force caused considerable improvement in the hip joint, but its normal function as an internal rotator can easily be restored by means of a rotation and/or varus osteotomy which will replace the insertion of psoas lateral to the axis of rotation. This will have the effect of pulling the head of the femur back into the acetabulum away from the anterior capsule and restoring concentric movement after which the acetabulum will once more develop normally. The result of such a procedure is seen in Figs 9 and 10.

The question must then be asked: when does eccentric movement develop in a hip joint? It can develop at any time. At birth there may be a persistent mild subluxation which is nothing more than eccentric movement of the head of the femur within the acetabulum which will result in progressive deterioration. It may on the other hand be that a hip will develop normally until the age of 10 as in Fig 14A. It may then develop a very mild degree of eccentric movement (Fig 14B) which in the course of the next five years (Fig 14C) will have produced extensive deterioration in the hip joint which must inevitably lead to arthritis. It seems highly probable in fact that eccentricity of movement can develop in a hip joint at any age, and that the development of such movement in hips at the age of 45, 55, or perhaps more may well be the beginnings of an unexplained arthritis. It is also likely that this mechanism can be applied to almost any other joint, and that the development of so-called primary arthritis is simply an indication that an abnormal form of movement has developed where previously the type of movement was normal.

It is also possible that if we could detect the development of this abnormal type of movement early we might well be able to correct it and thereby allow the hip or other joint to develop normally once more.

Summary

The problem of growth and the developing hip joint has been discussed with particular reference to congenital dislocation of the hip, and there seems to be increasing evidence that its normal development depends far more on its physiological function than on its anatomical configuration. Our object of treatment need not be the development of instant anatomical perfection because even the best hip will deteriorate if the movement of the joint is in some way abnormal, whereas conversely even an apparently unsatisfactory hip will progressively improve if a normal type of movement has been established.

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Short Papers

Congenital Postural Deformities: Further Perinatal Associations

by Peter Dunn MD MRCP
(*Department of Child Health, Southmead Hospital, Southmead Road, Bristol, BS10 5NB*)

In 1972 I attempted to show that congenital postural abnormalities were a group of anomalies of the musculoskeletal system caused by intrauterine moulding of a previously normally formed part (Dunn 1972). While the pressure required to produce such moulding might occasionally arise intrinsically (due to muscle imbalance secondary to neuromuscular disease or malformation), in most cases it was of extrinsic origin – arising in the later weeks of pregnancy as the fetus grew larger within its limited container, and the volume of protecting amniotic fluid decreased. All parts of the infant might be affected including skull, face, jaw, thorax, spine, and limbs, the most important deformities from the medical viewpoint being those of the feet, congenital dislocation of the hips, congenital postural scoliosis and congenital sternomastoid torticollis. In an epidemiological study involving 6756 newborn infants born in Birmingham, it was possible to show that the various deformities occurred in association with each other to a statistically highly significant degree. They also occurred in association with two groups of teratological malformations: infants with spina bifida, when the deformations were found to be confined to the lower limbs; and infants with anomalies of the urinary tract causing fetal anuria and hence oligohydramnios, when all parts of the body were liable to be deformed. Similar deformities were found in association with chronic oligohydramnios secondary to prolonged drainage of amniotic fluid. In contrast, postural deformation was not found to occur in the presence of maternal hydramnios. I ended my 1972 paper by mentioning three other prenatal factors which, in addition to pressure, influence the incidence of congenital deformation, namely rate of fetal growth, fetal plasticity and fetal mobility – and by saying:

'All these factors are, of course, themselves directly or indirectly under the influence of heredity and are involved in a dynamic interplay throughout fœtal life. Nature plays her hand to the limit. The price we

pay for a larger and more mature infant at birth, better able to withstand the stresses of extrauterine life, is a 2% incidence of congenital postural deformities. Perhaps we ought rather to marvel at the fact that 98% of infants are not deformed at birth and that 90% of those that are will correct themselves spontaneously after birth; with early postural assistance this last figure may be brought near to 100%.'

Anthropology provides many examples of postnatal deformation. For example, the curious shape of the skull of the Chinook Indians used to be achieved by splinting the head between boards during infancy (Jones 1968), while the Chinese were accustomed to bind the feet of their little girls in order to produce a crippling deformity that was regarded as beautiful. Both recognized the importance of applying constant pressure during infancy while the parts were still relatively plastic and growing rapidly. In such circumstances quite gentle forces, if persistently applied, will lead to deformation. If this is true for the infant, then the fetus must be much more vulnerable because of its far greater plasticity and much more rapid rate of growth. In addition, especially near term, the fetus is closely confined within a very small container and subjected to the tone of both uterine and abdominal wall muscles (Fig 1A).

In postnatal life a 5-year-old child requires no less than six years to double its weight. In contrast, a fetus of 28 weeks gestation takes only 6 weeks, while an embryo of 8 weeks gestation takes only 6 days. Thus if the rate of growth was the only factor determining deformation, the fetus might be described as 52 and the embryo 365 times more vulnerable than the 5-year-old child. Using similar criteria it can be said that 90% of all growth between conception and adult life has taken place by the time of birth at term.

The importance of fetal plasticity, which depends so much on the rigidity of the skeleton, also needs to be emphasized. During early pregnancy when the form and size of the fetus is rapidly changing, a rigid well-developed skeleton would be an embarrassment. Most of the calcification of the fetal skeleton is laid down during the second half of pregnancy. While the skeleton is barely visible on X-ray at 20 weeks gestation, it becomes increasingly radio-opaque in the succeeding months, though even at term a considerable portion, and this particularly applies to the growing ends of the bones, remains unossified and much more plastic than in later life. Thus by reason of both its slowing rate of growth and diminishing plasticity the fetus becomes increasingly able to resist deformation as term approaches. However, in girls another factor obtrudes: their pelvic ligaments, like those of their mothers, are relaxed and softened by the hormone aptly named relaxin, whose prime function is to prepare the birth canal for parturition. Girls' hips are thus more vulnerable to dislocating pressure *in utero* and are more often found to be dislocated or dislocatable at birth.

In the early months of pregnancy when the fetus is fragile and growing rapidly, it is easily deformed. Fortunately at this time it is completely protected from the pressure of the uterine wall by the presence of amniotic fluid which also permits the fetus to exercise freely and change its position (Fig 1B). However, as pregnancy progresses the fetus gets larger and, because there is a limit to the space available within the mother's abdomen, eventually this enlargement can only take place at the expense of the amniotic fluid. While the fetus usually continues to grow right up to term, the volume of amniotic fluid stops increasing in the middle of the last trimester and steadily decreases from about 37 weeks gestation onwards; the ratio of amniotic fluid volume to fetal volume falls steadily from a value of 3 at 12 weeks gestation to close to zero at 42 weeks. Thus the fetus becomes increasingly exposed to extrinsic pressure while at the same time becoming more and more able to resist deformation.

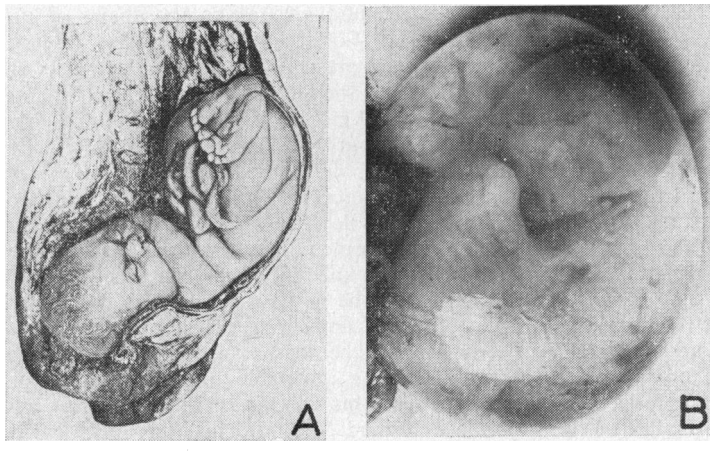

Fig 1 A, *frozen section of abdomen of pregnant woman who died during labour (from Edgar 1904). Note fetus flexed laterally round sacral promontory.* B, *fetus of 13 weeks gestation lying within amniotic sac removed at hysterotomy. The amniotic fluid completely protects the fragile fetus from pressure*

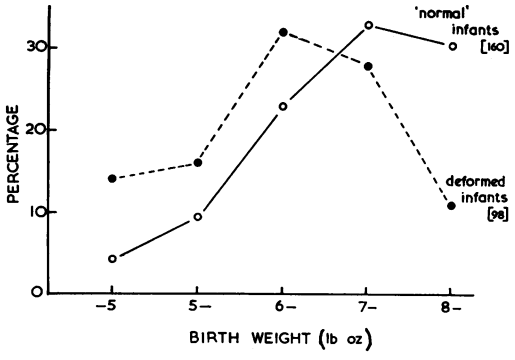

Fig 2 *Birthweight distribution for 98 infants with congenital postural deformities born at the Birmingham Maternity Hospital during 1960–61, compared with a random sample of 160 normal infants (Dunn 1969)*

The terminal fall in amniotic fluid volume is thought to be related, at any rate in part, to ageing of the placenta. Certainly an association can be shown between oligohydramnios at and even well before term and a number of conditions that tend to impair the efficiency of the uteroplacental circulation and placental function. Elliott & Inman (1961) measured the volume of amniotic fluid by a dye dilution technique and demonstrated an association between maternal hypertension and oligohydramnios. This was of great interest to me, as in a study made in Birmingham in 1960–61 (Dunn 1969) it was possible to show that hypertension complicated the pregnancies of mothers giving birth to an infant with a postural deformity twice as frequently as it did those having a normally formed child ($P<0.001$).

Although exact measurements of the volume of amniotic fluids are rarely available, oligohydramnios may be readily diagnosed both clinically and radiologically during pregnancy and also directly observed at Cæsarean delivery and at the time the membranes rupture. During the last fifteen years it has been possible on very many occasions to document the association between oligohydramnios and deformation at birth. It has also been possible again and again to observe the close correspondence between the posture of the fetus before delivery (as determined by X-ray), and that assumed by the baby after birth, the 'position of comfort' as it has been termed. Moreover, as such an infant assumes once more its prenatal posture, it can usually be clearly seen that the moulded deformities reflect the shape of the uterine cavity within which the fetus has been constrained. Perhaps, though, the most convincing evidence emphasizing the significance of oligohydramnios is that to which reference has been made earlier, namely, the common occurrence of deformities among infants born to mothers with chronic leakage of amniotic fluid, and also among those having anomalies of the urinary tract capable of causing fetal anuria, and hence oligohydramnios.

If there is an association between oligohydramnios and placental insufficiency, then one might anticipate that infants with congenital postural deformities might tend to be malnourished and small-for-dates at birth. This was indeed found to be the case in the Birmingham study (Dunn 1969). The birthweight groups of the 98 deformed infants (excluding infants with malformation) observed in this study and of a control sample of 160 normal infants are shown in Fig 2. There is clearly a marked deficit of heavier infants among those that were deformed. When gestational age was also taken into account it was possible to confirm that these infants tended to be small-for-dates ($P<0.001$). In addition, 14% of the deformed infants had exhibited signs of fetal distress and 12% appeared malnourished at birth.

If we recognize that most fetuses are exposed to extrinsic pressure during the later weeks of pregnancy, why should only 2% have signs of moulding deformity at birth? The explanation probably lies in the strength of the legs of the fetus during the later weeks of pregnancy which enables it to kick and change its position, and hence to alter the direction along which potentially deforming extrinsic forces may be acting. Certainly, conditions that impair or deprive the fetus of its ability to move may be shown to have a highly significant association with congenital deformation. The ability to kick may be impaired by malformations of the lower limbs such as phocomelia and congenital amputation, or by paralysis secondary to spina bifida or other neuromuscular disease, or the legs may be weak either because of prematurity or from disuse atrophy. Alternatively the legs may be trapped in a position of mechanical disadvantage or have no room to move because of oligohydramnios or an abnormally shaped amniotic cavity.

Whether the legs of the fetus are flexed or extended at the knees affects both its ability to kick *in utero* and also alters the shape of its folded form. For these reasons, leg folding influences fetal presentation in the later weeks of gestation. Thus the fetus with flexed legs (Fig 3A) is in a powerful kicking position and will be able to turn itself until its head comes to lie in the smaller pelvic pole of the uterus, while the legs continue to exercise themselves in the broader elastic upper pole. These infants are very much less often deformed than those whose legs are extended at the knee (Fig 3B). The breech of such infants forms the narrow rather than the broad pole of the fetus and slips readily into the mother's pelvis leaving the legs extended alongside the body and quite incapable of either flexing or kicking. In this predicament (in wrestlers' parlance termed 'the folding body press') the

fetus may remain for several weeks growing, but immobile, and steadily being moulded by the gentle pressures surrounding it. Not surprisingly infants presenting by the breech are approximately ten times more likely to be deformed than those presenting by the vertex. In the Birmingham study, 32% of deformed infants presented by the breech ($P<0.001$), the incidence of breech delivery for each of the main individual deformities being mandibular asymmetry 22%, contracture of the sternomastoid muscle 20%, postural scoliosis 42%, dislocation of the hip 50%, and talipes 22% (Dunn 1969).

Another way in which the legs of the fetus may be folded that puts them at great mechanical disadvantage from the point of view of kicking is for them to be crossed, usually at the level of the ankles. Once again the fetus may be rendered immobile and then exposed to deformation. The feet are particularly vulnerable to deformation in this position and readily become moulded into the club-foot position. Usually both feet are affected, though characteristically the outer foot is more severely deformed and has the typical pressure atrophy and skin dimpling over external malleolus and outer border (Dunn 1972).

Legs that have been trapped *in utero* commonly show impaired power and movement for days or even weeks after birth. This is not surprising, for immobility and compression for any length of time may be expected to lead to neural ischæmia, impaired muscle nutrition and development, and finally to weakness and even atrophy.

Another important factor in the etiology of prenatal deformation is the shape of the amniotic cavity within which the fetus lies. In part this will be determined by the shape of the uterus itself, which not only varies from person to person like any other part of the body, but may also be distorted by malformation such as, for example, unicornuate uterus. The shape of the amniotic cavity will obviously also be influenced by the volume of liquor amnii and the size and shape of the fetus. The latter will depend in part on the way it is folded as well as on its presentation and orientation within the uterus. Additional factors include the presence of more than one fetus, the site of placental implantation, the presence of uterine fibroids or other tumours, and the shape of the abdominal cavity within which the uterus lies. Here the pelvis, sacral promontory and neighbouring abdominal organs all exert an influence.

Clinical experience has provided many illustrations of the significance of these factors. One example must suffice. It has been possible to explain in terms of fetal position *in utero* the long recognized observation that congenital dislocation of the hip is twice as common on the left side as it is on the right. Some years ago it was shown that the fetus tended to lie with its back towards the mother's left side nearly twice as often as towards her right ($P<0.01$). Further, it was demonstrated that the leg of the fetus lying posteriorly was more likely to be dislocated than that lying anteriorly, whether the presentation was vertex or breech ($P<0.01$) (Dunn 1969). The probable explanation is that, because of the presence of the mother's spine, the posterior leg of the fetus is more likely to be adducted and hence to lie in a position in which the hip-joint is more exposed to dislocation.

A further factor having a profound effect on the shape of the amniotic cavity is the degree of tightness of the mother's abdominal muscles. When these have not been stretched by previous pregnancies, their increased tone tends to compress the uterus backwards against the spine (Fig 4). As a result the uterine cavity tends to become flattened in its anterior-posterior diameter. This means that, for any given volume of amniotic fluid, the fetus will have relatively less room to move and therefore will be more exposed to pressure. In the Birmingham study it was shown that first-born infants were considerably more often found to be deformed at birth than were those born to multiparæ (54% as against 35%; $P<0.001$). This observation held true for each of the main individual deformities; mandi-

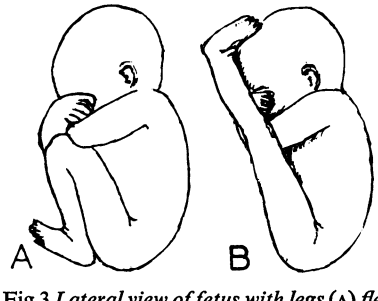

Fig 3 *Lateral view of fetus with legs* (A) *flexed at knees, the normal posture, or* (B) *extended with feet alongside face. Diameter of 'breech' is greater than that of vertex when legs are flexed, smaller when they are extended*

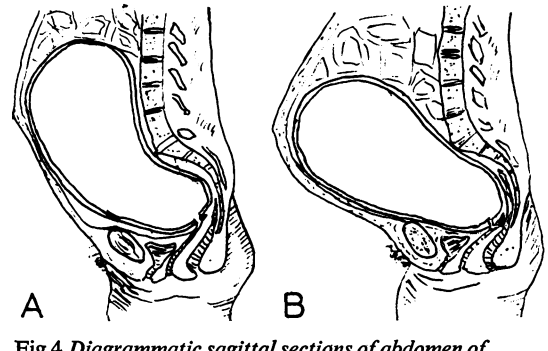

Fig 4 *Diagrammatic sagittal sections of abdomen of* (A) *primigravida and* (B) *multigravida, to illustrate influence that unstretched abdominal wall muscles may have on shape of uterine cavity during later weeks*

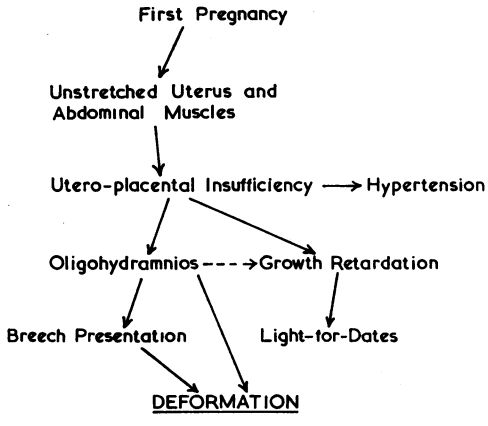

Fig 5 *Interrelation of some of the pregnancy associations with postural deformation at birth*

bular asymmetry 55% firstborn, sternomastoid contracture 53%, postural scoliosis 42%, dislocation of hip 56%, talipes 62% (Dunn 1969).

In extrauterine pregnancies the amniotic sac may lie right outside the uterus. In such a circumstance the fetus is especially exposed to pressure, and judging from the literature, deformation is the rule rather than the exception. In the only case I have had the opportunity to observe, the infant was found at birth to have a dislocated hip and deformities of the face and one foot.

Many interesting and important facets of this subject have been hardly mentioned or only briefly discussed, but I have tried to show that congenital postural deformities not only occur in association with each other but also share a number of pregnancy characteristics such as primigravidity, maternal hypertension, oligohydramnios, growth retardation and breech presentation (Fig 5). I have also tried to indicate the way in which intrauterine forces capable of moulding the fetus increase throughout pregnancy as the infant grows, the mother's uterus and abdominal wall are stretched, and as the volume of amniotic fluid diminishes (Fig 6, unbroken line). At the same time the ability of the fetus to resist moulding (Fig 6, broken line) also increases as the rate of fetal growth slows, the skeleton ossifies, and leg movements become more powerful. In the great majority of cases the lines may converge but do not cross and the infant is normally formed at birth. However, the margins are narrow and any of a host of factors may tip the scale (Fig 6, dotted lines) so that deformity results. Such deformation is found in about 2% of newborn infants, though the great majority correct themselves spontaneously once the infant has been removed from its adverse intrauterine environment. With early diagnosis and active postural correction this recovery rate can be brought close to 100%.

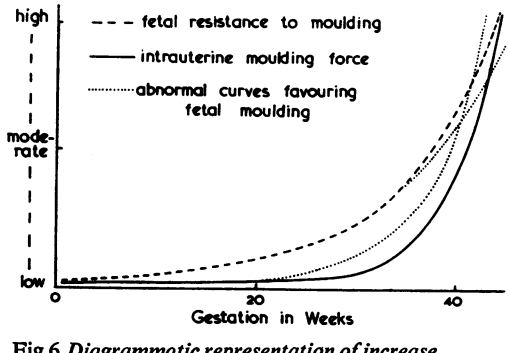

Fig 6 *Diagrammatic representation of increase throughout pregnancy of intrauterine moulding forces* (—) *and ability of fetus to resist deformation* (- - - -). *Normally the two curves do not cross, but small changes in either curve may create conditions leading to deformation* (.....)

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Acetabular Dysplasia in Congenital Dislocation of the Hip

by J Rowland Hughes MD FRCS
(*CDH Assessment Unit, Robert Jones & Agnes Hunt Orthopædic Hospital, Oswestry, Salop*)

There is a prevalent philosophy that the treatment of congenital dislocation of the hip (CDH) consists essentially in putting the femoral head into the acetabulum and that by encouraging movement and activity, the stimulus for normal joint development, nature will do the rest. If this were so, there would be few problems in the treatment of this challenging condition. In traumatic dislocation of the hip, although the capsule and ligamentum teres may be completely ruptured, as the component parts are self-locking, it requires an effort to pull them apart unless the shape of the acetabulum has been altered by fracture (Fig 1A). Dunn (1969) has shown this also to be true at birth and that, although the normal hip joint in the 13–40 week fetus increases in size and mechanical strength, it changes little in relative acetabular depth or general morpho-