tions to sit the membership examination are now higher than ever. A continuing injection of new blood and new ideas and a strong regional faculty organization should ensure that the College remains outgoing in its policies, sensitive to the needs of its wider membership and their patients, and yet able to discharge newly-acquired responsibilities in general practice.

1 British Medical Journal, 1952, 2, 1321.
5 Residencies of the College to the Royal Commission on Medical Education, Report from General Practice No. 5. London, Royal College of General Practitioners, 1966.
9 McKnight, J. E., Journal of Royal College of General Practitioners, 1971, 21, 315.
10 Journal of Royal College of General Practitioners, 1972, 22, 415.
11 British Medical Journal, 1972, 2, 245.
12 British Medical Journal, 1972, 4, 96.

Brown Spots

To physicians other than dermatologists the distinction between the freckle and the lentigo may seem to be at best of academic interest. Yet these two small brown spots are fundamentally different in structure, natural history, and associations. The freckle is induced by light in susceptible people and may fade if exposure to light is reduced. The melanocytes which form it are abnormally large. The lentigo is not influenced by light, tends to persist for years or indefinitely, and is formed of an increased number of apparently normal melanocytes. The development of a few or many lentigines during childhood or early adult life, apparently randomly distributed over the body surface, is so common as to be regarded as physiological. The term lentiginosis has been applied when lentigines are present in exceptionally large numbers or in a distinctive distribution.

The distribution of lentigines in a characteristic pattern in more than one member of a family has attracted the attention of dermatologists, and A. Touraine in 1955 reviewed the scattered literature. He recognized two distinct patterns. In one of these, which he had earlier described and named centrofacial lentiginosis,1 the lentigines appear in early childhood in a horizontal band across the centre of the face. Associated defects include confluence of the eyebrows, a high arched palate, spina bifida, and mental retardation. Inheritance is apparently determined by an autosomal dominant gene.

The second distinctive pattern of lentiginosis occurs mainly around the orifices, and the principal associated abnormality is polyposis of the gastrointestinal tract. This syndrome, also determined by an autosomal dominant gene, was first described by J. L. A. Peutz in 1921.3 In 1946 Touraine and F. Couder4 gave a detailed account of the syndrome and collected 31 cases. H. Jeghers and his colleagues in 1949 reported further cases;5 but the now familiar eponym Peutz-Jeghers syndrome shows a regrettable disregard for historical precedence.

Other patterns of lentiginosis have been reported in single patients or in two or more members of a family, but in many of the reports the clinical description is incomplete, and if associated defects in other organs were sought they were not mentioned. But it is now clear that widespread lentiginosis is a feature of at least one distinctive hereditary syndrome. That syndrome, for which the term progressive cardiomyopathic lentiginosis has been proposed, was first identified by E. J. Mounahan6 and has recently been critically reviewed and characterized by P. E. Polani and Mounahan,7 who have studied eight patients. Lentiginosis is present in infancy, but the number of lesions increases during childhood until many hundreds are present, irregularly scattered over the body. Hypertrophic obstructive cardiomyopathy, affecting predominantly the left side, may cause severe symptoms or be detected only on electrocardiography. Other features of the syndrome are impaired sexual development and stunted growth. In some cases there is also some degree of intellectual impairment.

The inheritance of this syndrome is determined by an autosomal dominant gene. Polani and Mounahan discuss the possible relationship between the two principal defects. The cardiomyopathy seems likely to be related to the lentiginosis because melanocytes which form the latter are derived from the neural crest, which also contributes to the structure of the heart. Less plausible is the hypothesis that a disorder of inotropic amines in the lentigines gives rise to abnormal cardiac function.

The relationship of cardiomyopathic lentiginosis to other reported syndromes with profuse lentiginosis has not yet been established. For example, O. F. Hornstein and F. Weidner8 have recently reported a further case of the association of lentiginosis with congenital deafness, which A. J. Capute and colleagues9 had described in 1969. The patients described by R. J. Gorlin and colleagues10 showed generalized lentiginosis, electrocardiographic abnormalities, pulmonary stenosis, and deafness. Inheritance was of autosomal dominant type, with variable expressivity. Other reports present different variations, with lentiginosis, cardiac defects, and deafness as the key features. Only the careful study of further families will elucidate the genetic and pathological problems raised by this association of defects. It is evident that any person presenting either generalized, centrofacial, or peri-orificial lentiginosis deserves thorough investigation.

Phoenix from Physical Medicine

In 1931 a decision was taken to amalgamate two sections of the Royal Society of Medicine—the Section of Balneology and Climatology and the Section of Electrotherapy. The name chosen to represent the interests of members of these sections was “Physical Medicine,” and from this originated a new specialty under the same name.
The British Association of Physical Medicine was formed in 1943, and its early members pioneered the first clinics devoted entirely to the diagnosis and management of the rheumatic diseases. By virtue of the type of patient with which they dealt they became of necessity expert in techniques of rehabilitation. Some developed this as a major interest and in ensuing years made important contributions to the subject.

The term “physical medicine” has been the cause of some confusion in the past. In general, those who have been appointed under this title have had clinical rheumatology as a primary interest, but in most cases have also undertaken the organization, development, and administration of hospital rehabilitation departments. The diverse nomenclature under which such appointments have been advertised has added to the confusion—some as “Physical Medicine,” some as “Physical Medicine and Rheumatology,” and others as “Physical Medicine and Rehabilitation.”

The advent of the Joint Committee on Higher Medical Training has done much to clarify the situation, since it has been accepted that there should be a single specialist advisory committee on rheumatology which will advise on the training necessary for accreditation in the clinical field of connective tissue diseases and medical disorders of the locomotor system. This committee has also agreed to concern itself with the training of those rheumatologists whose future interest will lie predominantly in rehabilitation.

It is clear that the term “physical medicine” is obsolete in the context of modern medicine. Accordingly, at a meeting held on 12 October, at the Royal College of Physicians of London, a resolution was adopted to dissolve the British Association of Physical Medicine and Rheumatology and to form a new association called the British Association for Rheumatology and Rehabilitation. This association will welcome into its membership and present the interests of doctors whose primary concern is clinical rheumatology, those who in addition to this take responsibility for hospital remedial departments, and those whose predominant interest lies in rehabilitation.

The impact of the recently published Tunbridge Report on rehabilitation has yet to be assessed. Among its important recommendations are that every district general hospital or district group of hospitals serving a community of 200,000 should have a general rehabilitation department of 100 places and that there should be a consultant in charge of such departments who should devote a substantial part of his time to this work. The report accepts that there would be an advantage in such a consultant having his own clinical field, that he could be drawn from any specialty, but that “in the immediate future the most likely source of such heads of department will be from the ranks of consultants in physical medicine and rheumatology, many of whom are already working in this field.”

Rehabilitation is seen to be one of the growing points of medicine, but the principle should not be forgotten that all consultants, whatever their discipline, should take ultimate responsibility for the rehabilitation of their patients. It would seem essential that if more consultants are to be appointed to direct hospital rehabilitation departments they should have a defined clinical interest of their own in order to ensure recruitment of men of suitable calibre. In addition it is to be hoped that this type of appointment would go some way to allay the growing apprehension of the remedial professions that a consultant whose sole interest lay in rehabilitation might undermine their own professional status.

The new British Association for Rheumatology and Rehabilitation will clearly have a leading role in delineating and advising on problems affecting the rheumatic sufferer and the rehabilitation of the physically disabled.

2 British Medical Journal, 1972, 1, 526.

Genetics of Hypospadias

Like most common congenital malformations hypospadias may be caused in several different ways. In most cases it is not associated with other malformations, apart perhaps from cryptorchidism and inguinal hernia. A small minority of these uncomplicated cases are the result of the administration of progesterin to the mother in the first trimester and another small minority to sex-chromosome anomalies. But the majority probably have multiple causes comprising both a polygenic predisposition and unknown environmental factors. In a different and small proportion of cases the hypospadias is just one part of a syndrome and the causation is that of the syndrome. Examples are X-linked Reifenstein’s syndrome (familial hypogonadism), recessive Smith-Lemli-Opitz syndrome (microcephaly and other defects), recessive adrenal hyperplasia, and Down’s syndrome, or mongolism (trisomy 21).

In an early, large-scale and thorough family study in Denmark H. R. Sørensens showed that about 10% of brothers of index patients were also affected. This is some 30 times the frequency in the general population of about 3-3 per 1,000 live births. The frequency in second- and third-degree relatives in this series was not reliably established but appears not to have been inconsistent with polygenic inheritance. The findings in relation to twins in this series combined with those in the Paris series of M. Lamy were likewise compatible with polygenic inheritance and a fairly high heritability. Sørensen’s study was too early to include chromosome tests on the patients.

A more recent study by D. Aarskog from Bergen has evaluated the contribution of progesterin and of sex chromosome anomalies. In a series of 80 patients five cases were apparently due to the intrauterine action of progesterin and five to an anomaly of the sex chromosomes. In three of the latter patients there was XX/XY mosaicism and in two XO/XY mosaicism, the XY clone being in a minority in all five patients. These five patients were among those with a severe scrotal or perineoscrotal form of hypospadias. Aarskog did not make any family study of his patients. In a recent study on 50 patients, from Michigan, Y. C. Chen and P. V. Woolley found that of 26 patients studied cytogenetically two had a sex chromosome anomaly, one boy having an extra Y chromosome and a translocation probably between chromosome 8 and 12, and one XX/XY mosaicism. This is consistent with the cytogenetic findings of the series from Bergen. In the Michigan study six brothers were found to be affected among 62, a finding close to the 10% in Sørensens’s series. The Michigan workers point out that, on the assumption of a multifactorial aetiology, this is compatible with the view that inheritance represents about 75% of the causation of the defect.

For genetic counselling of parents of an affected child