[9] King, R. D., Raynes, N. V., and Tizard, J., *Patterns of Residential Care: Sociological Studies in Institutions for Handicapped Children*. London, Routledge and Kegan Paul, 1971.
[10] Seglow, J., Pringle, M. K., and Wedge, P., *Growing Up Adopted*. Windsor, National Foundation for Educational Research in England and Wales, 1972.
[11] Carr, J., *Young Children With Down's Syndrome*. London, Butterworths, 1975.

Unexplained Prolonged Fever in Children

Paediatricians and family doctors from time to time face one of the most challenging clinical problems—pyrexia of unknown origin, or P.U.O. Most such fevers are short-lasting and are probably due to viral infections; but when the fever persists diagnosis is more difficult and more important. Fourteen years ago Petersdorf and Beeson[1] suggested that P.U.O. should be defined as fever of at least three weeks, exceeding 38·3°C on several occasions, and defying diagnosis after a week of intensive hospital investigation. They claimed that most cases proved to be atypical manifestations of common diseases rather than exotic conditions. In their study of 100 cases in Boston 36% were due to infections, 19% were neoplasms, and 20% were collagen-vascular diseases. The figures in another series of 128 cases[2] were almost identical. These studies mainly concerned adults; when Pizzo, Loveday, and Smith[3] studied 100 children at Boston they found that 52% of fevers were due to infection, 20% to collagen-vascular diseases, and 6% to neoplasms—but their cases were not comparable because they included such conditions as pneumonia and anaphylactoid purpura which should have been readily diagnosed.

The infections in children which present diagnostic difficulties include brucellosis, toxocariasis, toxoplasmosis, subacute bacterial endocarditis (before a murmur develops), low grade osteitis (which may sometimes elude diagnosis even for a few weeks), malaria in the tropics, an apical tooth infection (when there has been a root filling and the tooth is dead), a subphrenic abscess (after an abdominal operation), colonization of a Spitz-Holter valve, or miliary tuberculosis (with a negative tuberculin reaction due to severe malnutrition, measles, or overwhelming infection). Otherwise unexplained postoperative fever in child or adult should suggest the possibility of a retained swab or instrument.

In a review of fever in malignant disease[4] fever was found to be common in leukaemia, to occur at some stage in 20% of cases of Wilms's tumour, and to be common in various reticuloses but rare in Hodgkin's disease. Fever in malignant disease being treated by cytotoxic drugs may be wrongly ascribed to the underlying disease when it is due to infection associated with granulopenia.

Prolonged fever may precede joint changes in juvenile rheumatoid arthritis by many months,[5 6] causing great diagnostic difficulties. In children specific tests for rheumatoid arthritis are usually negative and rarely help. In one study of 124 cases of juvenile rheumatoid arthritis episodes of high fever were prominent in 26%; and 10% had prolonged fever before joint symptoms developed.

When taking the history in a case of P.U.O. it is essential to list all drugs being taken. Many drugs cause "drug fever"—and several workers[1 3 7] have decried the blind use of antibiotics for unexplained fever because they may obscure the diagnosis by themselves causing such a reaction—but one must not be so scientific that one lets a patient die rather than treat an ill patient empirically until the organism has been identified. Drugs known occasionally to cause fever include acetazolamide, amphetamine, azathioprine, barbiturates, carbamazepine, cephalosporins, chlorpromazine, colistin, erythromycin, haloperidol, indomethacin, isoniazid, meprobamate, methicillin, methimazole, nitrofurantoin, nortriptyline, P.A.S., phenytoin, potassium iodide, rifampicin, sulphonamides, and thiouracil.

Fever in young children may be due to overheating or dehydration: dehydration may be due to excessive fluid loss from polyuria due to hypercalcaemia, renal acidosis, or nephrogenic diabetes insipidus; fever may also occur in anhydrotic ectodermal dysplasia.

If the episodes of "fever" are notably short, the pulse is normal at the time, and the patient looks well, the possibility of malingering should be considered. The obvious method of making the temperature rise is putting the thermometer on to a hot water bottle; less obvious methods include two or three vigorous shakes of the inverted thermometer, while a 9-year-old girl successfully deceived doctors and nurses by vigorously rubbing the bulb of the thermometer: vigorous rubbing may make the mercury rise by 2 or 3°C in less than ten seconds.

Finally it is not always possible to detect organic disease even after full investigation and prolonged observation. Eleven of a series of 60 cases of P.U.O. in children in one study[7] and 12 of 100 children with P.U.O. in another series[3] fell into this category.

Blunderbuss investigation is costly, unwise, and unpleasant for the patient: the investigations requested should be decided on the individual's history, symptoms, and signs. They will certainly include blood and urine culture and perhaps culture of the faeces; a complete blood count, *x*-ray of the chest, and a tuberculin test. A sedimentation rate is a useful non-specific investigation: but it was normal in 9 of 60 cases of P.U.O. with proved systemic disease.[7] Other investigations may include serological studies, examination for LE cells, electrophoresis, and perhaps liver function tests.

Essentials to a diagnosis are a detailed history, including the medicines being taken, repeated physical examination, and the willingness to wait and see; and in certain circumstances patience may be augmented by biopsy of a lymph node if there is a suitable one, bone marrow examination, and, in the presence of abdominal symptoms, laparotomy.

[1] Petersdorf, R. G., and Beeson, P. B., *Medicine (Baltimore)*, 1961, **40**, 1.
[2] Jacoby, G. A., and Swartz, M. N., *New England Journal of Medicine*, 1973, **289**, 1407.
[3] Pizzo, P. A., Lovejoy, F. H., and Smith, D. H., *Pediatrics*, 1975, **55**, 468.
[4] *British Medical Journal*, 1974, **1**, 591.
[5] Laaksonen, A., *Acta Paediatrica Scandinavica*, 1966, Suppl. 166.
[6] Schaller, J., and Wedgwood, R. J., *Pediatrics*, 1972, **50**, 940.
[7] Sheon, R. P., and Van Ommen, R. A., *American Journal of Medicine*, 1962, **34**, 486.

Brain Abscess

The changes in the pattern of otological disease which have taken place in the last 30 years[1] have been reflected generally in a reduced incidence of otogenic abscesses of the brain.[2] However, this is not universal. Shaw and Russell[3] have recently noted a persistently high incidence of chronic ear disease in West Scotland and a constant incidence of intracranial abscesses as a result. Furthermore, the mortality from cere-