Changing patterns of birth asphyxia and trauma over 20 years

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Comparison of birth asphyxia and trauma in the same obstetric service during periods 18 years apart shows some reassuring and some disquieting findings. Liberalized cesarean sections, electronic monitoring of fetal heart in labor, and replacement of opiate sedation by epidural anesthesia have had their effect. There has been dramatic reduction in perinatal death and neonatal encephalopathy due to birth asphyxia and trauma and only rarely do affected infants now develop permanent cerebral injury. Severe birth asphyxia, defined by need for prolonged ventilation, has, however, remained unchanged in frequency. Unexpectedly, fractures and paralyses have dramatically increased. The major hazard today for the term infant is the use of midforceps, which has become much more common in parallel with the increased use of pain relief by continuous epidural anesthesia. (AM. J. OBSTET. GYNECOL. 148:490, 1984.)

Developments in obstetrics over the past 20 years have been directed largely at avoiding birth asphyxia and trauma. Mortality rates from these causes are now too low to provide adequate indicators of success. Neonatal morbidity, although usually of short-lived importance to the infant, provides a more sensitive index.

In a previous publication from this institution,[1] the incidence and causes of birth asphyxia and trauma from 1960 to 1962 were reviewed. Obvious obstetric complications or fetal pathology accounted for most of the morbidity. It was concluded that "improved obstetric management with more frequent use of cesarean section delivery might have been of value in preventing much of this fetal injury."

The present paper applies the same diagnostic criteria and method of assignment of cause to the births from 1978 to 1980. The purpose of this study was to determine whether deleterious outcomes seen in 1960 to 1962 have been avoided and whether any new hazards have developed with increased obstetric intervention.

In addition to the analysis of immediate neonatal morbidity, the study reviews the incidence and causes of postasphyxia/posttrauma perinatal death in both periods and reports the outcome on follow-up of the most severely asphyxiated infants in the 1978 to 1980 period.

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Presented at the Thirty-ninth Annual Meeting of the Society of Obstetricians and Gynaecologists of Canada, Vancouver, British Columbia, Canada, June 14-18, 1983.

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Material and methods

The Royal Victoria Hospital is a major city-center teaching institution serving a population of mixed ethnic and socioeconomic background. High-risk referrals from other hospitals (4% of deliveries) were excluded from consideration to permit comparison with previous experience.

The following definitions of severe birth asphyxia and trauma were used in this study, as in the previous one: severe asphyxia—requiring more than 3 minutes of resuscitation by positive-pressure ventilation prior to onset of sustained regular breathing; postasphyxia/posttrauma encephalopathy—neonatal convulsions (tonic/clonic, usually repetitive, after perinatal asphyxia or trauma) or abnormal cerebral signs (cerebral irritation, that is, hypertonia, opisthotonus, tremor, irritability, bicycling movements, and/or depression, that is, hypotonia, lethargy, poor sucking) after perinatal asphyxia or trauma; meconium aspiration syndrome—clinical and radiologic evidence; paralyses—weakness from peripheral nerve injury, often improved by discharge; fractures—crepitation, confirmed radiologically in most cases.

Neonatal diagnoses were assigned at the time of discharge by one of the authors (R. U.), who was also involved in the hospital care of most of the affected infants in both time periods. Perinatal information for 1978 to 1980 was computer coded shortly after dis-

Table I. Comparative obstetrical statistics, 1978 to 1980 and 1960 to 1962, Royal Victoria Hospital

Parameter	*1960-1962*	*1978-1980*
No. of live births	10,995	9901
Low-birth weight incidence (No./1,000)		
500 to 999 gm	5.1	4.2
1,000 to 2499 gm	65.2	49.3
Perinatal mortality rate to 7 days (No./1,000)		
>500 gm	22.0	11.0
>1,000 gm	16.5	7.0
>2,500 gm	6.1	3.5
Opiate analgesia (%)	48.1	2.8
Epidural analgesia/anesthesia (% vaginal deliveries ≥ 1,000 gm)	22.0	65.1
Electronic fetal monitoring in labor (%)	0	74.9
Low forceps (%)		
Vaginal deliveries	21.8	27.5
Total deliveries	20.7	22.2
Midforceps (%)		
Vaginal deliveries	5.3	15.3
Total deliveries	5.0	12.4
Cesarean section (%)		
Total	5.1	19.1
Primary	2.9	11.9
For low-birth weight infants	8.0	31.5

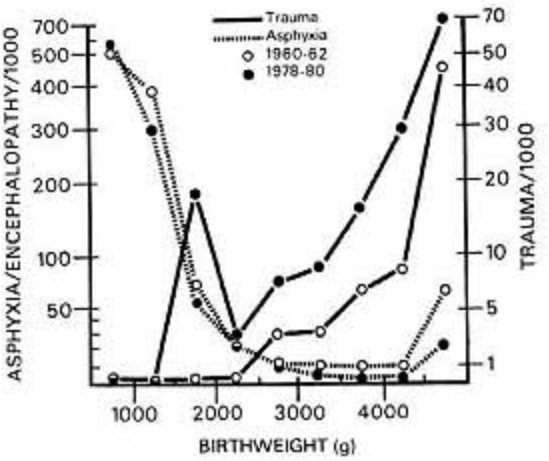

Fig. 1. Incidence of severe birth asphyxia or neonatal encephalopathy and of birth trauma (fractures, paralyses) per 1,000 live births by 500 gm weight groups.

charge. This data bank was used to identify the affected infants and to determine the frequency and risk of specific obstetric procedures and complications in the Royal Victoria Hospital population.

One of the authors (R. C.) reviewed the maternal and neonatal chart for each case of severe asphyxia or trauma and, wherever possible, determined the primary and contributing causes of these complications. All such assignments were reviewed by R. U. in order to enhance consistency with the earlier study.

Perinatal deaths due to asphyxia and trauma, not specifically dealt with in the previous paper, were reviewed from detailed perinatal mortality committee summaries prepared at the time of death; these were available for 1978 to 1980 and 1961 to 1962 but not for 1960. The incidence and causes of deaths that could be attributed to birth asphyxia and/or trauma were determined in both eras.

Infants born in 1978 to 1980 who manifested postasphyxia/posttrauma encephalopathy were followed up by the Developmental Pediatric Programme of the Royal Victoria Hospital or the Neurology Department of the Montreal Children's Hospital.

Results

Characteristics of the obstetric populations and methods of management in 1960 to 1962 and 1978 to 1980 are compared in Table I. Notable are a large decrease in perinatal mortality and a significant decrease in low—birth weight infants. Opiate analgesia has been replaced by continuous epidural analgesia/anesthesia. Electronic fetal monitoring is now routine, and cesarean sections are much more liberally performed. At the same time, however, forceps deliveries have increased rather than declined. Midforceps, particularly, have increased threefold.

Different types of neonatal postasphyxia/posttrauma morbidity have responded in a variable manner to the changes in obstetric management (Table II). Encephalopathy, which represents the most hazardous outcome, has declined by two thirds. Severe birth asphyxia (need for prolonged ventilation) remains as frequent as before, over all, and within each weight category. Fractures and paralyses have increased markedly, despite the liberal cesarean section policy. Meconium aspiration cannot be compared with previous figures as it was not included in the earlier study. Fractures, paralyses, encephalopathy, and meconium aspiration were seldom observed in low-birth weight infants.

A number of infants had combinations of various postasphyxia or posttrauma outcomes (Table III). Infants with a prolonged need for ventilation occasionally developed encephalopathy. The contribution of traumatic delivery to birth asphyxia is seen in the combination of fractures or paralyses (particularly skull or brachial) with severe birth asphyxia or encephalopathy. Meconium aspiration was often associated with severe birth asphyxia and occasionally with paralyses or fractures.

The close correlation between degree of birth asphyxia, as measured by minutes of ventilation required, and encephalopathy is evident from Table IV.

Table II. Incidence of severe asphyxia and trauma in 1978 to 1980 (with comparative rates from 1960 to 1962)

Parameter	Birth weight (gm)					
	500-999			1,001-2,499		
	1978-1980 (42 live births)		1960-1962 (56 live births)	1978-1980 (488 live births)		1960-1962 (717 live births)
	No.	No./1,000	(No./1,000)	No.	No./1,000	(No./1,000)
Severe asphyxia, postasphyxic/post trauma encephalopathy						
Severe asphyxia	25	595.2	500.0	23	47.1	55.8
Cerebral depression/irritation	0	0.0	18.0	2	4.1	8.4
Convulsions	0	0.0	0.0	1	2.0	2.8
Total infants affected	25	595.2	500.0	24	49.2	58.7
Meconium aspiration syndrome	0	0.0	—	1	2.0	—
Trauma						
Skull fracture	0	0.0	0.0	0	0.0	0.0
Clavicular fracture	0	0.0	0.0	0	0.0	0.0
Brachial paralysis	0	0.0	0.0	2	4.1*	0.0
Facial paralysis	0	0.0	0.0	1	2.0	0.0
Other fractures and paralyses	0	0.0	0.0	1	2.0	0.0
Total infants affected	0	0.0	0.0	3	6.1*	0.0
Total infants affected (asphyxia, meconium aspiration or trauma)	25	595.2		28	5.7	

*$p < 0.05$.

Table III. Combinations of asphyxia/trauma outcomes (≥2,500 gm)

	Severe asphyxia	Encephalopathy	Meconium aspiration	Clavicular fracture	Skull fracture	Brachial palsy	Facial palsy	None	Total
Severe asphyxia	—	12	4	0	1	2	0	32	45
Encephalopathy	12	—	6	1	4	3	0	2	21
Meconium aspiration	4	6	—	1	1	3	1	23	33
Clavicular fracture	0	1	1	—	0	4	2	47	54
Skull fracture	1	4	1	0	—	1	0	4	8
Brachial palsy	2	3	3	4	1	—	4	17	28
Facial palsy	0	0	1	2	0	4	—	32	38

Causes of severe birth asphyxia and encephalopathy have not changed over the 18 years, although their relative importance has altered (Table V). Prolonged labor and malpresentation play much smaller roles, as expected with a higher rate of cesarean section delivery. Opiate sedation of low-birth weight infants has been eliminated by epidural anesthesia, but some term infants are quite depressed after elective repeat cesarean section with the mother under general anesthesia. Unattended delivery and inadequate resuscitation of low-birth weight infants are no longer problems. Intrauterine growth retardation is declining in importance, while maternal hypertensive disease and intrauterine infection are increasingly recognized as etiologic factors in birth asphyxia.

Midforceps delivery remains the single most important cause of birth asphyxia in term infants (Table V) and is the major contributor to serious birth trauma (Table VI). Deliveries described as low forceps were associated with less marked increases in incidence of asphyxia, clavicular fracture, and brachial paralysis. Primary cesarean sections had a high incidence of severe asphyxia and meconium aspiration. Breech presentation, although usually managed by cesarean section, was associated with an increased risk of severe asphyxia. There was one case of meconium aspiration and one of femoral epiphyseal fracture occurring in a breech delivery by cesarean section.

Large fetal size was a factor in meconium aspiration, clavicular fracture, and brachial and facial paralyses (Table VI), whereas severe asphyxia/encephalopathy and skull fracture occurred in infants of average size. In Fig. 1 the comparative incidence of severe birth asphyxia and trauma is portrayed by 500 gm weight groups for the two periods; asphyxia has remained relatively constant while trauma has increased for each weight group.

Of the maternal and fetal factors analyzed in 1960 to

Birth weight (gm)					
≥2,500			Total		
1978-1980 (9,371 live births)		1960-1962 (10,222 live births)	1978-1980 (9,901 live births)		1960-1962 (10,995 live births)
No.	No./1,000	No./1,000	No.	No./1,000	No./1,000
45	4.8	5.2	93	9.4	10.9
19	2.0*	5.4	21	2.1*	5.6
7	0.7*	1.8	8	0.8*	1.8
54	5.8*	9.8	103	10.4*	15.4
33	3.5	—	34	3.4	—
8	0.9*	0.2	8	0.8*	0.2
54	5.8*	1.0	54	5.5*	0.9
28	3.0*	1.5	30	3.0*	1.4
38	4.1	2.4	39	3.9*	2.3
4	0.4	0.2	5	0.4	0.1
122	13.0*	4.8	125	12.6*	4.5
192	20.5		245	24.7	

1962, the risk of nulliparity remains while the risk of advanced maternal age has disappeared (Table VII). The increased risk found in postterm births and in male infants is in both cases due to macrosomia and is not seen when fetal weight is held constant.

Analysis of asphyxia or trauma with different obstetric complications (Table VIII) shows a high risk of severe asphyxia/encephalopathy after prolapsed cord, antepartum hemorrhage, and shoulder dystocia. The risk of severe asphyxia has decreased with prolonged labor, malpresentation, and macrosomia, although there is more trauma now with macrosomia. Midforceps delivery, three times more common than in the past, has one third the incidence of severe asphyxia but a higher rate of birth trauma per 100 midforceps deliveries than before.

Table IX was constructed to see whether the severe asphyxia and trauma associated with midforceps delivery was related to the experience of the accoucheur. There was no tendency for asphyxia and trauma to decrease with experience; if anything, the more recently trained obstetricians had fewer unfortunate outcomes.

Table X demonstrates the ninefold reduction in perinatal deaths attributable to birth asphyxia or trauma over the past 18 years. This decrease occurred mainly from the elimination of deaths due to prolonged labor, difficult delivery, and fetal malnutrition.

Follow-up information was available for 21 of 25 infants who developed postasphyxia/posttrauma encephalopathy in 1978 to 1980.

At an average age of 22 months the incidence of significant cerebral palsy, convulsions, hemiparesis, and/or developmental delay was one of 15 (7%) for infants with neonatal cerebral depression or irritation and four of six (67%) after postasphyxia/posttrauma convulsions. This gives a cerebral sequelae rate from severe birth asphyxia/trauma of 0.5/1,000 total live births. No comparable figures are available for 1960 to 1962.

Comment

Changes in obstetric practice over the past 20 years have been associated with almost complete elimination of perinatal death (0.6/1,000) and permanent cerebral handicap (0.5/1,000) caused by peripartum asphyxia and trauma. Neonatal postasphyxia encephalopathy has also become much less frequent. Concern today centers on the inability to reduce the incidence of birth asphyxia, defined by need for prolonged ventilation, in any weight group. Even more worrisome is a definite increase in incidence of birth trauma associated particularly with midforceps delivery of the full-size infant.

Rates of birth asphyxia in our study are almost identical for each weight group to those reported in 1970 to 1975 by MacDonald and associates.[2] Their incidence of postasphyxia convulsions (1.4/1,000) is midway between our earlier and later rates.

The failure to note a decrease in need for ventilation is at variance with two similar studies[3, 4] which attributed decreasing frequency of low Apgar scores to fetal monitoring.

Table IV. Incidence of postasphyxia/posttrauma encephalopathy and severity of birth asphyxia (≥2,500 gm)

Birth asphyxia (duration of ventilation) (mins)	*No. of infants*	*Cerebral irritation/depression without convulsions*	*Convulsions*	*Total*	
				No.	*%*
0	9,032	4	0	4	—
1-3	294	4	1	5	2
4-5	28	2	1	3	11
6-10	11	2	1	3	27
≥11	6	2	4	6	100

The criterion of more than 3 minutes' ventilation for the diagnosis of severe asphyxia in this study is not perfectly equable with low Apgar scores. Apgar scores of <4 at 1 minute of age were assigned in 67% of our "severely asphyxiated" infants but occurred overall in three times as many infants as required prolonged ventilation. Apgar scores of <7 at 5 minutes were recorded in 60% of our cases, but the overall incidence was two times greater than by our definition.

Addy,[5] summarizing the literature on birth asphyxia and its relationship to subsequent outcome, found that the time to spontaneous respiration appeared to be the most useful guide to prognosis with regard to brain damage. Nelson and Ellenberg[6] found that cerebral palsy causing substantial handicap occurred in 1% of survivors with a low 1-minute Apgar score, 5% of those with a low 5-minute Apgar score, and 41% of those with a low 20-minute Apgar score. Mulligan and associates[7] described neonatal seizures in 5% of infants requiring 2 to 5 minutes' ventilation and in 52% of those needing more than 5 minutes to sustain breathing. In our study, it is relevant to note in this regard that duration of ventilation required was closely correlated with the frequency of encephalopathy and encephalopathy (especially convulsions) with brain damage.

Of the causes of birth asphyxia found in our studies, fetal heart rate patterns not associated with complications of pregnancy or labor were of little predictive value. They may, in fact, contribute to birth asphyxia and trauma by prompting rapid forceps extraction, often unnecessary as end-stage decelerations are frequently benign.

Among low-birth weight infants, hypertensive disease now plays a major role as a cause of asphyxia. Intrauterine infection has become of great importance, possibly because of more effective methods of inhibiting premature labor and the use of steroids. Sedation and unattended delivery have disappeared as factors with the replacement of opiates by epidural analgesia and the closer supervision of progress of premature labor.

The marked reduction in asphyxia caused by prolonged labor and malpresentation in 1978 to 1980 can be attributed to more liberal use of cesarean section delivery than in 1960 to 1962. Cesarean delivery, however, no longer appears to be as hopeful a means to reduce birth asphyxia as was previously thought.[1] The overall incidence of need for prolonged ventilation has not altered with a fourfold increase in primary cesarean rate, although rates of postasphyxia encephalopathy and deaths have improved greatly. Low-birth weight infants are as often asphyxiated as before with four times as many now being delivered by cesarean section. An indication that today's liberal cesarean section rate may be higher than necessary is provided by two other studies from this institution. In these, no improvement in need for prolonged ventilation could be shown over the last decade with a fourfold increase in cesarean section rate for the term breech infant[8] and a threefold increase among macrosomic infants.[9]

In cephalic presentations of full-size infants during 1978 to 1980, severe asphyxia, encephalopathy, or meconium aspiration often occurred in infants who were delivered by cesarean section. This implies that cesarean section for fetal distress or failure to progress is sometimes performed too late to prevent fetal asphyxia. General anesthesia, the cause of severe asphyxia in four infants delivered by elective repeat cesarean section, plays a less definable role in primary cesarean sections in which the indication for the cesarean section may also contribute to birth asphyxia.

Midforceps delivery was associated with more than half of the severe asphyxia/encephalopathy found in vaginal vertex deliveries of full-size infants. Its role in the production of asphyxia must be discussed along with that of the trauma which often coexists.

Although it has been claimed that the incidence of birth trauma is declining, the higher rates for individual fractures and paralyses found in recent years at the Royal Victoria Hospital are not dissimilar to those reported for these lesions in recent literature. Rates of clavicular fracture of 2.8 to 7.2 per 1,000 have been reported (compare with our rate of 5.5/1,000). Rubin[10] found that most clavicular fractures were related to shoulder dystocia in cephalic presentations, whereas Cohen and Otto[11] noted shoulder dystocia in only one of 24 such fractures, midforceps in one third, and macrosomic babies in 60% of their cases. It is possible that

Table V. Primary cause of severe asphyxia, abnormal cerebral signs, and convulsions in 1978 to 1980 compared with 1960 to 1962

Primary cause	*500-999 gm*		*1,000-2,499 gm*		*≥2,500 gm*	
	1978-1980	*1960-1962*	*1978-1980*	*1960-1962*	*1978-1980*	*1960-1962*
No. of affected infants	25	28	24	42	54	99
Complications of labor (%)						
Antepartum hemorrhage	16	14	8	12	4	0
Prolapsed cord	4	0	4	7	6	3
Cord loops, knots	0	0	0	0	9	4
Prolonged labor	0	4	0	2	4*	28
Hypertensive disease	0	0	25*	0	4	0
Unexplained fetal distress	0	0	4	0	19	9
Total	20	18	41	21	46	44
Complications of delivery (%)						
Midforceps delivery	0	0	4	0	19	18
Shoulder dystocia	0	0	0	0	6	5
Malpresentation	16	11	4	19	4	11
Birth weight 4,500 gm	0	0	0	0	0	11
Precipitate delivery	0	0	0	0	2	3
Total	16	11	8	19	31	39
Problems in management (%)						
Sedation	0*	18	0	7	7	4
Unattended delivery	0*	14	0	0	0	0
Inadequate resuscitation	0	3	0	3	0	0
Total	0	35	0	10	7	4
Fetal pathology (%)						
Congenital anomalies	4	0	8	7	9*	0
Rh erythroblastosis	0	0	4	12	0	1
Intrauterine infection	44*	7	21*	2	0	2
Intrauterine growth retardation	8	4	13	24	4	6
Other	0	0	4	0	0	0
Total	56†	11	50	45	13	9
Unexplained	8	25	0	5	6	4
Total	100	100	99	100	103	100

*$p < 0.05$ for differences between 1978 to 1980 and 1960 to 1962.

the rapid descent associated with midforceps delivery often does not permit normal rotation of the shoulders and that traction on the head can lead to fracture of the clavicle or damage to the brachial plexus.

Skull fracture is reported by Rubin[10] to have an incidence of 0.065/1,000, much lower than that observed here. The fourfold increase observed over the past 18 years at our hospital parallels the threefold increase in midforceps deliveries during this interval.

The incidence of brachial paralysis in the literature has been quoted as anywhere from 0.14 to 1.89 per 1,000, our own being 3.0/1,000 recently. Rubin[10] found 44% were associated with breech presentation (none of ours were so related, even in 1960 to 1962, with breech deliveries usually vaginal), and 56% with shoulder dystocia (compared with 27% of our own). Hardy[12] found an incidence of brachial paralysis of 0.87/1,000 with 84% in cephalic presentations, of which 80% were delivered by forceps. It is evident from both of the populations studied in our institution that brachial paralysis is usually produced here by midforceps delivery, with shoulder dystocia being the other important factor.

The incidence of facial paralysis in the literature has been 0.7 to 1.4 per 1,000 compared with 3.8/1,000 in our hospital. Rubin[10] found that 19 of 21 were associated with forceps delivery compared with 74% here.

The literature in recent years has supported the concept that midforceps deliveries are dangerous.[13-15] Bowes and Bowes[14] reported no significant perinatal mortality in a control group of patients who had a prolonged second stage of labor and who underwent cesarean section. In contrast, 14 of 71 cases of midforceps delivery were followed by birth injury, including severe asphyxia in four. Hughey and co-workers[15] reviewed 458 consecutive midforceps deliveries, of which 31% produced neonates with unfavorable outcome compared with 0% of a similar control group delivered by cesarean section. The speculation that obstetricians

Table VI. Birth asphyxia and trauma by method of delivery for infants ≥2,500 gm, 1978 to 1980

Method of delivery	*Live births (no.)*	*Severe* asphyxia*		*Meconium aspiration*		*Skull fracture*	
		No.	*No./1,000 live births*	*No.*	*No./1,000 live births*	*No.*	*No./1,000 live births*
Mean weight (gm)	3,418	3,332		3,762		3,348	
Vaginal							
Vertex, spontaneous	4,355	8	1.8	15	3.4	0	0
Vertex, low forceps	2,094	6	2.9	5	2.4	0	0
Vertex, midforceps	1,171	13	11.1†	5	4.3	7	6†
Breech	67	2	29.9†	0	0.0	0	0
Cesarean							
Cephalic, elective repeat	673	4	5.9	1	1.5	1	1.5
Cephalic, primary	719	18	25.0	6	8.3	0	0
Breech, transverse	315	3	9.5	1	3.2	0	

*Severe birth asphyxia, postasphyxia/posttrauma abnormal cerebral signs or convulsions.
†Different from spontaneous vertex delivery: $p < 0.05$.
‡Includes one fractured femur.

Table VII. Maternal and fetal factors associated with birth asphyxia and trauma, 1978 to 1980 (No./1,000 live births)

Maternal/fetal factors	*No. of live births*	*Severe birth asphyxia**	*Meconium aspiration*	*Birth trauma†*	*Total affected infants*	*Total for 1960 to 1962*
Parity						
Nulliparous	4,691	11.7	4.5	16.4‡	30.5‡	26.7
Multiparous	5,206	9.2	2.5	9.2‡	19.6‡	15.4
Age						
Nulliparous						
<35 yr	4,492	12.0	4.5	16.5	30.7	25.3
≥35 yr	176	5.7	5.7	17.0	28.4	72.7
Multiparous						
<35 yr	4,593	9.1	2.4	9.4	17.6	13.3
≥35 yr	603	10.0	3.3	8.3	18.2	26.8
Gestational age (≥2,500 g)						
<42 wk	7,883	6.2	3.7	13.8	21.8	14.2
≥42 wk	617	8.1	4.9	19.4	29.2	12.3
Fetal sex						
Male	5,130	11.9	5.3‡	14.6	28.5‡	22.8
Female	4,767	8.8	1.5‡	10.5	20.8‡	14.9

*Severe asphyxia neonatorum and postasphyxia/posttrauma abnormal cerebral signs or convulsions.
†Fractures and paralyses.
‡$p < 0.05$.

trained in recent years are less proficient in the use of midforceps has been laid to rest here. Friedman[13] advocates abolition of midforceps delivery, a conclusion which in our institution could increase the primary cesarean rate from 12% to 24%.

Is it possible that the frequent use of continuous epidural analgesia and anesthesia is responsible for the increase in forceps use[16] despite a high cesarean section rate? Or could it be that the modern obstetricians have become less willing to wait for spontaneous delivery when rotation and descent occur slowly during the second stage? In 59% of midforceps-related severe asphyxia or trauma, fetal distress was not a factor. In these cases the median duration of second stage was 73 minutes in nulliparous patients and 35 minutes in multiparous patients. In only nine of 72 did the obstetrician await the 2 hours in nulliparous or 1 hour in multiparous patients usually considered as acceptable upper limits of normal second-stage labor. With adequate monitoring and scalp pH available, even longer times than these are recognized as without risk.[13] Low-forceps delivery also does not escape risk-free in this analysis and raises the same questions, although to a lesser degree, discussed for midforceps.

On the other hand, the vast majority of forceps deliveries (98% of low forceps and 94% of midforceps) produced healthy, uninjured babies. Asphyxia and trauma caused by forceps use are presumably the result

Clavicle fracture		Brachial paralysis		Facial paralysis		Infants affected	
No.	No./1,000 live births	No.	No./1,000 live births	No.	No./1,000 live births	No.	No./1,000 live births
3,764		3,711		3,678			
14	3.2	3	0.7	8	1.8	45	10.3
21	10.0†	6	2.9†	5	2.4	36	17.2†
19	16.2†	18	15.4†	24	20.4†	73	62.3†
0	0	0	0	0	0	2	29.9
0	0	0	0	1	1.5	6	8.9
0	0	1	1.4	1	1.4	24	33.4†
0	0	0	0	0	0	5	15.9‡

Table VIII. Risk of severe birth asphyxia or trauma with different obstetric complications, 1978 to 1980 compared with 1960 to 1962

Complication	Incidence of obstetric complication, 1978-1980 (No.)	Risk of severe asphyxia*		Risk of trauma†	
		1978-1980 (No./1,000)	1960-1962 (No./1,000)	1978-1980 (No./1,000)	1960-1962 (No./1,000)
With complications of labor					
Antepartum hemorrhage	103	97	124		
Prolapsed cord	34	147	176		
Prolonged labor without fetal distress	185	0	44		
Prolonged labor with fetal distress	81	49	153		
Prolonged trial of labor (cesarean section)	42	71	156		
Hypertensive disease	275	51	Unknown		
Unexplained fetal distress	1,077	10	7		
With complications of delivery					
Midforceps	1,208	16	50	55	36
Shoulder dystocia	47	234	133	362	220
Malpresentation (term only)	348	22	49	9	10
Large baby (>4,500 gm)	130	23	62	62	46
Total live births	9,901	10	15	13	5

*Severe birth asphyxia or postasphyxia/posttrauma abnormal cerebral signs or convulsions.
†Fractures or paralyses.

Table IX. Midforceps and primary cesarean section rates by experience of accoucheur (≥2,500 gm, 1978 to 1980)

Attending staff (years since training)	Total deliveries (No.)	Primary cesarean section rate (%)	Midforceps deliveries		
			No.	% of vaginal deliveries	Incidence of severe asphyxial trauma (% of midforceps deliveries)
Senior (14+)	2,737	12.7	331	15.3	8.5
Middle (6 to 13)	3,035	10.7	386	15.7	7.0
Junior (0 to 5)	2,532	13.2	379	18.7	5.3

of excessive force and do not occur when low-forceps or midforceps delivery can be performed gently.

To reduce birth asphyxia and trauma in the full-size baby, forceps deliveries must be used less often and more carefully. Improved training for childbirth and psychological support during labor could reduce the 65% incidence of patients requesting pain relief in this series. The temptation to expedite delivery by use of midforceps when second stage is slowed should be resisted unless a precarious fetal state demands it. When

Table X. Mortality rates associated with peripartum asphyxia and trauma (birth weight ≥1,000 gm)

Years	*No. of deliveries*	*Intrapartum fetal deaths (No./1,000)*	*Neonatal deaths (No./1,000)*	*Perinatal deaths (No./1,000)*
1961 to 1962	7,409	2.7	2.9	5.6
1978 to 1980	9,895	0.3	0.3	0.6

forceps delivery is attempted but is difficult to perform, cesarean section for failed forceps should not be considered as a professional defeat but as a more acceptable alternative than forceful rotation and extraction.

Computerization of the obstetric and neonatal data base was performed by the Biomedical Engineering Department of McGill University, by Dr. Robert Funnel. Developmental pediatric assessments were performed by Dr. Diana Willis of the Royal Victoria Hospital.

REFERENCES

1. O'Brien, J. R., Usher, R. H., and Maughan, G. B.: Causes of birth asphyxia and trauma, Can. Med. Assoc. J. **94:**1077, 1966.
2. MacDonald, H. M., Mulligan, J. C., Allen, A. C., and Taylor, P. M.: Neonatal asphyxia. I. Relationship of obstetric and neonatal mortality in 38,403 consecutive deliveries, J. Pediatr. **96:**898, 1980.
3. Shamsi, H. H., Petrie, R. H., and Steer, C. M.: Changing obstetric practices and amelioration of perinatal outcome in a university hospital, Am. J. Obstet. Gynecol. **133:** 855, 1979.
4. Ingemarsson, E., Ingemarsson, I., and Svenninger, N. W.: Impact of routine fetal monitoring during labor on fetal outcome, Am. J. Obstet. Gynecol. **141:**24, 1981.
5. Addy, D. P.: Birth asphyxia, Br. Med. J. **284:**1288, 1982.
6. Nelson, K. B., and Ellenberg, J. N.: Apgar scores as predictors of chronic neurologic disability, Pediatrics **68:**36, 1981.
7. Mulligan, J. C., Painter, M. M., O'Donoghue, P. A., MacDonald, H. M., Allen, A. C., and Taylor, P. M.: Neonatal asphyxia. II. Neonatal mortality and long-term sequelae, J. Pediatr. **96:**903, 1980.
8. Green, J., McLean, F., Smith, L. P., and Usher, R. H.: Has an increased cesarean section rate for term breech delivery reduced the incidence of birth asphyxia, trauma, and death? Am. J. Obstet. Gynecol. **142:**643, 1982.
9. Boyd, M., McLean, F., and Usher, R. H.: Macrosomia, Obstet. Gynecol. **61:**715, 1983.
10. Rubin, A.: Birth injuries: Incidence, mechanisms and end results, Obstet. Gynecol. **23:**218, 1964.
11. Cohen, A. W., and Otto, S. R.: Obstetric clavicular fractures, J. Reprod. Med. **25:**119, 1980.
12. Hardy, A. E.: Birth injuries of the brachial plexus, J. Bone Joint Surg. **63:**98, 1982.
13. Friedman, E. A.: Whither midforceps? Its place in obstetrics today, Contemp. Ob/Gyn **21:**85, 1983.
14. Bowes, W. A., and Bowes, C.: Current role of midforceps operations, Clin. Obstet. Gynecol. **23:**549, 1980.
15. Hughey, M. J., McElin, T. W., and Lussky, R.: Forceps operations in perspective. I. Midforceps rotation operations, J. Reprod. Med. **20:**253, 1978.
16. Studd, J. W., Crawford, J. S., Duignan, N. M., Rowbotham, C. J., and Hughes, A. O.: The effect of lumbar epidural analgesia on the rate of cervical dilatation and the outcome of labour of spontaneous onset, Br. J. Obstet. Gynaecol. **87:**1015, 1980.