information on the origin of particular lymphocytes particularly in relation to tumours of the lymphoreticular system.

Functional Disorders of the Lymphoreticular System H. E. M. KAY (*Royal Marsden Hospital, London*) Functional disorders of the lymphoreticular system may arise through primary defects of phagocytosis of antibody formation or of T-cell function. There is considerable interaction between the components of the system so that an apparent failure of macrophage function may be due to lack of antibodies or of lymphokines, eg, lack of MIF in lepromatous leprosy. Conversely, failure of macrophages to process antigen, as in the Wiskott-Aldrich syndrome, leads to lack of antibodies and effective lymphocyte activity against certain classes of antigen.

The very number of components of the system, however, enables compensatory mechanisms to come into play when a single activity is absent, eg, pure IgA deficiency, thymic aplasia, and the lazy leucocyte syndrome. At present methods are being elaborated to identify the precise step which is at fault in each functional disorder, as in the different forms of chronic mucocutaneous candidiasis, so that appropriate therapy, eg, by transfer factor or thymic transplantation, can be given.

Autoimmunity can arise in many ways through deficiency of T-cells, or by misinformation during T-cell/B-cell collaboration. Neoplasm of the lymphoid system can arise from any of the components and may give rise to functional disorders such as autoimmunity or to deficient normal function by interference with homeostasis, eg, hypogammaglobulinaemia in chronic lymphatic leukaemia.

Symposium II

Decompression sickness

Aetiology of Decompression Sickness R. I. MACCALLUM (*Nuffield Department of Industrial Health, University of Newcastle-upon-Tyne*) Tunnellers, caisson workers, and divers who are exposed to air pressure above normal atmospheric pressure may suffer from decompression sickness after the pressure is reduced to the normal level. Acute decompression sickness presents as pain in a limb (the bends, type I decompression sickness) or as a variety of signs and symptoms affecting the central nervous, vascular, or respiratory systems (type II). Chronic sequelae are aseptic necrosis of bone (avascular necrosis, caisson disease of bone) and neurological complications. There may be other long-term defects such as damage to the vestibular apparatus. There is at present no decompression procedure which will avoid with certainty any of the types of decompression sickness, including bone necrosis.

It has been assumed that all types of decompression sickness are due to the formation of bubbles of nitrogen during decompression and that more efficient decompression would prevent this. These assumptions are being questioned and alternative theories of the pathogenesis of decompression sickness are being put forward, but at present no convincing and coherent explanation of all these phenomena of decompression sickness has been constructed.

It seems probable that all decompressions are accompanied by some bubble formation, but that other body changes, eg, in the blood, may be more important than has been thought hitherto.

Aseptic necrosis of bone occurs in about 20% of compressed air workers and divers. In a small proportion of cases it can lead to marked disability, particularly if the hip joints are affected. It seems likely that bone necrosis may arise from the operation of several factors during the processes of compression or decompression or both, but further observations are required on human bone tissue of which there is a great scarcity in these cases.

Observations on Haematological and Biochemical Parameters K. J. MARTIN (*Royal Naval Physiological Laboratory, Gosport*) A series of experiments designed to determine normal levels for selected haematological and biochemical parameters in the context of a simulated hyperbaric exposure devoid of signs or symptoms of decompression sickness.

The parameters studied included platelets, lipids, enzymes, plasma cortisol, and coagulation factors. The experiments were designed to differentiate between the true effect of pressure and the psychosomatic response to the situation. A control group was included.

The results indicate a biphasic response to the exposure. Immediate effects were noted with regard to steroid and free fatty acids indicative of an influence on metabolism, and changes in euglobulin lysis activity pointed to a psychosomatic response. Delayed effects were found in the platelet, aspartate aminotransferase, alkaline phosphatase, and creatine phosphokinase studies. Residual effects of previous diving experience were attributed to some of the enzyme patterns elicited.

It was concluded that a normal symptom-free hyperbaric exposure induces a series of changes, some of which are similar to those seen in the post-traumatic situation. It is upon this baseline that results obtained in cases of decompression sickness are superimposed. Subclinical changes of this nature

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 juxtaarticular 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: juxtaarticular 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 iliofemoral 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.