femoral side. Loosening of an uncemented prosthesis is occasionally demonstrable on the radiographs, but is rarely associated with pain, and the incidence of loosening is certainly not greater in patients with osteoporosis than those with a normal bone structure. Under these circumstances, it seems unreasonable to expose patients to the additional hazards of bone cement, particularly after a femoral fracture, when an uncemented technique can be expected to give an equally satisfactory result.—I am, etc.,

P. A. RING

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SIR,—I was most interested to read the report from Dr. G. A. Gresham and others (12 June, p. 617) concerning fat embolism following replacement arthroplasty for transcervical fractures of the femur.

I have now had three patients with fat embolism occurring after total knee replacement, one of whom died, and one occurring after a McKee arthroplasty. The one common feature in all these patients was that they were suffering from rheumatoid arthritis. In these patients the marrow fat is very excessive and almost liquid in consistency. It may well be that the rheumatoid patient is more liable to fat embolism following the insertion of acrylic cement.

Whatever the mechanism I feel certain that all surgeons who are using this material should be aware of the possibility of this complication. The diagnosis I am sure is often missed because the signs are masked when the patient is having a general anaesthetic.—I am, etc,

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### Accidental Poisoning with Malathion

SIR,—A three-year-old girl was admitted with a history of having swallowed malathion, an organophosphorous insecticide. The patient presented with unconsciousness, cyanosis, constricted pupils, and excessive salivation. Thoracic respiratory movement was diminished and the rate of abdominal respiration was 28 per minute, the pulse was 130, and the blood pressure was 80/60.

Initial treatment was intubation with positive pressure oxygen and stomach aspiration. Atropine was given in doses of 0·03 mg subcutaneously at ten-minute intervals on seven occasions. On this treatment the patient improved within one hour, by which time the colour was pink, the chest was moving normally at the rate of 16 per minute, the pupils were dilated, and the excessive salivation had dried up. However, two hours after her apparent recovery there was a complete relapse; there was again cyanosis, respiratory distress, constricted pupils, and salivation. This time oxygen was delivered with a "Venti-mask" and atropine 0·03 mg was given on six occasions at ten-minute intervals. The patient responded well.

The next morning the patient had a light breakfast and was up playing with other children in the ward. The only abnormal clinical finding was a few scattered sibilant rhonchi in the left lung. Later on the same day the patient suffered repeated petit mal attacks tending to fall to one side. The attacks passed off without anticonvulsant therapy. The patient did not suffer any other ill effects and progressed uneventfully. Chest x-ray was normal as was the blood picture and she was discharged after five days' observation.

Malathion, dimethylphosphorothiolothionate, is the least toxic of the organophosphate insecticides and is used extensively in agriculture and in the veterinary field. The insecticide is approved by the U.K. Ministry of Agriculture, Fisheries, and Food, under the following names: Malathexo Dust, Malathion 60 Cyanamid, Malastan 60, Malathexo 60, Murphy Malathion 60, and Vitax Malathion 60. Few cases of malathion poisoning in young children have been reported.[1-3] The recommended dose of atropine in children should be 1 mg to 2 mg intravenously or intramuscularly every ten to fifteen minutes until the signs of atropinization appear and also that cholinesterase activators should be avoided.[4] However, the value of pralidoxime chloride (Protopam) was advocated for treating the muscarinic effects[5 6] and also that treatment with pralidixime is effective only during the first day or two provided that absorption of the insecticide is complete.[6] A report was made of respiratory paralysis due to acute necrotic myelitis following inhalation of fumes four days previously.[7]

It is hoped that this case will refresh memories on the treatment of organophosphate insecticide poisoning.—I am, etc.,

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### Penicillin and the Mouth Flora

SIR,—I beg your readers not to be misled by the claim of Professor O. Khairat (12 June, p. 650) that an intravenous dose of 275 mg pyrrolidinomethyl tetracycline will abolish the bacteriaemia following dental extraction. I have refuted this claim in two other journals,[1 2] but since he has repeated it in yours I must ask to be allowed to do so again. He persists in ignoring the fact that the very high immediate blood concentration achieved invalidates the results of his blood cultures. Taking this concentration as 24 μg/ml, his broth cultures and pour plates must have contained 8 and 1·6 μg/ml respectively, and even the lower of these concentrations is more than enough to inhibit the growth of most mouth bacteria. This level remains constant in a culture, whereas that in the body falls as the antibiotic is excreted. In fact the "sterility" of these cultures is only apparent.

There is no such thing as an antibacterial drug which will kill bacteria in the circulation within one minute. Penicillin and other appropriate bactericidal antibiotics require several hours. Tetracyclines, the action of which is little more than bacteristatic, cannot be expected to do so at all.—I am, etc.,

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Radlett,
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### Management of Malignant Disease

SIR,—The report of the M.R.C. Working Party (22 May, p. 421) makes two points: (1) cytotoxic drugs as used by the method described has not increased survival of lung cancer, and (2) it may possibly have shortened the survival.

Those people practising the management of malignant disease and who have much experience in its control now feel unquestionably that it is vital that the immunological state should not be disturbed any more than is essential. As a result of 2,000 tests done at this hospital on the study of immune response certain conclusions have already been published.[1 2] These suggest that a good deal of immunological damage is done by cytotoxic drug treatment as in the manner employed in the trial, and it is not the slightest bit surprising that the results have been so bad. Conversely to general cytotoxic damage restriction of radiation to a precise zone of neoplastic invasion with the avoidance of radiation of normal tissue has resulted in a better response and it appears that metastases are now infrequently seen.

However, one swallow does not make a summer and a trial will be necessary to prove this.—I am, etc.,

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### Dermatoglyphics and Chromosomes in Cat-eye Syndrome

SIR,—We have recently seen a 2½-year-old girl with features of the cat-eye syndrome: iris collobomata, preauricular fistulae, and anal atresia, as first described by Haab in 1878[1] and clearly definable as a syndrome with the discovery of its chromosome anomaly by Schachenmann *et al.* in 1965.[2] Renal abnormalities have also been previously found in this syndrome, although no pathological changes have yet been demonstrated on intravenous pyelography in the present child. However, recurrent urinary infections have occurred.

In this patient, as in previously published cases,[2-5] there is an extra chromosome, about half the size of those in group G. Morphologically the anomalous chromosome, in our patient, is submetacentric with satellites on the longer arm. Fifty metaphases examined in blood culture all had the extra marker chromosome.

The patient's father has a normal karyotype, but the mother was found to have the same marker chromosome in fifty metaphases studied in a blood culture. Since the mother lacked signs of cat-eye syndrome it is possible that she is in fact a mosaic in other tissues, as was actually found in another study.[2] Similarly a previously reported example of the syndrome itself, reported to have a normal karyotype,[9] may possibly have been an undetected mosaic.

Chromosomal anomalies occurring before the 13th week of fetal growth (when dermal patterns are being formed[6]) can be expressed phenotypically by abnormal dermatoglyphics, as we found in our patient.

Palm prints showed an unusually distal t (axial) triradius and an ulnar loop in the proximal hypothenar area on both palms.