century AD), while agreeing that fetal movements were associated with a normal pregnancy, doubted whether their vigor was related to the sex of the fetus. Raynalde, in his 1545 translation of Rösslin’s early sixteenth-century work Der Swangern Frauen und Hebammen Rosengarten, noted that the absence of fetal movement was a sign of intrauterine death. Paré (1634)4 taught: “By what signs it may be known whether the child be in the womb bee dead or alive. If neither the Chirurgical hand, nor the mother can perceive the infant to move…”

Inquiring about fetal movements must therefore rank as one of the first specifically diagnostic questions that doctors ever posed. But only in the past decade or so has an association been suggested between the amount of fetal movements experienced by the mother and fetal wellbeing. The usual suggestion is that the mother should record the number of movements her fetus makes over 30 minutes or one hour; if they decrease below an agreed limit the mother reports to her doctor.

Such a form of self-assessment poses several questions. Firstly, is every movement made by a healthy fetus appreciated by the mother? Sadovsky et al2 used an electromagnetic device to detect fetal movements and reported that on average mothers were aware of 87%, but the range was 64% to 100%, suggesting relatively poor perception on the part of some women. Ultrasound scanning methods showed3 that the fetus spent 10% to 20% of its time in movement—but the range was from 1% to 90%. If fetal breathing movements were included one or other form of fetal activity occupied at least 20% of the time. Two years later, however, the same workers5 considered that as little as 10% of the time spent in movement or breathing activity was compatible with fetal health. Unfortunately mothers cannot perceive fetal breathing movements, so less than 10% of any observation period may be occupied by fetal body movements; if Sadovsky and co-workers are correct and, for example, a given mother appreciates only 70% of these movements, then 7% or less of any observation period will be occupied by perceived fetal activity. In practical terms, a mother asked to note fetal movements for three periods of 30 minutes each night might experience six minutes or less of activity; her vigilance would have to be keen and her stopwatch to hand if other matters were not to cause her to miss some movements. It may not be the time spent in movement which is clinically meaningful but their number;6 nevertheless, the constraints regarding the strength of fetal movement and the degree of maternal perception would still apply.

What degree of patient compliance might be expected with a request for a daily record of fetal movements? In one report4 61 healthy women who subsequently delivered live, normal infants were asked to record the number of movements their fetuses made daily between 9 am and 9 pm from 32 weeks’ gestation to delivery. This should have yielded at least 3400 records for analysis; only 1654 were reported, suggesting that patient compliance is relatively low even in a group presumably volunteering to help.

Finally, at what lower limit of fetal activity should the clinician be alerted to intervene? The worst case would presumably be the complete absence of fetal activity over as long as 12 to 24 hours. A recent study7 of 67 patients who had not experienced fetal movements for at least 12 hours found that 55 of them subsequently experienced fetal activity and delivered live, normal infants one to 10 weeks later. The other 12 patients had either meconium in the amniotic fluid or an abnormal unstressed or oxytocin-stressed cardiocogram and were delivered of live babies within 24 hours. Even this result begs the question in some degree—if left in utero these infants would possibly still have survived; one had a five-minute Apgar score of five and another of six, but the remaining 10 infants had a score of eight or nine. Conversely, Payne et al8 have recently described a fetal death at 34 weeks with no preceding change in fetal movements.

A mother and her fetus are always together and any simple, daily observations she could make that had clinical importance would obviously be extremely useful. Even so, the practical utility of recording fetal activity seems doubtful.

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Care of children in chronic renal failure

Twenty-six children were known to be under treatment for end-stage renal failure in Europe in 1968. By 31 December 1979 this number had increased to 1469—those still alive out of a total of 2175 patients registered by the European Dialysis and Transplant Association. Some 261 of the survivors were being treated in the United Kingdom, a rate of 19-9 patients per million children. Six of the 28 countries contributing to the European Dialysis and Transplant Association register were treating more than this, with Switzerland the leader at 28-5 per million. The difference is almost certainly the result of a rather slow British start in the early 1970s, since new patients are being taken on at remarkably similar rates in the six countries leading the table (France, Italy, Netherlands, Switzerland, United Kingdom, and West Germany)—around three or four patients per million. (The remarkable figure of 9-7 per million achieved by Israel is thought to reflect the transfer of children from other countries for treatment.) As younger and younger children are being treated, the rate is expected to stabilise at about five or six per million, leading at equilibrium to a constant population of around 800 to 900 children under the age of 15 being treated at any one time in the United Kingdom.

Sick children require specialised paediatric care if their special needs are to be met, and in 1974 the British Paediatric Association endorsed a report9 by the British Association for Paediatric Nephrology recommending that dialysis and transplantation facilities for children should be provided in a...
few regional or supraregional units, where the necessary skill could be concentrated. The report suggested 12 such centres: nine in England, two in Scotland, and one in Northern Ireland. These recommendations are in close agreement with those of the European Dialysis and Transplant Association committee, which has defined a "specialised paediatric centre" as one which fulfils six criteria: the children are in the direct care of a paediatrician, in a child's ward, and have access to a hospital school, a paediatric social worker, a dietitian with experience in paediatrics and renal medicine, and a child psychiatrist or psychologist. Substantially fewer than half of all children dialysed or given transplants in Europe in 1979 were treated in such centres.

How important is it for an individual child to be treated in a specialist centre? Does it really matter? Analysis of the 1978 European data suggests that it does. Results were distinctly better in patients treated in specialised centres than those treated elsewhere: this applied to patient survival, both on hospital dialysis and after transplantation, and to cadaveric graft survival with a difference between 68% and 56%, at two years. The annual death rate on hospital haemodialysis was 34 per 1000 children in specialised centres and 92 per 1000 in non-specialised centres; the excess mortality was due mainly to hyperkalaemia, cerebrovascular accidents, and cachexia.

In 1979 the British Association for Paediatric Nephrology produced a follow-up report as a supplement to its original recommendations. Considerable progress has been made: of the original 12 suggested centres (since increased to 13), 10 had appointed at least one consultant paediatric nephrologist, though only five satisfied the European Dialysis and Transplant Association criteria for a specialist paediatric centre. The report praised the high level of co-operation between adult and paediatric services but regretted that in certain parts of Britain "services for children remain embryonic rather than formative." This is reflected in the fact that (again in 1979) 34 centres were treating at least one child but only three were treating 10 or more. Only a relatively small investment of staff and other resources is necessary for the recommendations to be carried out in full: such expenditure is justified on the grounds that no accident of geography should deprive a child in chronic renal failure of access to the best available treatment.


Regular Review

Prostaglandins in vascular disease: a seminal approach

J R A MITCHELL

Despite the universal agreement that occlusive vascular disease is the main cause of death and disability in most developed countries, there is no such agreement about its nature, causation, prevention, and treatment. Even on the venous side of the circulation, where thrombi form in vessels whose walls are normal, we can offer only a lame theory, wrongly attributed to Virchow, to explain why thrombi form at a particular site, at a particular time, and from particular blood constituents. As soon as we move to the arterial tree the difficulties increase, because disease of the wall (atheroma) and events in the lumen (thrombosis) invariably coexist, and this has led to a century of still-unresolved controversy about the interrelation of the two processes.

The atherogenic school—The traditional view, championed by Virchow, was that atheroma was an intrinsic disease of the vessel wall. He believed it to be inflammatory (endarteritis deformans), but subsequent workers focused on the lipid rather than the inflammatory component of plaques, suggesting that atheroma was caused by insudation and infiltration of plasma lipids into the wall. This unsubstantiated hypothesis led to the view that atheroma must be caused by abnormalities of the plasma lipids and that it could be prevented or made to regress by alterations in dietary lipids. Even within the atherogenic school, alternative hypotheses to the original inflammatory and the subsequent lipid-infiltration theory have been passionately espoused: that mechanical wear and tear, accentuated by hypertension, disrupted the fabric of the artery, or that the plaques were monoclonal tumours akin to uterine fibroids. All these views shared common ground in that they either ignored the existence of thrombosis or they relegated it to the role of a mere vassal obeying the will of the diseased wall. Thrombosis was thus regarded as a secondary consequence of the plaques, which instructed the hitherto normal passing blood to adhere to them.

The proponents of this "atheroma-is-all" school invariably describe clinical events such as stroke, myocardial infarction, and gangrene of a limb as "atherosclerotic diseases," and meetings may spend several days discussing the cause of these clinical conditions without the word thrombosis being mentioned. This intellectual blind spot, which is particularly prevalent in the United States, has led to attempts to prevent or modify the clinical endpoints such as stroke or myocardial