may produce short- or long-term effects, both in terms of adaptation and possibly even of pathology.

Radiology of Dysbaric Osteonecrosis J. DAVIDSON (*Department of Radiology, Western Infirmary, Glasgow*) Exposure to a hyperbaric environment either in tunnel work or diving may result in the acute type I or type II forms of decompression sickness ('the bends') and also the late complication of aseptic necrosis of bone—sometimes called caisson disease of bone, barotraumatic osteoarthropathy, etc. Dysbaric osteonecrosis is now the accepted term and includes the rare forms of osteonecrosis following exposure to a low pressure environment.

Dysbaric osteonecrosis is a major hazard to compressed air workers and an increasing danger to divers especially with longer exposure times at greater depths. Young men, often in their 20s, may develop a painful arthritis of the shoulder or hip joint with consequent permanent disability.

While the condition has been known for many years the diagnosis was rarely made until the bone changes were sufficiently advanced with an associated structural failure of the articular surface causing symptoms. The first attempt to identify the incidence of dysbaric osteonecrosis was carried out in Glasgow in 1963 when the entire labour force of 250 men constructing the Clyde tunnel were examined radiographically and the findings related to the occupational history. The incidence of osteonecrosis was 19% and in 10% of the men the lesions were juxta-articular and, therefore, potentially disabling. Since then all compressed air workers in the United Kingdom have had a radiographic skeletal survey and the MRC Decompression Sickness Registry has examined 1694 men showing an incidence of 19.7% with definite osteonecrosis and 11% with a juxta-articular lesion.

The head of the humerus is most frequently involved and 36% of the lesions are bilateral. The next most common site is the distal shaft of the femur. Other sites included the head of the femur, proximal shaft of the tibia, and less commonly the talus, fibula, or humerus. The lesions have been classified into two broad groups: juxta-articular which are next to the surface and liable to cause structural failure with symptoms, and medullary where the lesion is at some distance from the joint surface and will never give rise to symptoms. Further subclassifications have been made according to the radiographic appearances.

The earliest radiographic features are areas of increased density adjacent to the articular surface and these result from new bone laid on dead trabeculae resulting in an overall increase in bone bulk. These develop about eight months to a year after

initial exposure to a hyperbaric environment and may be seen within four months. These lesions may remain static or progress to a structural failure of the joint surface and then to secondary osteoarthritis. In the medulla the earliest features are small areas of increased density and small foci of calcification which eventually become quite large. Any attempt to reduce the incidence of the condition must be monitored by high quality radiology and there must be a high index of suspicion in those areas at risk. In a few cases we have been able to correlate the radiographic and pathological findings and this has been of immense value in identifying the earliest features with confidence. Many report thickening of the trabeculae or small cyst as the earliest evidence of osteonecrosis but these radiographic findings have not been substantiated at histopathology. For this reason and because these changes can be identified in those not at risk we have not recorded these findings as positive. Clearly then there is much scope for further correlation between pathology and radiology. This would be of immense value.

The diagnosis of dysbaric osteonecrosis is not usually difficult and all other causes of aseptic necrosis of bone must be kept in mind. The most important ones to be excluded are those following fracture of the femoral neck or dislocation of the hip, the idiopathic form sometimes associated with large doses of steroids, the haemoglobinopathies, Gaucher's disease, and Schandler's disease.

Necropsy radiographs of the chest following diving fatalities have been of considerable interest, demonstrating widespread intravascular gas/air in the axillary and carotid vessels. Some have shown either a pneumothorax or bulla formation which supports the theory that pulmonary air trapping could be a cause of massive air embolism. Two such cases will be included in the paper.

NECROPSY RADIOGRAPHY OF DIVING AND COMPRESSED AIR FATALITIES

A necropsy chest radiograph can be of considerable value. It may show evidence of a pneumothorax, cyst or bulla formation, mediastinal emphysema. Careful inspection of the axilla may show extensive gas/air in the axillary vessels. Surgical emphysema may be present. Similarly a radiograph of both groins may show widespread gas/air in the ilio-femoral artery and vein segments.

Antero-posterior radiographs of the head of each humerus should be coned to show optimum trabecular detail and the body rotated slightly in order that the central x-ray beam passes vertically through the shoulder joint.

Antero-posterior radiographs of the head of each femur should be coned to show trabecular detail.

Lateral radiographs of the knee should show good trabecular detail and include the mid shaft of the femur to the mid shaft of the tibia. Correlation of the radiographic and pathological findings is of considerable value and importance in identifying the earliest radiographic features of osteonecrosis with confidence and differentiating this from variation in normal trabecular structure.

Pathology of Caisson Disease of Bone MARY E. CATTO (*Department of Pathology, Western Infirmary, Glasgow*) While histological examination is of little help in elucidating the pathogenesis of aseptic bone necrosis in compressed air workers, it throws some light on the sequence of events and radiological changes following bone death. Revascularization of both medullary and juxtaarticular lesions may begin but halt short of completion, the revascularization front becoming collagenous. Bone trabeculae adjacent to this fibrous tissue are often greatly thickened and may give rise to a sclerotic line on clinical radiographs. When such a radio-dense line is seen traversing a bone end it is highly probable that the tissue between it and the joint surface is still dead. The necrotic bone trabeculae may later fracture, with collapse of the articular surface associated with pain. Incongruity of the joint surface is often followed by formation of osteophytes at the living joint margins. At first the joint space remains normal and the articular cartilage covering dead bone is relatively well preserved but later it and the underlying dead bone may be ground away, the end result sometimes being difficult to distinguish from primary osteoarthritis. A similar pattern of events and morphological changes may be seen following juxtaarticular bone necrosis due to other causes.

Scientific communications II

Cervical Adenitis Caused by *Mycobacterium chelonae* (*M. abscessus*) C. A. MORRIS AND G. H. GRANT (*Public Health Laboratory and Royal Salop Infirmary, Shrewsbury*) In February 1972, an 8-year-old boy presented with a painless submandibular swelling causing some neck discomfort. The swelling persisted in spite of antibiotics; it was explored surgically and a soft tissue mass excised. The wound healed uneventfully. No other abnormalities were found on clinical and x-ray examination.

Histology of the excised mass showed lymphatic tissue with a tuberculoid reaction. There were numerous large collections of epithelioid cells, some with central necrosis and giant cells. There was no anisotropic material. Very scanty acid-fast bacilli were seen. *Mycobacterium tuberculosis* was not

isolated by culture or by guinea pig inoculation. A pure growth of a non-pigmented mycobacterium was cultured, which was slow to grow on primary isolation, but rapid on subculture. Dr J. Marks of the Tuberculosis Reference Laboratory, Cardiff, examined the isolate by lipid analysis and identified it as *Mycobacterium abscessus*.

The patient showed a negative Mantoux tuberculin reaction at 1/10000 and weakly positive at 1/1000. There was marked skin hypersensitivity to an extract of sonically disrupted live organisms of *M. chelonae* (synonym *M. abscessus*) at 1/10 000, but none to that of *M. ranae* (synonym *M. fortuitum*) at 1/1000. A cat-scratch fever intradermal test was negative.

Mycobacterium chelonae has caused abscesses following the injection of contaminated drugs or vaccines. This child had an injection of dental anaesthetic four months before the appearance of the lesion; this may indicate the portal of entry of infection. This is thought to be the first recorded case of cervical adenitis caused by *M. chelonae*, and the strain has been deposited in the National Collection of Type Cultures (NCTC 10882).

Intravascular Coagulation and Renal Failure in *E. coli* Septicaemia F. E. PRESTON, R. G. MALIA, M. J. SWORN AND E. K. BLACKBURN (*Departments of Haematology and Pathology, The Royal Infirmary, Sheffield*) Although various haematological abnormalities have been described in patients with Gram-negative septicaemia, the precise pathogenesis of the reported findings has remained obscure. In the eight patients described in this study, *E. coli* septicaemia associated with oliguric renal failure showed evidence of intravascular coagulation.

Haematological evidence of intravascular coagulation was obtained in all eight patients. Thus elevated fibrin degradation products in the serum and thrombocytopenia were constant features, while plasma fibrinogen depletion was noted in six out of eight. The diagnosis of intravascular coagulation was subsequently confirmed by histological examination of necropsy material from the five patients who died: there was no constant pattern of distribution of intravascular fibrin.

A falling haemoglobin and a peripheral blood picture characteristic of a microangiopathic haemolytic anaemia was observed in five of the patients. These changes which can be attributed to mechanical damage of red cells by intravascular strands of fibrin returned to normal after successful clinical management.

Although oliguric renal failure is a recognized complication of septicaemia, the precise mechanism by which it occurs has remained obscure. Recently,