder dystocia: a fetal-physician risk. Am J Obstet Gynecol 156: 1408-1418.
66. Gross SJ, Shime J, Farine D (1987) Shoulder dystocia: predictors and outcome. A five-year review. Am J Obstet Gynecol 156: 334-336.
67. Baskett TF, Allen AC (1995) Perinatal implications of shoulder dystocia. Obstet Gynecol 86: 14-17.
68. Nesbitt TS, Gilbert WM, Herrschen B (1998) Shoulder dystocia and associated risk factors with macrosomic infants born in California. Am J Obstet Gynecol 179: 465-480.
69. Lewis DF, Raymond RC, Perkins MB, Brooks GG, Heymann AR (1995) Recurrence rate of shoulder dystocia. Am J Obstet Gynecol 172: 1369-1371.
70. Ecker JL, Greenberg JA, Norwitz ER, Nadel AS, Repke JT (1997) Birth weight as a predictor of brachial plexus injury. Obstet Gynecol 89: 643-647.
71. McFarland MB, Langer O, Piper JM, Berkus MD (1996) Perinatal outcome and the type and number of maneuvers in shoulder dystocia. Int J Gynaecol Obstet 55: 219-224.
72. Gherman RB, Ouzounian JG, Goodwin TM (1998) Obstetric maneuvers for shoulder dystocia and associated fetal morbidity. Am J Obstet Gynecol 178: 1126-1130.
73. Stallings SP, Spong CY, Devidas M, McParland P, Keane D, et al. (2001) Shoulder dystocia and umbilical artery acidosis. Am J Obstet Gynecol 75: 268-274.
74. Leung TY, Stuart O, Sahota DS, Suen SS, Lau TK, et al. (2011) Head-to-body

delivery interval and risk of fetal acidosis and hypoxic ischaemic encephalopathy in shoulder dystocia: a retrospective review. BJOG 118: 474-479.

75. Locatelli A, Incerti M, Ghidini A, Longoni A, Casarico G, et al. (2011) Head-to-body delivery interval using 'two-step' approach in vaginal deliveries: effect on umbilical artery pH. J Matern Fetal Neonatal Med 24: 799-803.

76. Heazell AE, Judge JK, Bhatti NR (2004) A retrospective study to determine if umbilical cord pH correlates with duration of delay between delivery of the head and body in shoulder dystocia. J Obstet Gynaecol 24: 776-777.

77. Norwitz ER, Robinson JN, Repke JT (2002) Labor and delivery. In: Gabbe SG, Niebyl JR, Simpson JL (Eds.),- Obstetrics, (4th Edn.), Churchill Livingstone, New York, 353-394.

78. Gurewitsch ED (2007) Optimizing shoulder dystocia management to prevent birth injury. Clin Obstet Gynecol 50: 592-606.

79. Allen RH (2003) Complete brachial plexus impairment: a traction-related injury. Am J Obstet Gynecol 188: 858-859.

80. Metaizeau JP, Gayet C, Plenat F (1979) [Brachial plexus birth injuries. An experimental study (author's transl)]. Chir Pediatr 20: 159-163.

81. Gurewitsch ED, Allen RH (2011) Reducing the risk of shoulder dystocia and associated brachial plexus injury. Obstet Gynecol Clin North Am 38: 247-269.

82. Baggish MS (1981) Vacuum extraction. In: Iffy L, Kaminetzhy HA (Eds.) Principles and Practice of Obtetrics & Perinatology. Wiley, New York, 1509-1520.