

forceps delivery should compare so unfavourably with spontaneous vaginal delivery. In no sense was the Manchester study a controlled trial, but the report cannot be discounted or the results attributed to inexperienced operators. The pulling and turning associated with rotational forceps delivery must be a cause for concern. Chiswick and James attribute the transient neurological symptoms to cerebral oedema, but there are more sinister possibilities. Yates<sup>4-6</sup> has described the injuries that may occur to the cervical portions of the vertebral arteries—two of the principal arteries supplying the brain—when the baby's neck is pulled or twisted during delivery. His purpose was to draw attention to traumatic lesions in the necks of infants delivered by various methods and to underline the part they possibly played in the production of brain damage in those infants who survived. Short of killing the child, these injuries to the vertebral arteries could, in his view, leave ischaemic damage in the brain stem or cerebral hemispheres that might in later years be associated with spasticity, deafness, or epilepsy. In general, Yates found that the injuries to the vertebral arteries were most severe after breech delivery, but the pulling and twisting of the baby's neck that take place during rotational forceps delivery are comparable.

The concept of a "continuum of reproductive casualty" proposed by Lilienfeld and Pasamanick<sup>7</sup> raises the worrying possibility that survivors of any form of difficult delivery are more liable to suffer some form of brain damage. In extreme examples, cause and effect seem to be clearly established; but this is a difficult, complex problem and the obvious conclusions may not be the correct ones. The subtle interplay of social and medical factors that contribute to the long-term outcome for survivors of breech delivery has been studied in Newcastle upon Tyne,<sup>8-10</sup> where the conclusion reached was that the overall picture of handicap in that community would not be altered significantly by increasing the rate of caesarean section in breech presentation. Other studies of the aetiology of handicap in Aberdeen<sup>11,12</sup> broadly support the view that though obstetric trauma may kill children at birth it seems less important than social factors in causing handicap among survivors.

In the face of a difficult delivery, the obstetrician's immediate concern must be to reduce the risk of fetal death. This may well justify an increased caesarean section rate, as envisaged by Chiswick and James. But caesarean section with the fetal head jammed firmly in mid-cavity carries risks. It can be quite difficult to disengage the head from above, and the skull or brain may be damaged; moreover, there are immediate and long-term risks for the mother which must be placed in the balance when a decision is being made between vaginal and abdominal delivery. There is nothing simple or straightforward about the decision that has to be made and very little hard evidence to help the obstetrician in his choice. At present he is guided largely by experience and by confidence in his manipulative skill. A controlled trial, especially one including long-term assessment of survivors, would be difficult to organise. Nevertheless, until this sort of information is available the obstetrician must continue to rely heavily on his clinical judgment.

<sup>7</sup> Lilienfeld, A M, and Pasamanick, B, *American Journal of Obstetrics and Gynecology*, 1955, **70**, 93.

<sup>8</sup> Russell, J K, et al, *Lancet*, 1963, **1**, 711.

<sup>9</sup> Russell, J K, et al, in *Physical Trauma as an Etiological Agent in Mental Retardation*, ed C Angle and E Bering, p 101. Bethesda, National Institutes of Health, 1970.

<sup>10</sup> Neligan, G, et al, *The Formative Years*, p 125. London, Oxford University Press, 1974.

<sup>11</sup> Fairweather, D V I, and Illsley, R, *British Journal of Preventive and Social Medicine*, 1960, **14**, 149.

<sup>12</sup> Birch, H G, et al, *Mental Subnormality in the Community. A Clinical and Epidemiologic Study*, p 200. Baltimore, Williams and Wilkins, 1970.

## Synovial biopsy in arthritis

Percutaneous synovial biopsy of the knee is little known and much underused. Yet it is safe and easily repeated and is as simple as liver or pleural biopsy, though it shares the problem of sampling errors, since only small pieces of tissue are obtained. Synovial biopsy may be performed on outpatients with little more discomfort than a routine joint aspiration. Sufficient tissue may be obtained for routine histological and immunofluorescent and electron microscopical examination, and the quality is such that open biopsy can be reserved for less accessible joints.<sup>1</sup>

Synovial biopsy is indicated in inflammatory joint disease when the cause remains in doubt—usually when only one joint is affected. If examination and culture of the synovial fluid produce unhelpful results a biopsy should distinguish conditions such as tuberculosis, other subacute infection, and villonodular synovitis, in which specific treatment is indicated, from the more common chronic inflammatory joint diseases or osteoarthritis. The early characteristic changes of rheumatoid arthritis are usually evident even in the first weeks after the clinical onset, with synovial hyperplasia and infiltration with plasma cells and lymphocytes. Biopsy may also be diagnostic in inflammatory polyarthritis when the conventional tests are unhelpful; it is particularly helpful in separating diseases such as sarcoidosis, amyloidosis, Whipple's disease, haemochromatosis, or malignancies presenting as polyarthritis from the usual rheumatic diseases.<sup>2</sup> Occasionally gross infiltration of the synovium with crystals of uric acid or calcium pyrophosphate will be found in this group, particularly if the specimen is alcohol-fixed.

Some overlap occurs in the histological appearances of biopsy specimens from the chronic forms of inflammatory arthritis, particularly in rheumatoid arthritis and psoriatic arthritis, and occasionally in Reiter's syndrome.<sup>2</sup> There is gross hyperplasia of the synovial membrane, deposition of fibrin on its surface, and infiltration with plasma cells. All these changes are more frequent and more appreciable in rheumatoid arthritis, and the presence of lymphoid follicles is usually confined to this disease. Such follicles are usually a feature of chronicity, but they may occasionally be found within three months of the clinical onset of the disease. Again, while hyperplasia of the synovial lining cells may occur in several diseases, considerable synovial hyperplasia from the usual one or two cells to a layer up to six cells thick is virtually confined to rheumatoid arthritis.<sup>3</sup> A predominantly polymorph leucocytosis in the synovium may be found early in a few patients with rheumatoid arthritis but is more often due to bacterial infection; a heavy polymorph infiltrate is highly characteristic of infection. Such an infiltrate may, however, also be found in Behçet's disease, in which it may progress to

<sup>1</sup> Parry-Jones, E, *Kielland's Forceps*. London, Butterworth, 1952.

<sup>2</sup> Chiswick, M L, and James, D K, *British Medical Journal*, 1979, **1**, 7.

<sup>3</sup> James, D K, and Chiswick, M L, *British Medical Journal*, 1979, **1**, 10.

<sup>4</sup> Yates, P O, *Archives of Disease in Childhood*, 1959, **34**, 436.

<sup>5</sup> Yates, P O, *Spastics Quarterly*, 1962, **11**, No 3, 15.

<sup>6</sup> Yates, P O, in *Physical Trauma as an Etiological Agent in Mental Retardation*, ed C Angle and E Bering, p 167. Bethesda, National Institutes of Health, 1970.

dense granulation tissue,<sup>4</sup> and in familial Mediterranean fever.<sup>5</sup> In contrast, in osteoarthritis the histological changes are mild even when quite definite synovial thickening is present—namely, some surface fibrin and minimal hyperplasia of the lining cells, often together with some proliferation of small blood vessels.

Examination of the synovial fluid can yield useful clues and should always be performed before biopsy. Specific diagnostic features include crystals (identified by polarising microscopy) and bacteria (seen on a Gram stain). The viscosity of the fluid is high in osteoarthritis and low in inflammatory joint disease. But in clinical practice disease entities may rarely be separated by performing total leucocyte and differential cell counts, and synovial biopsy specimens almost always provide better diagnostic material. Nevertheless, the interpretation of any biopsy depends on adequate clinical information. This is particularly so in the rheumatic diseases, and diagnostic information from the biopsy material is best achieved by a combined review of the biopsy specimen by both clinician and pathologist.

<sup>1</sup> Polley, H F, and Bickel, W W, *Annals of the Rheumatic Diseases*, 1951, **10**, 277.

<sup>2</sup> Schumacher, H R, and Kulka, J P, *New England Journal of Medicine*, 1972, **286**, 416.

<sup>3</sup> Goldenberg, D L, and Cohen, A S, *Medicine*, 1978, **57**, 239.

<sup>4</sup> Vernon-Roberts, B, Barnes, C G, and Revell, P A, *Annals of the Rheumatic Diseases*, 1978, **37**, 139.

<sup>5</sup> Heller, H, *et al*, *Arthritis and Rheumatism*, 1966, **9**, 1.

## Industrial anarchy in the NHS

The prime cause of the industrial anarchy that has closed hospitals this week and last—and is threatening to destroy the NHS—is that strikers risk little by their actions. Union militants have learnt that more often than not they can insist on their members being paid in full for the time they are on strike—and they sometimes demand and get additional payments for coping with the work that has piled up during the stoppage. In these circumstances the surprising fact is not the number of hospitals paralysed by strike action (p 426) but the gratifyingly large proportion in which most of the staff are working normally.

For the underlying grievances of hospital staff are genuine. Porters, cleaners, and laundry workers are among the lowest paid in the country; some groups of skilled staff such as ambulance men have been negotiating about pay anomalies for over four years without success; and neither the DHSS at the top nor the pyramid of managers below it have shown any recognition of the urgency of the pay problems of the NHS. What has become plain in recent years is that militant industrial action has usually brought immediate rewards: it has

proved the most effective way to secure the attention of Mr Ennals and the Prime Minister, and in most cases the response of NHS management has been to agree to the strikers' demands. As Dr Roger Dyson said<sup>1</sup> (in the context of the Normansfield inquiry), "so long as the NHS continues to settle its strikes in ways that are immediately favourable to the striking staff it will encourage more strikes. . . ."

As this issue of the *BMJ* went to press the outcome of the current pay dispute was still unknown. The number and frequency of stoppages, works to rule, and all-out strikes within the hospital service have, however, now become unacceptable to doctors, nurses, patients, and the public. Union platitudes about patients not suffering have become seen to be false (and doctors who themselves argue a case for limited industrial action should remember that). Patients whose admission to hospital is stopped by a strike suffer immediate anxiety and some may die as a result of delays in diagnosis or treatment. The well-intentioned conspiracy of silence that has prevented public discussion of these consequences should stop: any doctor who believes that an industrial dispute was a factor in the death of one of his patients should speak out.

Can anything be done to prevent recurrences of the present epidemic of industrial unrest in the NHS? We think the condition is treatable. Firstly, the DHSS should tell its managers that the "low profile" that it seems to have encouraged should cease. Administrators should be urged to be more aggressive in minimising the effects of strike action—and in particular they should make more use of the store of public good will by enrolling volunteers. Secondly, in order to discourage wildcat strikes a principle should be established by which pay should be forfeited for time not worked: the DHSS should, we suggest, issue a specific directive that compensation for loss of earnings should not be included in any settlement without the specific approval of the Secretary of State. Finally, however, we need to remember how many hospital staff have worked hard and long for many years for miserly pay. So long as the NHS remains subject to tight financial restraints there seems only one way for that injustice to be corrected. The NHS is overmanned (at virtually every level). Lower-paid workers can be given a reasonable rate of pay if the total NHS work force is reduced by a realistic examination of staffing establishments. If the unions are really concerned with the interests of their workers they will co-operate in such a plan.

<sup>1</sup> Dyson, R, *British Medical Journal*, 1979, **1**, 283.

### Correction

#### Tumour antigens and optimism

In the leading article on "Tumour antigens and optimism" (20 January, p 149) line 7 of the second paragraph should have read: "quantitatively. Burnett's theories of clonal selection seemed to".