Much less is known of the lymphatics of the pleura and chest wall. There are two main directions of lymph flow—forwards to the internal mammary chain of nodes and vessels, and backwards to the posterior mediastinum. Some of the lymphatics of the lower intercostal spaces drain downwards, through the diaphragm, to reach the cisterna chyli. Some of the mediastinal lymphatics communicate directly with the thoracic duct while the majority run upwards to join the paratracheal and pulmonary lymphatics and then drain by one or more vessels into the veins at the base of the neck near the thoracic duct.

The radiological appearances of the abnormal lymphatics in lymphangitis carcinomatosa, pulmonary oedema, etc., are discussed.

Lymphography

W. F. WHITE The lymphatics and lymph nodes draining many parts of the body may now be demonstrated by lymphography.

Visualization of the lymphatics is achieved by the subcutaneous injection of a suitable coloured material, and following cannulation radio-opaque material may be injected. In this way it is possible, by injecting the lymphatics on the dorsum of the foot, to visualize the inguinal, iliac and para-aortic nodes, together with the cisterna chyli, and the thoracic duct in some instances. The para-vertebral lymph nodes in the chest may be seen, but this is extremely variable.

The oily contrast medium enters the great veins and forms microemboli in the small pulmonary vessels. The complications of the technique are invariably manifest in the chest.

This communication concerns the demonstration of lymph nodes, the thoracic duct and the pulmonary complications of the technique.

Chylothorax and Chylous Reflux

K. ROSS A review is presented describing briefly the normal physiology of the thoracic duct and the aetiology of chylothorax.

The practical management of chylothorax is discussed in greater detail and the problem of chylous reflux, as it affects the pleural lymphatic system, is commented upon.

Physiological and Pathological Mechanisms Governing Fluid Accumulation within the Pleural Space

W. LECKIE Animal work illustrating the mechanisms responsible for preventing fluid accumulation within the pleural space is briefly reviewed. Predicted values for fluid and protein clearance in health are also deduced from animal studies.

Pathological mechanisms affecting fluid and protein balance within the pleural space are discussed, and the three factors (1) alterations in pleural capillary hydrostatic pressure, (2) alterations in pleural capillary permeability and (3) changes in lymphatic drainage, are related to different clinical conditions, *e.g*, pleural tuberculosis, malignancy, pulmonary infarct, congestive cardiac failure and Meigs's syndrome.

The effect of intrapleural corticosteroid administration on protein turnover in the pleural space is discussed.

Finally, in the light of these observations, a few recommendations regarding terminology and management are made.

Clinical Manifestations of Disorders of the Pulmonary and Pleural Lymphatics

PETER EMERSON The role of the lymphatics in pulmonary oedema is described with mention of drainage of the thoracic duct as a method of resolving intractable oedema in congestive failure and cirrhosis of the liver. The concept of chylous reflux and its clinical presentation is described together with the effect and the developmental abnormalities of the lymphatics of the lungs and pleura, *i.e.*, congenital pulmonary lymphangiectasis in infants and pulmonary and pleural disorders associated with developmental lymphoedema in adults.

SPONTANEOUS PNEUMOTHORAX IN CYSTIC FIBROSIS

P. F. MITCHELL-HEGGS Five cases of cystic fibrosis complicated by eight pneumothoraces are described. These cases come from a group of 49 patients who have attended the Brompton Hospital between 1964 and 1969 for management of their cystic fibrosis. Three of the patients, who had a total of six pneumothoraces, were managed by pleurectomy. Spontaneous pneumothoraces were a terminal event in a fourth patient and an incidental finding which required no specific treatment in a fifth patient.

The possible aetiology of the pneumothorax in cystic fibrosis is discussed with particular consideration of the known alveolar hypoplasia and overdistension and the abnormality of the visceral pleura. The results of pleurectomy in three patients are discussed both in relation to the successful avoidance of further pneumothoraces and in the notable absence of a restrictive ventilatory defect.

In view of the relative success of pleurectomy it is suggested that this treatment should be considered for the management of any patient with cystic fibrosis who develops a spontaneous pneumothorax.

PROBLEMS WITH LONG-TERM ENDOCARDIAL PACING IN CHILDREN

A. H. M. SIDDONS, K. CHATTERJEE, A. M. HARRIS and M. PEARD Fortunately it is uncommon that a child needs long-term cardiac pacing. Such patients usually have persistent complete heart block after cardiac surgery or repeated Stokes Adams attacks associated with congenital (and sometimes acquired) complete heart block.

Eight children aged 7 months to 15 years have had long-term pacemakers implanted, and all are still being paced at 9 to 31 months. Complications encountered relate mainly to the need to lengthen the endocardial electrode with growth, and the implantation of a large foreign body.

Various techniques have been tried to allow easy lengthening of the electrode wire. These include:
1. Leaving a loop of wire in the heart (in two out of three the electrode tip displaced);
2. Manually feeding more wire into the vein from spare loops retained in a subcutaneous nylon sleeve;
3. Initial suturing of the electrode tip to the endocardium, allowing spontaneous feeding of excess wire from a nylon bag into the vein, as the electrode straightens with growth.

Endocardial pacing seems a feasible long-term approach in children.

A DECADE OF THORACIC PAPERS

A. JOHN ROBERTSON During the last 10 years, over 700 papers have been received by the medical editor of *Thorax*. About 53% of these have been from the United Kingdom and 45% from 40 different overseas countries. About 40% of all papers were rejected, the acceptance rate being 64% from Great Britain and 56% from elsewhere. The countries with the highest acceptance rate were New Zealand and Australia: some examples of percentages from other places are shown, as well as some reasons for rejection. In 1960 the journal contained 360 pages, but in 1970, 768 pages are scheduled. The change in subject matter is briefly discussed, as well as methods of assessing and editing papers.

BUSULPHAN LUNG

Clinical Features

W. A. LITTLER Diffuse pulmonary fibrosis is now a recognized complication of busulphan therapy. Heard and Cooke (1968) examined the lungs of 14 patients dying of chronic myeloid leukaemia and found histological changes attributable to busulphan in six, but in only one of these had busulphan lung been suspected during life. This high incidence of busulphan lung at necropsy has prompted a prospective study to discover whether the condition can be detected earlier by means of serial clinical, radiographic and physiological examinations. The methods employed in this study and the preliminary findings are reported along with details of the first patient from the series in whom busulphan lung was diagnosed during life and confirmed at necropsy. This patient was a man of 61 years with chronic myeloid leukaemia treated over a period of 19 months with a total dose of 1,000 mg. busulphan. Evidence of pulmonary disease did not arise until one month after withdrawal of busulphan, and consisted of intense dyspnoea, a dry cough, central cyanosis and persistent crepitations at the lung bases. The chest radiograph showed a progressive diffuse mottling throughout both lungs and physiological tests demonstrated a restrictive ventilatory defect associated with a gas transfer factor of only 25% of the predicted normal. The pulmonary condition progressed to a fatal termination within three months of its onset despite the fact that busulphan had been withheld and large doses of prednisone given.

Histopathology

P. S. HASLETON Patients treated with busulphan may develop characteristic cellular and fibrous changes in their lungs. The alveoli are lined by large cells with big nuclei and copious cytoplasm which contains clear bodies resembling vacuoles. The cells desquamate into the alveolar spaces and disintegrate to form an amorphous eosinophilic debris. The identification of these cells and the ultrastructure of the debris are described in the following paper. The alveolar walls become thickened by chronic oedema and then interstitial fibrosis. Organization of the intra-alveolar debris also occurs. In the case described pulmonary cholesterol-ester granulomas were also present. They consisted of fibrous nodules around acicular cholesterol clefts with a surrounding chronic inflammatory exudate. The hypothesis is advanced that such granulomas are associated more with hyperplasia of granular pneumocytes than with pulmonary hypertension.

Electron Microscopy

J. M. KAY Electron microscopy was carried out to identify the large alveolar cells and to determine the nature of the intra-alveolar debris which characterize the histological picture of this condition. The enlarged alveolar cells had the ultrastructural features of granular (type II) pneumocytes. They possessed prominent intracytoplasmic lamellar secretory inclusions (lamellar bodies), and their free borders showed numerous irregular, short microvilli. The intra-alveolar debris consisted of strands of amorphous electron-dense fibrillary material interspersed with roughly circular bodies composed of concentric electron-dense membranes. The fibrillary material resembled fibrin and the circular bodies appeared identical to the lamellar secretory inclusion bodies of granular pneumocytes. In some instances, it appeared that granular pneumocytes had disintegrated and liberated their lamellar bodies, which had given rise to the intra-alveolar debris. Also scattered within the alveolar spaces were myelin figures which resembled phospholipid membranes. Evidence of organization of the intra-alveolar debris was seen in the form of groups of fibrils showing the characteristic periodic transverse striation of collagen.

PROSTHETIC TRACHEAL REPLACEMENT

JOHN BORRIE The principles governing successful tracheal reconstruction are reviewed. Over a 15-year experimental experience of reconstructing long tracheal defects (5 cm. or more), the methods of suturing alone, suturing with lateral relieving incisions, the use of ribbed Dacron prostheses, Marlex mesh, and one form of Silastic prosthesis have all been tried and rejected. More promising results from current research methods are shown.