Is permanent congenital facial palsy caused by birth trauma?

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Abstract

Objective—To study the relation between traumatic birth and the development of permanent facial palsy in the newborn. Design—Retrospective case control study of children with 'congenital' facial palsy. Setting—Two tertiary referral centres for patients with facial palsy.

Subjects—61 children with established facial palsy.

Main outcome measures—Odds ratios of recognised factors for birth injury: maternal primiparity, high birth weight, and the use of obstetric forceps at delivery.

Results—13.2% of those studied had forceps assisted delivery compared to 10.2% in the normal population (odds ratio 1.34; 95% confidence intervals 0.61 to 2.97) 39.6% were born to primiparae compared to a national rate of 36.7% (1.13; 0.65 to 1.96) and only 18.9% weighed more than 3500 g at birth (0.37; 0.19 to 0.74).

Conclusions—There is no association between the development of permanent 'congenital' facial palsy and recognised risk factors for birth injury. These data suggest an intrauterine rather than a traumatic aetiology.

(Arch Dis Child 1996; 74: 56-58)

Keywords: facial palsy, birth trauma, forceps.

Permanent congenital facial palsy is associated with considerable disfigurement (fig 1). It is distressing for parents and causes functional and emotional problems for the affected child, particularly at school entry when peer teasing is common. Many parents and health care professionals consider the palsy to be caused by perinatal trauma. The superficial course of the extracranial facial nerve in neonates is offered in support of this assertion and the *International Classification of Diseases* (ICD-9) retains only one code for congenital facial nerve palsy: [767.5] 'seventh nerve palsy due to birth trauma'.¹

Neonatal tissues generally heal with less scarring and deformity than those of adults. Facial nerve neuropraxia in the newborn, resulting from traumatic birth, might be expected to result in a *temporary* facial palsy. However, there are affected children in whom the palsy is not transient. Plastic surgeons with a special interest in surgery to reanimate the paralysed face see many children with *permanent* congenital facial palsy. This may occur both in isolation and as part of a recognised syndrome.

If injury to the facial nerve during delivery is a major cause of permanent facial palsy, a substantial number of cases might be expected to have had a traumatic delivery. There is an increased risk of trauma in infants who are born to primiparous women, whose birth weight is above 3500 g, or who are born by forceps assisted delivery.^{2 3} This study compares the proportion of cases with risk factors for trauma with those in the population as a whole.

Methods

Mount Vernon Hospital and Great Ormond Street Hospital for Children are tertiary referral centres for facial palsy.⁴ At Mount Vernon Hospital cases were identified from a maintained database of all patients with facial palsy. At the Hospital for Sick Children a generalised diagnostic database was searched and supplemented by reference to the photographic department diagnostic index. Case notes were extracted for patients with congenital facial palsy treated in these units between 1983 and 1993.

Children were included in the study if they had a permanent isolated unilateral facial palsy which was diagnosed at or shortly after birth. They were excluded if any other associated congenital abnormality had been diagnosed before or after referral to the units. Children were considered to have a permanent palsy if it had not recovered by the age of 5 years.

Detailed obstetric histories were obtained from the patient's mother by postal questionnaire, with clarification by telephone where necessary. Further information regarding delivery was obtained from the relevant

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Accepted 13 September 1995

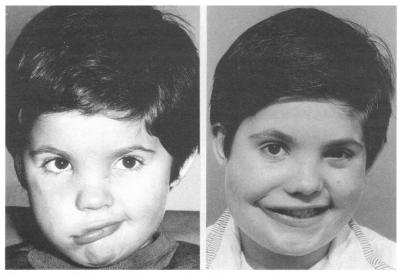


Figure 1 (A) Eight year old boy with right sided 'congenital' facial palsy attempting to smile; (B) one year after facial reanimation surgery using microsurgical nerve and muscle transfers.

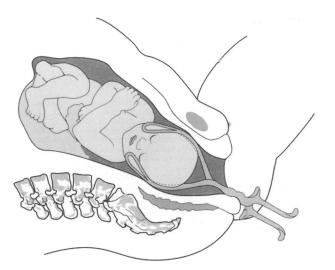


Figure 2 Obstetric forceps being applied to the fetal head. Their proximity to the facial nerve is why forceps are often implicated in congenital facial palsy.

obstetric unit and from the referring clinician's letter.

Data were collected about problems occurring during pregnancy, maternal age and parity, length of gestation, mode of delivery and use of forceps, the indication for instrumental or caesarean delivery, birth weight, and abnormal presentation. The presence of overt soft tissue injuries in the babies was also noted.

The proportions of cases born by forceps delivery, with abnormal presentation, whose birth weight was above 3500 g, or who were born to primiparous mothers, were calculated. These were compared with the population rates for these factors which were obtained from published national data.⁵⁶ The odds ratios and 95% confidence intervals were obtained by unpaired case control study.⁷

Results

Sixty one children with congenital facial palsy were identified. Of these eight were excluded because of other congenital abnormalities or syndromes (table 1), leaving a total of 53 children (23 boys and 30 girls) with isolated palsy. Response rate to the postal questionnaire was 100% and telephone follow up for clarification was required in only three cases.

Table 2 summarises the exposure of the children in the study to risk factors for traumatic birth. Seven of these children had a

Table 1Abnormalities leading to exclusion from the study(n=8).Some children had more than one abnormality

Congenital abnormality detected	Number	
Cleft lip and palate	2	
Limb deformity	3	
Hirschsprung's disease	1	
Microtia	ī	
DAOM-cardiac syndrome	ī	
Abducens palsy	1	
Moebius syndrome	3	

Table 2 Proportion of children with risk factors for traumatic birth

Risk factor	Study group	National data	Odds ratio	95% CI
Forceps delivery	7/53 (13·2%)	68 290/601 460 (10·2%)	1·34	0.61 to 2.97
Primiparity	21/53 (39·6%)	240 975/656 234 (36·7%)	1·13	0.65 to 1.96
Birth weight >3500 g	10/53 (18·9%)	257 191/666 612 (38·6%)	0·37	0.19 to 0.74

forceps assisted delivery, one of whom had extensive bruising and bilateral hip dislocation consistent with a difficult delivery. He weighed 2450 g and had fetal distress. Twenty six additional children were born to primiparous mothers or weighed more than 3500 g. None of the big babies was born to primigravidae. There was no clinical evidence of trauma and risk factors for injury were absent in the remaining 20 out of 53 cases.

There was no increased risk of forceps assisted delivery in cases with facial palsy when compared to the population as a whole (odds ratio 1.34: 95% confidence interval 0.61 to 2.97).

The prevalence of maternal primiparity was no greater among the mothers of affected children than expected from national data (odds ratio 1.13: 95% confidence interval 0.65to 1.96).

There were many fewer big babies (birth weight >3500 g) in the study group than in the general population (odds ratio 0.37: 95% confidence interval 0.19 to 0.74).

Discussion

It is well recognised that many children are born with transient weakness of facial expression after traumatic delivery.⁸ The extracranial course of the facial nerve as it leaves the stylomastoid foramen is very superficial and it might easily be contused, particularly in the event of a difficult birth, with impaction of the head against the pelvic outlet. The application of forceps for either traction or rotation might result in neuropraxia of one or both facial nerves (fig 2), particularly if applied incorrectly.9 The full recovery which is made by these children¹⁰ is consistent with current understanding of the regenerative capacity of neonatal tissues and their tendency to heal with less scar tissue than in later life.¹¹ Falco and Eriksson³ found that palsies with a clearly documented traumatic aetiology all had some degree of recovery, most having complete resolution within a mean of four months postgestation.

Over 40 years ago it was suggested by Hepner¹² that it was trauma in the birth canal rather than forceps that was responsible. Illingworth emphasised that there are many deformities for which obstetricians are held culpable inappropriately.¹³ None the less, the assumption that birth trauma is responsible for congenital facial palsy continues to be widely expressed.¹⁴ Many of the parents of affected children in this study had been told by clinicians or midwives that the palsy had been caused by birth trauma. This may have medicolegal consequences and could obscure the true aetiology of the condition.

By comparing the proportion of subjects with factors known to be associated with traumatic birth with those in the population as a whole, this study suggests that the risk of trauma was no higher in the study group than in the general population. In particular, these children were no more likely to have required forceps to assist delivery, nor was there other evidence for an association between birth trauma and permanent facial palsy.

The prevalence of major birth defects generally is high in low birthweight babies and low in big babies.¹⁵ The paucity of high birthweight babies in our study is striking. This further supports our hypothesis that this condition has an intrauterine and not a traumatic aetiology.

These children might also be expected to have associated developmental abnormalities. We accept that it would have been desirable to include developmental assessment in the study; however, the retrospective design and wide catchment of the two centres precluded this.

While the aetiology remains uncertain we should not allow the blame for permanent facial palsy to continue to fall upon the obstetrician or midwife.

This study was funded by the Restoration of Appearance and Function Trust (RAFT). We would like to thank Dr Martyn Sherriff, PhD, United Medical and Dental School, London, for assistance with statistical analysis and Jane Fallows, Medical Illustrator, Mount Vernon and Watford Hospitals NHS Trust, for fig 2.

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