had left nursing. Among those from the joint course still at work, health visiting was the most popular occupation, and on the whole they were earning a higher salary than those in the other groups; but it is a sad commentary on nurses’ scales of pay that only 5 of the 216 nurses studied were earning more than £1,000 a year.

Interplay of Learning

Havelock Ellis spent eight years as a medical student at St. Thomas’s Hospital before qualifying as a licentiate of the Society of Apothecaries, having failed to do so with the Conjoint diploma. He is said to have had a natural inaptitude for examinations despite preparing for them by hard work, but while a student he found time also to follow a literary bent. As well as contributing to magazines he edited the early volumes of the Mermaid series of Elizabethan dramatists. Could he have done this at St. Thomas’s (or any other medical school) today? To anyone passing from the Todd report1 to a report of a conference to discuss it held recently by the British Medical Students Association2 the question must seem as irrelevant as it is conjectural. Yet as well as being a great pioneer in the liberation of human thought and an attractive writer Ellis made a contribution to man’s welfare so highly regarded in a more specifically medical sense that he was elected F.R.C.P. under the special rule in 1936.

The report of the B.M.S.A. conference summarizes medical students’ opinions by saying that on the whole they are interested in the changes recommended by the Royal Commission “aiming at designing a more flexible course.” It may be that echoes from times past inspired this wish for rather more freedom of movement—which included also a recommendation that students should be able to transfer to another medical school of their own choosing for shorter or longer periods. But the report also says that there must be adequate supervision and thorough reappraisal of the material taught. In fact the students welcomed the idea of replacing the traditional form of examination by the much closer scrutiny entailed in continuous assessment, though some doubts about it were raised during the meeting.

The varying abilities of teachers to teach are a common topic of conversation among students and have often been examined in more academic fashion. But Dr. R. Mac Keith must have surmised the conference when he announced that of graduates attending courses on teaching 85% are medical, 10% are from other sciences, and 5% are from the arts. This statistic is probably one of the reasons why medical students are more contented with their lot than are the students of some other faculties. No doubt the sense of having a worthwhile goal is another, and a third in the clinical years is the relationship between teacher and taught, which happily still draws strength from old-fashioned apprenticeship in a task on which each is in a different way engaged, the care of patients.3 The honorary president of the B.M.S.A., Dr. Ian Sneddon, stressed in a letter to the meeting that “there is much still to be said for the apprenticeship methods.” Miss Josephine Barnes, a member of the Royal Commission (though speaking as an individual), said “the basic theme of the Todd report is to continue the traditional British idea of the student and young doctor as an apprentice.” And the students themselves clearly took this point in a present context when they stressed their desire for “student participation” and for the holding of regular meetings of “joint staff-student curriculum committees.” It is certainly a mistake to suppose, as some extremists in other faculties have contended, that students and teachers are equals engaged in a joint enterprise, for in one important respect students are not the equals of their teachers: that is in knowledge of the subject. Nor are they their equals in experience. But every good teacher is responsive to his students and learns something from them. Whether this lively interplay can survive regular meetings of curriculum committees is another matter, for it is evident from the increase in bargaining activities in universities that some people are determined to kill the spirit and preserve the letter.

On a variety of other matters the report gives the views of students who came to the meeting from many of Britain’s medical schools, and of some of their teachers too. Modestly presented by the organizing committee as a “record of student opinion on the main issues,” it deserves to be read for that reason.

Pulmonary Hypoplasia

Since descriptions by O. R. Swyer and G. C. W. James1 in 1953 and of W. M. MacLeod2 in 1954 there has been increasing recognition of a syndrome consisting of unilateral increased transradiancy of the lung, hypoplasia of the pulmonary blood vessels, and airways obstruction. It has been variously termed Swyer and James syndrome, MacLeod’s syndrome, unilateral pulmonary emphysema,1 unilateral hyperlucent lung,3 unilateral lung transradiancy,4 chronic obstructive pseudoemphysema,5 and acquired unilateral artery hypoplasia.6 The common feature of the cases in which the lung has been examined is hypoplasia of the affected pulmonary artery; and this can be diagnosed during life radiologically, with or without angiograms. Perhaps the simplest name for the syndrome might be pulmonary hypoplasia—which would indicate the type of defect that exists without specifying its cause.

Some circumpection is needed in arriving at a diagnosis of pulmonary hypoplasia. The possibility usually arises when a routine radiograph or one taken after recurrent pulmonary infections shows abnormal transradiancy of one lung field. The cause of abnormal transradiancy may lie in the chest wall—for example a kyphoscoliosis, absence of a breast, or defective pectoral muscles. There may be overdistension of

1. Swyer, O. R., and James, G. C. W., Thorax, 1953, 8, 133.
lungs secondary to atelectasis or after lobectomy. Partial bronchial obstruction from any cause may lead to air trapping. The pulmonary vasculature may be the site of congenital anomalies, or may be occluded by an embolus or thrombus. Occasionally, diminished radiolucency of the opposite lung fields from pleural thickening may cause confusion. Only when these possibilities have been eliminated can a diagnosis of pulmonary hypoplasia be made.

The characteristic radiographic signs are increased transradiancy of a lung or lobe, the affected part being either of normal size or somewhat reduced. The adjacent hilar vascular shadows are small. Screening shows that the volume of the hypoplastic lung remains virtually unchanged during respiration, and that there is a tendency for the mediastinum to move towards the affected side during inspiration. The bronchographic signs are variable and include spidery bronchi, poor peripheral filling, and irregular areas of bronchial dilatation. Angiography confirms the diminished blood flow with narrowing of the pulmonary artery and its branches. Respiratory function tests indicate airways obstruction, increased residual volume, and impaired gas mixing in the hypertranslucent lung.

The pathogenesis of pulmonary hypoplasia is still unclear. Most arguments are concerned with whether it is the blood vessels or the air passages that are primarily at fault—a congenital vascular defect or retarded development after a bronchopulmonary infection. Certainly, a history of a severe respiratory infection in infancy or childhood is often obtained, and the balance of evidence seems to favour more and more a failure of the immature lung to develop fully after such an infection as the most probable cause.

Treatment is usually not necessary, other than antibiotic therapy for pulmonary infections. Associated bronchiectasis may require surgical excision if it gives rise to recurrent infections or haemoptyses.

New Aspects of Human Genetics

It is a tribute to the vitality of human genetics that a second number of the British Medical Bulletin has been allotted to this topic after an interval of seven years. A comparison of the two numbers affords a useful review of some of the main advances in this time.

Two autosomal trisomies other than Down's syndrome (G trisomy) have now been clearly delineated, clinically and cytogenetically. These are Edwards's syndrome (E trisomy) and Patau's syndrome (D trisomy), which have incidences of the order of 1 in 5,000 and 1 in 9,000 live births respectively, whereas the incidence at birth of Down's syndrome is about 1 in 700. The E chromosome involved in Edwards's syndrome is probably 18, the D chromosome in Patau's probably 13. One syndrome due to a structural anomaly, the cri-du-chat syndrome, due to a deletion of the short arm of chromosome 5, is now also clearly delineated. Among the sex chromosome anomalies the remarkable XXY (extra Y) syndrome has been added to the XO (Turner's), the XXY (Klinefelter's) and the XXX syndrome. All but Turner's syndrome are common at birth, with incidences of 1 to 2 per 1,000 live births of the appropriate sex. Study of the chromosomes of foetuses aborted in the first trimester has now shown that infants with chromosome anomalies surviving to term are only the residue of a much larger group of abnormal zygotes. About half the spontaneous abortions in the first three months of pregnancy may have chromosome anomalies. Two of the commonest, present in perhaps 1% of zygotes, are triploidy with 69 chromosomes present, and an E trisomy which is probably of chromosome 16. Except in mosaics these appear almost always to cause spontaneous abortion.

The XO genotype appears to have the same order of zygote frequency, with only a small minority surviving to term. G and D trisomies are also not uncommon in abortions. In contrast the sex chromosome trisomies do not appear to predispose to spontaneous abortion.

In biochemical genetics one of the most interesting developments is the finding that multiple allelism—that is, the presence of numerous alternative genes at one gene locus—is a common situation. An excellent example is provided by the human haemoglobins, for many of which the amino-acid sequences have been worked out in detail. Amino-acid substitution can in most cases be interpreted as due to a single base change. Some amino-acid substitutions would be expected to, and do, make the haemoglobin molecule unstable, and so give rise to a congenital haemolytic anaemia. Some substitutions in the pocket of peptide containing the haem molecule cause methaemoglobinaemia. In the thalassaemias there is a failure in synthesis of either the α or the β chain of the haemoglobin molecule, but any α or β peptide that is synthesized is normal in composition. These could possibly be examples of "controller" gene diseases, but there are several other theoretical ways in which messenger-R.N.A. synthesis could be retarded. It is now known that in addition to the series of inborn errors of amino-acid metabolism due to a failure of synthesis of enzymes concerned in metabolic pathways—exemplified by classical phenylketonuria—a second series arise from deficiencies in enzymes concerned in amino-acid transport. Two types of transport of amino-acids appear to exist; one which has a capacity well above the usual concentration of the amino-acid in plasma and which is usually shared by a group of related amino-acids; and one which has a capacity equivalent to the usual plasma concentration of its sole mate and is usually very specific. Deficiencies of the second mechanism may cause severe diseases.

Work on mapping the human autosomes is proceeding steadily with the help of computer programming. Two new methods, however, are being developed which will enable linkage groups to be assigned to individual chromosomes. One depends on the linkage of genes to chromosomes marked by morphological peculiarities. The first example is the linkage of the gene locus for the Duffy blood type to chromosome 1 by means of the secondary constriction present in a proportion of these chromosomes. The other new method depends on the use of mouse-man hybrid cells cultured in vitro. As such hybrid cell cultures divide the human chromosomes gradually drop out unless they are supplying a gene product needed by a mouse cell which has been selected for having a specific enzymatic deficiency. In this way the gene locus producing thymidine kinase has been shown very probably to be present on chromosome 17.

The field of polygenic inheritance was discussed in the earlier volume, mostly in relation to normal characteristics such as stature, general intelligence, blood pressure, and finger ridge counts. In this volume it is discussed in relation to a number of common malformations and diseases. The family patterns shown by such common conditions suggest that the genetic predisposition to them is polygenic with a threshold.

1. British Medical Bulletin, 1961, 17, No. 3.