

both sexes are equally affected. Symptoms are malaise, low fever, and abdominal pain. A mass is usually felt. Twenty-two of these 34 patients underwent major bowel resection because of uncertain or mistaken clinical diagnosis, including three of six Europeans who were thought to have carcinoma of the caecum. Three characteristic pathological presentations may be distinguished: (1) In the acute phase a worm track is found leading to an abscess in or near the bowel wall. The contents are thick, odourless, sterile pus, and, often, a live parasite. (2) After some weeks the worm dies, the cavity is walled off and the histological appearances come to resemble caseating tuberculosis quite closely. (3) Eventually a fibrous mass results in which eosinophils remain prominent. It is likely that with increasing numbers of people travelling to, and returning from, the tropics these helminthic pseudotumours (helminthomas) will have to be considered in the differential diagnosis of intra-abdominal masses and inflammatory conditions.

**The Measurement of Circulating Ferritin** A. JACOBS, M. R. BEAMISH, AND M. ALLISON (*University Hospital of Wales, Cardiff*) Ferritin can be measured in serum by immunometric assay. The mean level in normal men is 69 ng/ml and in normal women 35 ng/ml. Patients with iron-deficiency anaemia have concentrations below 10 ng/ml and these levels are always associated with iron deficient erythropoiesis.

The concentration of ferritin in serum gives a quantitative measure of storage iron both in normal subjects and those with iron overload. In patients with Hodgkin's disease the fall in the serum iron concentration is associated with a rise in the serum ferritin levels.

## Symposium I

### The lymphoreticular system

**The Lymphoreticular System: Morphological Organization** KRISTIN HENRY (*Department of Pathology, Royal Postgraduate Medical School, London*) At both a functional and anatomical level lymphoreticular tissue can be horizontally divided into central or primary lymphoid organs such as thymus and the avian bursa of Fabricius, and peripheral or secondary lymphoid tissue such as lymph nodes, spleen, and gut-associated lymphoid tissue. Peripheral lymphoid tissue functions in an executive capacity subserving both cell-mediated immune reactions as well as antibody production, and also for clearing the blood, lymph, and tissue of particulate matter. Thus, these tissues are specifically organized and

equipped for antigen capture and processing, for antibody production, and for non-specific, ie, non-immunological, phagocytosis. Structural differences between these peripheral lymphoid tissues exists according to location, but all are characterized by their lymphocytic nature, the presence of primary lymphoid follicles and highly complex germinal centres, an intricate vasculature, and a framework of supporting cells and reticulin fibres between which are found the executive cells. According to their light- and ultrastructural features, the component nucleated cells of peripheral lymphoid tissues have been allocated to one of the following categories: (1) the lymphocyte series (T- and B- cell lines); (2) the dendritic (reticular) cells; (3) cells of the mononuclear phagocyte system; (4) endothelial cells; and (5) the supporting 'reticular' cells. The thymus differs in many respects from peripheral lymphoid tissue, but on a purely morphological basis can be distinguished by its unique lympho-epithelial structure and by a virtual absence of any morphological expression of local immune responsiveness. In the adult, bone marrow is the source of the stem cells for both central and peripheral tissues, and not the reticulum cell as previously supposed.

**Lymphoreticular System: Functional Organization** I. ROITT (*Immunology Department, Middlesex Hospital Medical School, London*) We now recognize two major lymphocyte populations, one dependent on the presence of the thymus gland (T cells) and the other controlled by the bursa or its equivalent in mammals (B cells). B lymphocytes mature to form the antibody-secreting cells, of which the plasma cell represents an end stage, and are therefore responsible for *humoral immunity*. T lymphocytes, when appropriately sensitized, confer a state of *cell-mediated immunity* on the host which affords defence against certain intracellular facultative organisms such as tubercle and leprosy bacilli and certain viruses including those of the pox group. Although not themselves capable of secreting immunoglobulins, T cells do cooperate with B cells in the antibody response enabling them to be triggered more readily by antigen. The other important functional characteristic of T lymphocytes is their ability to recognize and be activated by the surface antigens of viable 'foreign' cells of the same species and thence to transform into cytotoxic lymphoblasts capable of killing the target foreign cells in culture; this provides one important mechanism by which the host can reject homografts and presumably tumour cells which would be regarded as homografts if their specific neo-antigens were recognized as 'non-self'. Antibodies with specificities for surface markers on different lymphoid cells are providing increasing

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, hypogammaglobulinemia 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 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