Cerebral Palsies: Treatment and Prevention

The Croonian Lecture 1978

J. P. M. TIZARD, BM, FRCP
Professor of Paediatrics, University of Oxford

Dr William Croone died at the age of 51 in 1684. He was a most successful and much valued physician and a Censor of the Royal College of Physicians. He was also a founder member of the Royal Society and he had the curiosity, the imagination and the gift of experimenting that are the marks of a true scientist.

One of his major contributions to knowledge lay in his studies of muscular action published in his treatise entitled *De Ratione Motus Muscularum*[1] in which he set out his belief that muscular contraction was initiated by a nerve impulse originating in the brain. Thus, the group of conditions collectively and commonly known as cerebral palsy is not wholly inapposite as the subject of a Croonian lecture.

To begin with a brief case history: Mary, aged 42, is a helpless cripple. She cannot talk, although she can indicate yes and no and can smile and laugh. She is only able to feed herself with assistance and with great difficulty. She can hardly hold her head upright. She has very little voluntary movement of her arms and cannot attend to excretory functions, although she is not incontinent. She has never been able to stand up. And yet, and herein lies the real tragedy of her condition, she is of well above average intelligence.

For the first 18 months of her life no doctor could be persuaded that the child was abnormal although her mother was quite certain. Then a diagnosis of Little's disease was made and it was assumed that Mary was mentally defective. Only her mother's steadfast insistence that her child was intelligent procured for her a delayed education.

A home teacher was provided when Mary was 9½ years old. She was an exceptional person; she taught Mary to read, to type effectively if slowly with the forefinger of her left hand, and to learn the basic school subjects. Now she can express herself very well in a neatly typed letter. Deprived of most means of self-expression Mary has also developed a power of manipulating—I use the word figuratively—other people in a manner that would have called forth the reluctant admiration of the late Signor Niccolo Machiavelli.

This, then, exemplifies one extreme of the range of disabilities covered by the term cerebral palsy. The other extreme—and one of the difficulties of enumerating cases of cerebral palsy lies in the fact that no one has really agreed where it begins or ends—is sometimes exemplified by the clumsy child. Having been a clumsy child myself I can and do sympathise with others similarly handicapped but it cannot be regarded as one of life's greater tragedies.

The term cerebral palsy, only gradually and grudgingly accepted by neurologists, has been used by paediatricians for a long time, for instance by Robert Hutchison in the first edition of his *Lectures on Diseases of Children*[2] in 1905. It is a convenient term under which are categorised all defects of movement caused by lesions of the brain originating before birth, at birth, or in the early post-natal years. By convention it does not include defects of movement resulting from progressive cerebral disorders.

The first description of some forms of cerebral palsy was made by William Little*[3]* in 1843. He attributed these disorders to abnormal delivery. The connection may seem obvious now, but Little's discovery was a shrewd clinical observation because, as a rule, defect of movement is not apparent at first in infants who months later show unmistakable signs of cerebral motor defect. Thus, the connection between events at birth and the subsequent malfunction is far from obvious.

For about 100 years after William Little's publications the term Little's disease was used to describe children thus afflicted, but Little had described but one broad type of what is now included under the diagnosis of cerebral palsy and gradually the more comprehensive non-eponymous term was accepted.

Classification of the cerebral palsyes has been the subject of windy controversy for years, and well it might, since pathological processes do not respect anatomical boundaries in the brain, let alone functional boundaries to the extent that these exist. Defects of the brain other than those causing motor disability may co-exist: mental defect, sensory defects and epilepsy. The motor defects alone are variably manifest. Thus, the late Dr Winthrop M. Phelps*[4]* classified athetosis into no less than 18 subgroups.

Figure 1 shows my simplified classification of the cerebral palsyes and its only claim to originality lies in the fact that it is not precisely like anyone else's.

Thirty years ago Dr Philip Evans*[5]* gave a learned, useful and amusing talk on the classification of the cerebral palsyes to a meeting at the Royal Society of Medicine. He said, '... a simple division of cases into spastic and athetoid has been known to arouse in a neurologist the unbecoming passion of anger'. Never-
The cerebral palsies

Spastic
- Monoplegia
- Hemiplegia
- Paraplegia
- Tetraplegia

Dyskinetic (bilateral)
- Athetosis
- Choreaathetosis
- Dystonia

Ataxic

Fig. 1. The cerebral palsies.

theless, in practice, and bearing in mind that a child may exhibit both spasticity and athetoid movements, this is a useful sub-division. Dr Evans went on to say that the nomenclature for the classification of spastic cerebral palsy according to distribution was even more absurd. 'Starting,' he said, 'with a monoplegia, a one stroke, we go on to a half stroke, which is twice as extensive as a monoplegia; a diplegia or two stroke is actually four times as extensive as a one stroke but affects twice as many limbs as a half stroke.'

The term diplegia was introduced by Sigmund Freud[6] to describe what is, in effect, an extension of cerebral spastic paraplegia in which the spasticity is less marked in the arms than in the legs. In hemiplegia, single or double, the spasticity is worse in the arms than in the legs. The coining of the word diplegia was Sigmund Freud's only disservice to the study of cerebral palsy because he made by far the most distinguished contribution to this subject after William Little's publication and before his interests moved into psychiatry.

In the spastic cerebral palsies one or both cerebral hemispheres are damaged, whereas in the dyskinesias pathological changes are located in the basal ganglia. In cases of bilateral athetosis, the caudate nucleus and putamen are involved and involuntary movements are the distinctive clinical feature. In dystonia the cerebral pathology may be widespread but always seems to involve the globus pallidus, and the characteristic clinical findings are shifting hypertonus causing the involuntary assumption of weird postures, preservation into childhood of reflex activity normal in the newborn and very severe motor disability.

Treatment

Surgery

In cases of spastic cerebral palsy the role of orthopaedic surgery is to prevent and correct the structural deformity secondary to inequality of muscle power between opposing muscle groups; it has no role in the dyskinetic forms. In the early days of orthopaedic treatment over-enthusiasm on the part of surgeons led to over-correction and often a worsening of disabilities. Today the orthopaedic surgeons seem to have got it just about right and, for example, children with spastic limbs may be enabled to walk, or to walk in a more gainly fashion, only after tendon lengthening, fasciotomy, tenotomy, neurectomy, alteration of the origins or insertion of muscles, osteotomies and so on.

Neurosurgery, at least at present, has clearly less to offer. In some cases hemispherectomy has been effective in the treatment of fits or behaviour disorders accompanying congenital hemiplegia, and this procedure has sometimes even led to an improvement in the hemiparesis itself. As early as 1890, Sir Victor Horsley[7] reported improvement in unilateral athetosis by removal of the precentral gyrus but, although many neurological operations have been devised and used, attempts to ameliorate involuntary movements in the cerebral palsies have been disappointing and have not paralleled the successes in system diseases. In the 1890s Horsley and Sherrington[8] independently showed that stimulation of the surface of the cerebellum in the decerebrate cat consistently lessened rigidity in extensor muscles. In recent years, Dr Irving Cooper[9] has tried the effect of implanting electrodes on the surface of the cerebellum in patients with spastic paralysis but although stimulation undoubtedly does bring about relaxation, functional improvement has not been striking. I do not for one moment suggest that the combined ingenuity of neurophysiologists and neurosurgeons may not in the future bring important if expensive relief in both spastic and dyskinetic cerebral palsy.

Drugs

The use of drugs, apart from those given for the control of epilepsy, has been disappointing. A number of drugs has been tried in attempts to reduce spasticity, sometimes with apparent success, but overall improvement has not been sustained.

Other drugs have been administered for control of involuntary movements in cases of athetosis, perhaps the most successful being ethyl alcohol. However, its administration to young children was considered imprudent by most other paediatricians.

Physiotherapy

Physiotherapy of one sort or another has been the principal form of treatment offered for cerebral palsy. There is little doubt of its necessity in helping patients to obtain the best results from orthopaedic surgery. Apart from this special application, physiotherapy is designed,
on the one hand, to prevent additional deformities and, on the other, to promote functionally useful posture and movements.

Physiotherapy had certainly been introduced by the beginning of this century. By 1909 Sir Frederic Still[11] was writing with approval of the efficacy of passive exercises in the prevention of contractures and of active exercises to encourage use.

A more talented lecturer, writer and researcher than I is needed to do justice to the story of the treatment of cerebral palsy by various forms of physiotherapy in the last half century. The history would surely include character studies of those who advocated and popularised systems of treatment and those who maintained a profound scepticism. The latter were usually honest, pessimistic, conventional, unhelpful to parents and mostly neurologists. I would not have it supposed that the former necessarily or usually had the opposite of all these characteristics.

One of the difficulties inherent in a thorough study of this subject is that so much of the literature is not to be found indexed in the Cumulated Index Medicus but in the uncatalogued contents of the popular press, popular magazines and periodicals intended, sometimes rather insultingly, for a female readership. They have usually exaggerated the benefits of a new form of treatment while castigating conventional medical practice for indolence, cynicism, unhelpfulness and ignorance. And those whose work they have been propagating with enthusiasm have often not been averse to this sort of publicity. There is no doubt that parents of severely disabled children have sometimes acquired exaggerated hopes of their improvement, even false hopes of their cure, from the popular press.

In the film Bunny, shown on television six years ago to demonstrate the Doman-Delacato methods, one of the staff in Philadelphia says: 'You can land a man on the moon yet you can't teach your kid to talk; I just can't understand that'. That statement seems to me to carry the implication that if we put as much effort into teaching brain-damaged children to talk as we have into getting a man on the moon, we would be able to achieve advances hitherto considered to be impossible.

One has to realise, however, that there are certain things that are genuinely impossible, such as landing a man on the sun. So many extraordinary things are being done in medicine today, heart transplants for instance, that when doctors say they cannot do something it is sometimes interpreted by patients or parents as being a failure of will rather than of power.

I have been interested in this subject for long enough to have witnessed one 'method' taking over from another every decade or so. To some extent a new system has represented a development of that which it superseded, as in other branches of therapy, but it has also seemed to represent disconnected changes in fashion, again as in other forms of therapy.

There has, however, been a gradual move away from emphasis on individual muscles towards movements and maintenance of posture to functional activities. The links in this chain are represented by Dr Phelps of Baltimore, followed by his English disciple Mrs Eirene Collis, Dr Temple Fay of Philadelphia and his successors, Dr and Mrs Bobath in London, and Dr Peto of Budapest, the emphasis of whose treatment lies in the more direct attainment of useful activities. This list is, of course, far from complete.

The work in London of Dr and Mrs Bobath perhaps comes into a rather different category from the work of most of those mentioned, since much of it is accepted by therapists of a cautious and non-innovative character and because the Bobs have learned so much, and imparted so much, of the reflex patterns of posture and movement in early infancy and their abnormal persistence into childhood in cases of cerebral palsy.

Is physiotherapy for cerebral palsy worthwhile? The late Dr Bronson Crothers[12] wrote in 1926:

The only question is whether the possible improvement is sufficiently great to justify the long course of training. Here differences of opinion arise. On the whole I was pessimistic. On the other hand, the orthopaedists have accepted the challenge and have started to work. Here, as in every such case, the pessimist steps aside and, with due shame, hands problems to the only ones who can possibly solve them, that is, the optimists.

In fact Dr Crothers himself advocated the use of physiotherapy but remained sceptical of its benefits.

Children with cerebral palsy undoubtedly improve functionally under the care of physiotherapists. But it is only a few sceptical paediatricians such as myself who have been able to witness over the years how well some children with cerebral palsy develop without physiotherapy, always provided their parents steer the difficult path between helping too much and helping too little.

I once discussed with the mother of a child with cerebral spastic paraplegia the advisability of her making a difficult journey by public transport across London for regular attendance at a spastic centre. The child was 2½ years old and showed no signs of walking. We decided to defer a decision for three months. Three months later and much to my surprise he walked independently if clumsily into my consulting room. 'If we had sent him to the Centre you would have given them the credit', I said to his mother. 'Yes, and so would you, Dr Tizard', she replied.

Physiotherapy is, of course, often carried out in a special centre for cerebral palsy and these centres bring benefits to the child other than physiotherapy. Twenty years ago I wrote[13]:

The special centre does certainly contribute usefully to the problem. The doctors and physiotherapists involved become expert advisers in the everyday problems of the child with cerebral palsy. The child is taught somewhat earlier how to clothe himself, lace his shoes, hold a pencil, etc., partly because of expert teaching and partly because in the clinics there is less emotional stress than at home. The child benefits by
mixing with his physical equals without the sense of inferiority which competition with normal children induces. The mother gains through much needed rest, relaxation and the sharing of responsibility.

O. Henry's 'Gentle Grafter' was once accused by a jealous competitor of selling coloured sand to Southern Missouri housewives at half a dollar a teaspoonful to put into oil lamps to keep them from exploding. The Gentle Grafter admitted the accusation to be true.

'Listen', he said. 'I instruct her to keep her lamp clean and well filled. If she does that it can't burst. And with the sand in it she knows it can't, and she doesn't worry. It's a kind of Industrial Christian Science.'

Is physiotherapy the coloured sand that brings with it useful knowledge? Perhaps I have modified my views very slightly in recent years. In 1952 the late Doctors Bronson Crothers and Richmond Paine[14] and I found that a high proportion of children with congenital hemiplegia had peripheral sensory defects, a fact that had previously been overlooked. Relatively few children or adults with congenital hemiplegia showed a loss of simple sensation; the defect was of discriminatory sensation and even an intelligent child has to be between four and five years of age before stereognosis and so on can be successfully tested for. And follow-up examinations are notoriously less thorough than initial examinations. Thus, these findings had been missed and yet they were of profound importance. For instance, following transplantation of the flexor carpi ulnaris tendon in the affected hand, functional success was found only in children without discriminatory sensory loss.

Then we were well aware of the importance of bringing sensory experience to the cerebral palsied child who was relatively immobile. A normal child learns without being taught the difference between light and heavy, soft and hard, rough and smooth — simply by personal experience. So those observations were good as far as they went but they did not go far enough. Perhaps we were not fully aware of the direct role that proprioceptive experience might have on developing muscle function, and the experience of normal postures is one of the contributions of the Bobaths to the handling of cerebral palsied children.

In 1973, Professor A. Brodal, the Norwegian neuroanatomist[15], wrote a fascinating account of his own stroke at the age of 62. It was a left-sided hemiplegia without obvious sensory loss, which he attributed to an embolic lesion of a branch of the right middle cerebral artery causing an infarction of the posterior part of the right internal capsule and its surroundings. He made a number of important observations on the course of recovery from a 'central paresis'. The first concerned what he called the 'force of innervation' and he gave a vivid account of the tremendous effort of will needed to contract a muscle just capable of being actively moved. He wrote: 'The expenditure of this mental energy is very exhausting, a fact of some importance in physiotherapeutic treatment'. This has a very obvious parallel in the treatment of children with cerebral palsy. But more relevant to sensory experience are his observations on the value of passive movements in training and on what he calls passive sense. He said that he would often find it impossible to perform a certain movement until the physiotherapist had made it passively a couple of times. 'Subjectively', he wrote, 'it was clearly felt as if the sensory information produced by the passive movement helped the patient to "direct" the "force of innervation" through the proper channels'. This added weight to the evidence he quoted on a debated subject, namely that there is a muscle sense and that impulses arising in primary sensory afferents from muscle spindles evoke potentials in the sensorimotor cortex, and that these sensations are involved in motor control.

With most forms of treatment it is possible to carry out properly controlled trials. I believe this to be impossible in the case of the cerebral palsies, partly because of the infinite variety of motor defect, sensory defect, intelligence, spirit and parental attitudes, and partly because of the 'coloured sand' effect to which I have already referred. Few controlled trials relating to the effect of physiotherapy on measurable defects in cerebral palsied children have been carried out. Five years ago Wright and Nicholson[16] published their findings of a study of physiotherapy on 47 spastic children under six years of age selected at random for either treatment or a control group. The study provided no evidence that physiotherapy affected the range of dorsiflexion of the ankle or abduction of the hip, nor that it had any effect on the retention or loss of primary automatisms. Inevitably the study was criticised on methodological grounds. In a letter to appease the anger of his opponents Dr Trevor Wright[17] wrote that he had 'a strong impression that the treated children and their parents were much happier and better adjusted to the handicap'.

Is physiotherapy worthwhile? Perhaps the answer to my question is a modified 'yes', but I can commend a thoroughly sensible and well-informed book Physiotherapy in Paediatric Practice by two London physiotherapists, Mr David Scrutton and Miss Moyra Gilbertson[18].

The diagram facing the title page of their book is intended to apply to all out-patient treatment but is perhaps particularly applicable to cerebral palsy. Does he need physiotherapy? Are the aims realistic? Will the time needed for treatment and travel cause him to miss something else of value? Will it seriously disrupt family life? All these are considerations that should be taken into account on making a decision about physiotherapy for a cerebral palsied child.

One final point about treatment. My considered opinion is that screening in infancy for cerebral palsy is not, in general, conducive to human health and happiness. Lest there be any misunderstanding let me say clearly that parents who are concerned about a baby's development should receive the most expert advice available and as quickly as possible. But actively to look for cases causes more parental misery when false doubt exists than benefit when diagnosis is certain. One effect of early diagnosis is falsely to enhance the reputation of treatment. Even those paediatricians very familiar with the neurological characteristics of very young infants can
make mistakes. I have seen several infants in whom I had
confidently, but secretly, diagnosed spastic paraplegia
and one in whom I had diagnosed athetosis turn out to be
normal children without the benefits of physiotherapy.

Prevention

In the case of cerebral palsy—humanitarian con-
siderations apart—the cost of keeping a victim in
reasonable comfort during his now not much shortened
life-span has been estimated at a quarter of a million
pounds. I believe that at least partially successful
preventive measures would be much cheaper.

The cerebral palsies are now being prevented, or
rather their incidence has fallen in parts of two Scan-
dinavian countries which have, on the one hand, high
standards of living and, on the other, relatively static
populations and accurate means of ascertainment.

Dr Paul Glentig[19] managed to identify all cases of
cerebral palsy in children born over a 20-year period—
1950-1969—in all Denmark east of the Little Belt. It is
probable that ascertainment was not complete in the first
5-year period, but since then there has been a steady fall
in total cases and in each subdivision of spastic, dyskinetic
and other cases. Professor Bengt Hagberg[20] traced all
cases of cerebral palsy in children born in the Gothenberg
region of Sweden (population 1.8 million), again over a
20-year period; there has been an equally remarkable fall
in incidence from over 2.2 to just over 1.4 per thousand
live births. It has frequently been said in the past that a
reduction in perinatal deaths would lead to an increase in
the numbers of chronically handicapped children. This
is, of course, untrue as a generalisation, as shown in Fig.
2 of Professor Hagberg, which shows the remarkable fall

in perinatal mortality, infant mortality and stillbirths
over a 25-year period accompanied by a decline in the
incidence of cerebral palsy. We are now trying to find out
what is the incidence of cerebral palsy in Oxfordshire.

In general, to prevent something happening, one has
first to know why and how it happens. The only form of
cerebral palsy in which there is a clear-cut aetiology and
pathogenesis is choreoathetosis, usually accompanied by
nerve deafness but not by mental defect, as a con-
sequence of hyperbilirubinaemia in the newborn.
However, the condition of kernicterus has gradually
disappeared following the introduction of exchange
transfusion by Louis Diamond in 1946[21], and the
commonest cause of hyperbilirubinaemia, haemolytic
disease, is itself rapidly disappearing following the work
of Sir Cyril Clarke and his colleagues.

There are two other forms of cerebral palsy which,
lacking the clear-cut aetiology and pathogenesis of
choreoathetosis, nevertheless have reasonably clear
associations. These are cerebral spastic paraplegia and
bilateral athetosis.

Spastic paraplegia has for long been associated with
premature birth. Sigmund Freud pointed out that this
deduction could be made from William Little’s 47 case
histories[22], although Little himself does not appear to
have appreciated the point.

Table 1 shows the incidence of cerebral diplegia in
babies of very low birth weight in a number of different
follow-up series[23-27]. There is a gradual fall from 32
per cent in Denver to between 3 per cent and 4 per cent
over a ten-year period at Hammersmith Hospital. Dr
Pamela Davies and I were unable to determine with
certainty what was the cause of the highly significant fall
in cases over our earlier four-year and later six-year
period. Five of our six diplegic babies in the earlier period
had not been seriously ill after birth and we supported
the suggestion, made earlier by John Churchill and his
colleagues from Bethesda[28], that they may have sur-
vived intraventricular haemorrhage.

Intraventricular haemorrhage is a very common cause
of death in very low birth-weight babies. Until recently,
there has not been a method of detecting the condition
with absolute certainty in life but the use of computerised
axial tomography has now made this possible and it has
become clear that minor degrees of intraventricular
haemorrhage, compatible with survival, are much
commoner than was hitherto supposed. These
haemorrhages are likely, through obstruction, to cause
dilatation of the lateral ventricles, and dilatation would
be most likely to damage the motor neurones innervating
the legs, since those innervating the arms enter the in-
ternal capsule more directly. The cause of in-
traventricular haemorrhage is not fully explained but
large changes in cerebral blood flow may be crucial.
Professor Kenneth Cross’s [29] ingenuity has supplied us
with the means of measuring cerebral blood flow in the
newborn baby non-invasively. The baby’s head is so
compliant that it can be used as its own plethysmograph
and a strain gauge can be used to measure the increase in
circumference when the jugular veins are compressed for
two seconds. My colleague Dr Richard Cooke[30] has

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Fig. 2. Rates of perinatal and infant mortality, stillbirths
and cerebral palsy in Sweden, 1951-75[38].
shown that during apnoeic attacks, known to be associated with cerebral palsy, aetiology and pathogenesis are even less clear than in the situations I have already described. To discuss these in detail would necessarily mean giving a resumé of almost the whole of what is known about fetal physiology, obstetric practice and the care of the newborn baby.

My omission of the pathology, the morbid anatomy, of the cerebral palsy is not entirely due to personal ignorance. The baby whose brain has sustained damage of a nature to cause motor defect is most likely to die at or soon after birth, times when one cannot predict with accuracy the ultimate effects of cerebral damage. Post-mortem examinations on children with established cerebral palsy are not too commonly carried out and, by the time they die, secondary changes such as gliosis, cyst formation and perhaps transsynaptic degeneration may have taken place. Thus, the nature of the original damage, viral infection, ischaemia, hypoglycaemia, hypoxia, acidemia etc. is most unlikely to be revealed by morbid anatomy. Nevertheless, certain deductions may reasonably be made. In the case of the hemiplegias, damage is usually confined to the contralateral hemisphere and in a majority of cases takes the form of a cyst in the distribution of the middle cerebral artery, sometimes possibly the result of an occlusion in fetal life. The quadriplegias, spastic or dystonic, show cerebral changes more characteristic of ischaemia than anoxia.

It has been well known for a long time, indeed it is obvious that it must be the case, that newborn babies and their brains have remarkable powers of resistance to hypoxaemia. The first experimental evidence of this resistance was provided long before oxygen was discovered, by Robert Boyle[34].

It was suggested some years ago by Dr K. C. Dixon[35] of Cambridge that the newborn's resistance to hypoxaemia might in part be accounted for by the relatively small surface area of neurones at birth, since dendritic development was far from complete. Dr Purpura[36] showed that the major dendritic branching of pyramidal neurones takes place between 28 and 35 weeks of fetal life. But dendrites lengthen in the first post-natal year and there is probably a marked increase in the number of synapses, with their high oxygen consumption, after birth. In addition, Dr Dixon's idea may provide an explanation for the clinical impression that the brains of very immature babies are less likely to be damaged by acute asphyxia than are the brains of term babies. Hopkins, working with Doctors Farkas-Bargeton and Larroche[37], has recently shown that necrosis in the cerebral cortex in cases of stillbirth or neonatal death seldom predominates in those layers in which oxidative enzyme activity is highest and that the

Table 1. Spastic diplegia in survivors of very low birthweight.

| Authors                  | Year of birth | Sample number | Spastic diplegia % |
|--------------------------|---------------|---------------|--------------------|
| Lubchenko et al. (1963)  | 1947-1950     | 133           | 32                 |
| Drilleen (1964)          | 1948-1960     | 91            | 20                 |
| McDonald (1967)          | 1951-1953     | 560           | 7                  |
| Wright et al. (1972)     | 1952-1956     | 65            | 9                  |
| Davies and Tizard (1975) | 1961-1964     | 58            | 10                 |
| Davies and Tizard (1975) | 1965-1970     | 107           | 0                  |

At one time it was thought to be wrong to attempt to resuscitate an apparently stillborn baby even though the fetal heart had been heard shortly before delivery. Dr Hilary Scott[31] studied the outcome of severe birth asphyxia in babies born over a seven-year period in the Hammersmith Hospital. There were 48 babies in all: no less than 15 of these were apparently fresh stillbirths, while the remaining 33 did not breathe spontaneously until more than 25 minutes after delivery. All were resuscitated, at least temporarily, and all were treated promptly with alkalis which, as Professor Dawes, Dr Windle and their colleagues[32] have shown, will protect against brain damage in experimental asphyxia of newborn Rhesus monkeys. Half of these babies died within a few hours or days of birth, but of the 23 long-term survivors all but 6 have escaped severe brain damage. The outcome was less favourable in terms of the incidence and severity of cerebral palsy in those asphyxiated babies in whom there had been evidence of fetal asphyxia. This is one of the problems encountered in trying to determine the timing of brain damage because the very babies who are most likely to suffer birth asphyxia are also the very babies who have suffered from chronic growth failure and, presumably, an inadequate supply of nutrients in utero. Dr R. E. Myers[33] has shown in his experiments on Rhesus monkeys that chronic or recurrent fetal asphyxia produces quite a different form of brain damage from that caused by acute asphyxia. However, in the human being, acute asphyxia is often associated with a chronic state that may also have damaged the fetal brain.

In the remaining categories of cerebral palsy, aetiology and pathogenesis are even less clear than in the situations I have already described. To discuss these in detail would necessarily mean giving a resumé of almost the whole of
distribution rather suggests impairment of delivery of other essential metabolites, besides oxygen, which might be a consequence of local or general vascular hypotension. It is relevant to this concept that there is an association between cerebral palsy and intrauterine growth failure. Hagberg has shown that low birth weight is associated with cerebral palsy, especially when the birth weight is low for the estimated length of gestation.

Studies of intrauterine growth failure are a growth industry in medical research. A very great deal has been learned in the last 20 years, including, of course, the effect of maternal smoking, but accumulated knowledge has not reached the point of accurate prediction, invariably reliable detection, or effective treatment.

What can be done now in the UK to reduce the incidence of the cerebral palsy? There is certainly room for improvement in our maternity services, although these are, in spite of their detractors, on the whole very good. We could make better efforts to ensure early attendance at antenatal clinics. Also, while obstetricians are becoming rapidly more skilful in detecting the abnormal pregnancy and especially the small-for-dates baby, the knowledge that has been accumulating so rapidly in the last decade or two is not always acted upon. Thirdly, confinement in the case of abnormal pregnancies should take place in large central maternity hospitals in which there are the facilities for fetal monitoring and a neonatal intensive care unit. It is not just a question of economising on equipment and staff, but rather that of concentration of experience which is so necessary if advances are to be made. I am well aware of the logistic and personal difficulties involved.

Finally, and in connection with these practical difficulties, I want to put the opposite point of view. Perinatal death or chronic disablement in survivors can to some extent be looked upon as accidents, and we must always ask if the price we pay in preventing accidents may sometimes be too high for the great majority who, in any case, escape accidents. Opponents of such a view usually argue from the Book of Ecclesiastes that it is better to be a live dog than a dead lion. But we should really be asking ourselves if it is better to have 10,000 live dogs or 9,999 live lions and one dead one.

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