finding that sublingual temperature was sometimes depressed by 1·1°C even in a normal clinical environment is perhaps more important. The possible consequences, for example, of relying on a misleading sublingual reading of 36·4°C in a case of appendicitis with a true central body temperature of 37·5°C need no emphasis.

In air above 25°C sublingual temperature readings were close to oesophageal ones (± 0·45°C). Maintenance of room temperature above this level might therefore seem the simplest way of obtaining reliable routine clinical measurements of body temperature. Such high environmental temperatures are most uncommon, however, and probably often unacceptable in British homes and waiting rooms. At a time of growing energy shortages there is a strong case for adopting a method of measuring body temperature that will be reliable in colder air.

Rectal temperature is inconvenient to measure and lags seriously when body temperature changes (Mellette, 1950; Cooper and Kenyon, 1957), as presumably does urine temperature (Fox *et al.*, 1971). Oesophageal probes are too uncomfortable for routine outpatient use. Local warming of the face would allow reliable sublingual readings if it was well controlled. A procedure that might prove satisfactory for monitoring is to measure external aural canal temperature while a simple servocontrolled heating device keeps the outer ear at the same temperature. The aural canal probe can then give readings to within 0·35°C of oesophageal temperature (Keatinge and Sloan, 1973, 1975), though stabilization is slow from the cold state. A similar device has been shown to give reliable results in the newborn infant (Cross and Stratton, 1974).

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References

Cooper, K. E., and Kenyon, J. R. (1957). *British Journal of Surgery*, **44**, 616.
Cranston, W. I., Gerbrandy, J., and Snell, E. S. (1954). *Journal of Physiology*, **126**, 347.
Cross, K. W., and Stratton, D. (1974). *Lancet*, **2**, 1179.
Dorodnitsina, A. A. (1937). *Fiziologicheskii Zhurnal SSSR*, **27**, (1), 111.
Fox, R. H., *et al.* (1971). *Lancet*, **1**, 424.
Hercus, V., Cohen, D., and Bowring, A. C. (1959). *British Medical Journal*, **1**, 1439.
Keatinge, W. R., and Sloan, R. E. G. (1973). *Journal of Physiology*, **234**, 8P.
Keatinge, W. R., and Sloan, R. E. G. (1975). *Journal of Applied Physiology*. In press.
Lashley, K. S. (1916). *Journal of Experimental Psychology*, **1**, 461.
Louridis, *et al.* (1970). *Journal of Dental Research*, **49**, 1136.
Mellette, H. C. (1950). *American Journal of Physiology*, **163**, 734.
Pembrey, M. S. (1898). *Textbook of Physiology*, ed E. A. Schafer, p. 786. London, Pentland.
Shannon, I. L. (1966). *Archives of Oral Biology*, **11**, 451.
Tanner, J. M. (1951). *Journal of Physiology*, **115**, 391.

MEDICAL MEMORANDA

Lymphoma Presenting as "Idiopathic" Juvenile Osteoporosis

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Bone involvement in the lymphomas is well recognized, though primary and more especially diffuse disease of bone is unusual. Osteoporosis principally of the axial skeleton with gross changes in the spine must be extremely rare as a presenting manifestation of lymphoma and has not been documented. For these reasons the following case is reported.

Case History

A 14-year-old prepubertal boy was immobilized in traction for 10 weeks in March 1972 after a motor-cycle accident in which he fractured his right femur. He was subsequently mobilized and discharged home. He returned to school for a month but then developed pain in the hips, back, and shoulders. By February 1973 he had developed kyphosis, and *x*-ray films showed marked osteoporosis with collapsed vertebrae. His plasma calcium was 2·75 mmol/l (11 mg/100 ml) and urinary calcium 146·2 mmol/l (585 mg/100 ml). The conclusion was that these changes could have been due to immobilization.

Physiotherapy resulted in some improvement with a gradual fall in urinary calcium excretion but progress seemed slow, and in June 1973 he was transferred to the metabolic unit of the Royal National Orthopaedic Hospital, Stanmore. At that time the positive findings were slight upper thoracic kyphosis and tender lumbar vertebrae. *X*-ray films showed severe reduction in bone density, chiefly confined to the axial skeleton (fig. 1). Blood values were: calcium 2·73 mmol/l (10·9 mg/100 ml), phosphorus 1·45 mmol/l (4·5 mg/100 ml), alkaline phosphatase 108 K.A. units/l, urea 4·4 mmol/l (27 mg/100 ml),

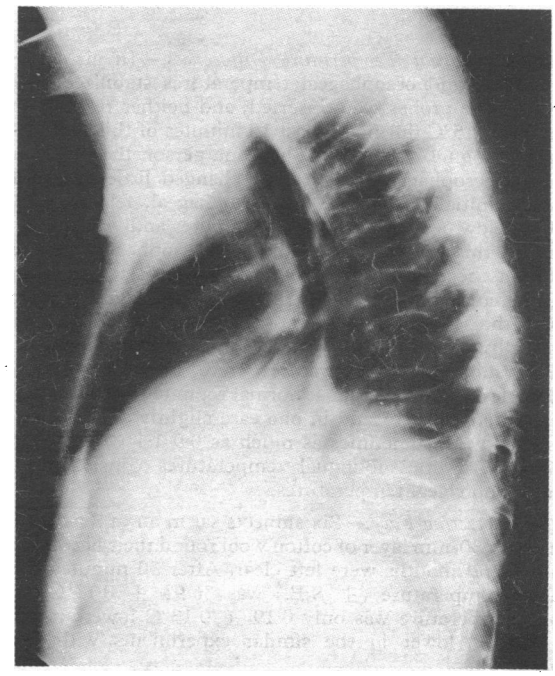

FIG. 1—*X*-ray film taken June 1973 showing severe reduction in spinal bone density with vertebral collapse.

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electrolytes normal, total proteins 73 g (albumin 47 g, globulin 26 g)/l (with raised α2-globulin on electrophoresis), haemoglobin 11·2 g/dl, E.S.R. 36 mm in the first hour, W.B.C. 5 × 10^9/l (normal differential), and platelets, in film, normal. Urinary calcium was 5·87–6·87 mmol, (235–275 mg)/24 h. Sternal marrow aspirate showed active haematopoiesis with erythroid hyperplasia. There was no evidence of leukaemia or malignant infiltration.

In the absence of evidence of malignant disease a tentative diagnosis of idiopathic juvenile osteoporosis was made and he was given a course of stilboestrol and methyltestosterone. He was discharged in August 1973 but made little progress and was readmitted in October with increasing malaise. He was anaemic, and there was a yet more marked thoracic kyphosis; movement of the legs was full but painful. Clinically there was no lymphadenopathy or hepatosplenomegaly. *X*-ray examination showed collapse of all dorsal and lumbar vertebrae. The pelvis, proximal ends of the femora, the ribs, and the scapulae showed numerous areas of osteolysis (fig. 2). A fracture through the sternum had occurred. Serum calcium was 3·07 mmol/l (12·3 mg/100 ml), phosphorus 1·58 mmol/l (4·9 mg/100 ml), and alkaline phosphatase 138 K.A. units/l. Urinary calcium was 7·75–8·12 mmol (310–325 mg)/24 h. The blood picture showed anaemia (haemoglobin 9·4 g/dl); the W.B.C. was 5 × 10^9/l (normal differential) and the platelet count was normal. An iliac crest aspirate and trephine biopsy specimen were obtained. The aspirate suggested that 90% of nucleated cells were lymphocytic; few of them were true blast cells. Histological examination showed sheets of cells with uniform appearance suggesting a malignant lymphoma, and this was classified as a diffuse lymphocytic lymphoma, moderately poorly differentiated (Rappaport (1966) classification). He was transferred to the Royal Marsden Hospital, Sutton.

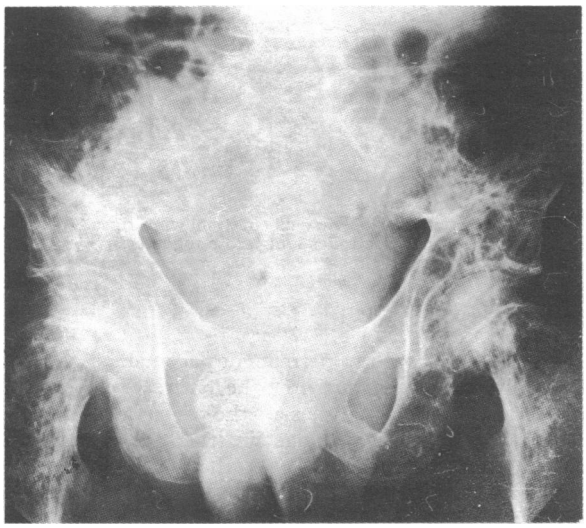

FIG. 2—*X*-ray film taken October 1973 showing development of numerous areas of osteolysis with vertebral collapse.

The diagnosis of lymphoma was confirmed. The lymphoma appeared to be confined to bone, though neither lymphography nor liver biopsy was carried out in view of his poor condition. He showed no response to a variety of chemotherapeutic agents including prednisone, vincristine, colaspase (L-asparaginase), cytarabine, methotrexate, and mercaptopurine. He was last given cyclophosphamide with relief of symptoms but without radiological evidence of improvement.

Comment

Lymphoma presenting with gross osteoporosis appears to be extremely unusual. Previous reports of bone involvement in non-Hodgkin's lymphoma provide conflicting evidence about its frequency and the form it may take. Changes in terminology complicate their appraisal. Rosenberg *et al.* (1958) recorded 7·5% of 42 children with "lymphosarcoma" as having presented with bone lesions, usually painful and solitary. Certainly, the incidence of such changes depends on how assiduously they are looked for, and this is implied by Hustu and Pinkel (1967), who claimed that osteolytic lesions were often found during the course of childhood lymphoma if searched for carefully. Schey *et al.* (1973) considered the occurrence of both osteolytic lesions and diffuse osteoporosis, observing that an apparent decrease in bone density was common in lymphoma, possibly induced by the "catabolic state" resulting from the disease or its treatment. They found no examples of *primary* bone involvement in their review of 60 infants and children with lymphoma.

Acute lymphoblastic leukaemia, which often pursues a clinical course similar to the more poorly differentiated lymphomas, is well recognized as producing skeletal abnormalities, including generalized demineralization, and has recently been described as presenting with vertebral osteoporosis and collapse (Neuman and Melhorn, 1973). Our patient had a peripheral blood picture and marrow aspirate ruling out this diagnosis. Acute juvenile osteoporosis is a clearly described, non-malignant entity which acute leukaemia can mimic in all respects, and one of Dent and Friedman's (1965) patients, originally thought to have idiopathic osteoporosis, turned out to have acute leukaemia.

The histological features in this case were reviewed by Professor R. J. Lukes according to a new classification of lymphomas based on morphology and immunological properties of the affected lymphoid cell (Lukes and Tindle, 1973). He considered the principal cell type involved here to be of a kind typically found in mediastinal lymphoma, usually with marrow involvement (sometimes called Sternberg sarcoma); he has seen no other case of this cell type involving bone in this way.

Finally, the disappointing resistance of the lymphoma to a variety of chemotherapeutic agents is also atypical, though not exceptional.

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References

Dent, C. E., and Friedman, M. (1965). *Quarterly Journal of Medicine*, **34**, 117.
Hustu, H. O., and Pinkel, D. (1967). *Clinical Orthopaedics and Related Research*, **52**, 83.
Lukes, R. J., and Tindle, B. H. (1973). *Workshop on Classification of Non-Hodgkin's Lymphomas*. Illinois, University of Chicago.
Neuman, A. J., and Melhorn, D. K. (1973). *American Journal of Diseases of Children*, **125**, 863.
Rappaport, H. (1966). *Tumours of the Haemopoietic System*. Washington, Armed Forces Institute of Pathology.
Rosenberg, S. A., *et al.* (1958). *New England Journal of Medicine*, **259**, 505.
Schey, W. L., *et al.* (1973). *American Journal of Roentgenology, Radium Therapy, and Nuclear Medicine*, **117**, 59.