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