and in the level of blood urea, while the salt-retaining effects may aggravate the oedema and lead to hypertension. In a recent controlled trial[9] of steroid treatment versus symptomatic treatment alone the early mortality was higher among the patients who received steroids, emphasizing that even the modest doses of steroids given were potentially dangerous. It may be argued that doses of about 20 mg. prednisolone daily serve only to suppress adrenal function and that doses of 60 mg. daily for several months are necessary to produce an effect on the renal disease. Such regimens are correspondingly more dangerous, and no controlled trial has been published that investigates their value. There is some evidence[10] that the administration of a double dose of steroids on alternate days is less toxic but as efficacious, and it is reasonable to give large doses in this way. Recently M. Friedman and L. B. Strang[11] have shown that corticotrophin (A.C.T.H.) produces less stunting of growth for a given effect than does prednisolone. However, the problems of hypertension and a cushingoid appearance remain.

Owing to the limitations of steroid therapy interest has grown in the cytotoxic drugs. Azathioprine,[12] cyclophosphamide,[13 14] and chlorambucil[15] have all been used extensively, and there can be no doubt that cyclophosphamide and chlorambucil are helpful in the treatment of the patient with a minimal renal lesion. The value of azathioprine is less certain. M. Abramowicz and his colleagues[16] concluded that, at least in children and in fairly low dosage, it was no better than a placebo. Remissions induced with cyclophosphamide last longer than those induced with steroids,[17 18] and many patients who were previously dependent on steroids now appear to have been cured and to require no further treatment. There is some doubt about the best therapeutic regimen. M. W. Moncrieff and his colleagues[14] have induced a leucopenia (and usually alopecia) with a high initial dose and then maintained the leucopenia for about three months with a relatively small dose. On the other hand, J. F. Soothill[17 18] has found that the continuous administration of an intermediate dose for two months produces a remission without inducing either leucopenia or alopecia. Relapse occasionally follows withdrawal of treatment, and in some cases it is related to inadequate therapy.[14]

Given to patients with proliferative glomerulonephritis, azathioprine has occasionally produced a dramatic response.[12] Relatively high and dangerous doses (4-6 mg./kg. body wt. per day) have been necessary, and recently there have been attempts to use smaller doses combined with modest doses of steroids. A small controlled trial[19] of this method of treatment in adults showed slight benefit that was just significant statistically, and a much larger trial at present in progress under the supervision of the Medical Research Council has led to the same conclusion. There have been no controlled trials of cyclophosphamide in the treatment of proliferative glomerulonephritis, though occasional patients, especially children, do seem to respond.

Patients with membranous nephropathy run a downhill course over many years. During this time their proteinuria fluctuates and occasionally their urine becomes free of protein for a period. The underlying lesion persists, however, and it is generally considered that neither steroids nor cytotoxic drugs affect the course of the disease.[20] Occasional reports[21 22] challenge this view, and recently S. P. Rastogi and colleagues[22] reported on four patients who were given prolonged courses of steroids and lost their proteinuria for periods up to 6·5 years. Repeated renal biopsies showed that the histological lesion persisted but had not progressed significantly.

Pericarditis and Rheumatoid Arthritis

Patients with systemic lupus erythematosus may present with rheumatoid arthritis, and patients with unequivocal rheumatoid disease may develop the serological changes of systemic lupus erythematosus. But the great majority of patients show the lesions of only one of these diseases, and the frequency of their overlap has been exaggerated. It is therefore surprising that little attention has been paid to the pericarditis of rheumatoid arthritis when it is so well recognized as a feature of systemic lupus erythematosus.

Necropsy studies have given an overall incidence of pericarditis in association with rheumatoid arthritis as high as 30%[1-3] and this complication is recognized in about 7% of cases of juvenile rheumatoid arthritis.[4] A recent study by J. Kirk and J. Cosh of 33 patients with rheumatoid pericarditis is therefore of interest.[5] The condition must be taken seriously, for acute or chronic pericardial effusion, chronic constriction, and even haemopericardium with tamponade may supervene. The pathological changes found in most cases at necropsy are obliterative pericarditis, with acute fibrinous pericarditis making up almost all the remainder. Specific granulomatous infiltration is rare; when it is present the lesions are found on the epicardial surface of the heart as small necrotic nodules similar histologically to the subcutaneous rheumatoid nodule.[2 3]

The onset of pericarditis seems to bear no relationship to the duration of the arthritis, though it is particularly apt to be found in middle-aged males in whom the arthritis was of acute onset. It is perhaps commoner when the arthritis is active than when it is dormant. Serological tests for rheumatoid arthritis are usually positive in patients developing pericarditis, and one-third of these patients have typical rheumatoid nodules. Anaemia and a raised erythrocyte sedimentation rate merely reflect the activity of the arthritis.

On careful inquiry a history of chest pain can be obtained in about half the patients with pericarditis. The pain is frequently pleuritic and fleeting in nature, lasting only a matter of hours or a day or so. In some the pain may closely simulate that of cardiac ischaemia, but it is distinguished by alteration in intensity with changes in posture. The remaining half of these patients have no symptoms, and the condition is diagnosed on routine clinical examination.

The characteristic auscultatory finding of a pericardial friction rub may be absent, but it is heard in most patients at some period of their illness. The rub may be atypical and may be confined to systole, making differentiation from a systolic murmur difficult. This difficulty may be increased by the persistence of the rub unchanged for several months. Low-grade fever is frequently present and should alert the physician to seek this particular complication.

Confirmation may come from electrocardiographic changes, alteration of the cardiac outline on chest radiograph, or aspiration of pericardial fluid. The characteristic E.C.G. pattern in pericarditis is the raising of the ST segment without T wave inversion. Later, abnormalities of the T waves may appear, including inversion; they are found only when the

1 Bywaters, E. G. L., *British Heart Journal*, 1950, **12**, 101.
2 Goehrs, H. R., Baggenstoss, A. H., and Slocumb, C. H., *Arthritis and Rheumatism*, 1960, **3**, 298.
3 Lebowitz, W. B., *Annals of Internal Medicine*, 1963, **58**, 102.
4 Leitman, P. S., and Bywaters, E. G. L., *Pediatrics*, 1963, **32**, 855.
5 Kirk, J., and Cosh, J., *Quarterly Journal of Medicine*, 1969, **152**, 397.
6 Hart, F., *British Medical Journal*, 1966, **2**, 131.
7 Harrold, B. P., *British Medical Journal*, 1968, **1**, 290.

pericarditis is active. Sometimes the pericarditis is present without any E.C.G. changes.[5]

About half the patients reported on by Kirk and Cosh also had rheumatoid lung or pleural lesions.[5] Pleural effusion is common, but pericardial effusion is rare. In the few cases in which pericardial effusion occurs the fluid closely resembles the straw-coloured non-viscous fluid found in the pleural cavity; typically it is high in protein but low in sugar. L.E. cells are found in the blood in a minority of patients,[6] but even then the diagnosis of rheumatoid arthritis as opposed to systemic lupus erythematosus is usually clear. To assess the incidence of rheumatoid pericarditis Kirk and Cosh made a prospective study of 100 inpatients with severe rheumatoid arthritis selected at random. They found it was 10%. But their patients had sufficiently severe disease to warrant admission to hospital, and so high an incidence would not be expected in patients outside hospital.

The prognosis of rheumatoid pericarditis is generally good. The condition appears to run a benign course, usually with rapid spontaneous resolution. Specific therapy does not prevent its onset or shorten its course once established. If pericardial effusion occurs, early aspiration is advised to prevent tamponade and to reduce the risk of later pericardial constriction. B. P. Harrold[7] reviewed 17 reported patients with rheumatoid pericarditis who had undergone pericardiectomy for constriction and noted that in only three did the interval between onset of arthritis and operation exceed five years. This suggests that if constriction is going to occur it is unlikely to do so in patients with arthritis of long duration. Rarely, heart failure may be the presenting symptom of rheumatoid pericarditis, and the onset of oedema in a patient with rheumatoid arthritis should prompt the doctor to consider this possibility.

Children's Eyes

Errors of refraction are almost universal, and few of us attain old age without recourse to spectacles. Yet, though the refractive state of the eye is simply the product of physical variations in the eye's anatomy which admit an exact measurement, our knowledge of these measurements in substantial numbers of the population at different ages has been very incomplete. A recent comprehensive report by Arnold Sorsby and G. A. Leary[1] is welcome not only because of the information it provides on the patterns of refractive changes in growing children but also because it helps to clear away some of the myths that are still being reported about the prophylaxis and "treatment" of short-sightedness.

The infant's eye is normally hypermetropic (long-sighted), and as the eyeball grows in length the hypermetropia decreases. The eye thus becomes more normal-sighted or even short-sighted until growth comes to an end by the age of 14. Between the ages of 3 and 14 the eye becomes about 1·2 mm. larger. But some 60% of the potential decrease in hypermetropia (or increase in myopia) that this elongation could be expected to produce is eliminated by a simultaneous reduction in the converging power of the cornea and lens, so that the resultant change in refraction is little more than 1 dioptre. And, as most infants are more than 1 dioptre hypermetropic, only exceptionally does this trend produce a frank myopia, and that of quite a low order. In about 28% of children a greater elongation of the eyeball during this growth period does cause a further shift towards myopia than can be compensated for by a slight additional decrease of the power of the cornea and lens. The change in cornea and lens is in part the direct sequel to this elongation and would itself tend to neutralize some of this adventitious myopia. The authors of the report found that bodily heights and weights were unrelated to the refraction at the beginning and end of the period of observation, and there were no obvious sex differences in these developments.

As the authors had shown in an earlier report,[2] the refraction and its components are genetically determined. And this, they state, must be assumed to apply also to the anomalous axial elongation and paradoxical changes in the cornea and lens. The provision of correct spectacles will thus have no influence on this predetermined refractive change[3 4] any more than on other organic disorders of the eyeball.

Caribbean Food and Nutrition Institute

Christopher Columbus made his first landfall in the Caribbean in October 1492 at an island in the Bahamas which he piously named San Salvador. Believing he had reached Asia, he imposed on posterity the confusing practice of calling the people of the New World "Indians" and the Caribbean islands the "West Indies." The area was at that time inhabited by the gentle Arawaks and the fierce Caribs, who gave us the word "cannibal." The Arawaks were soon exterminated by the Europeans who followed Columbus; the Caribs held out somewhat longer, and a few thousand survive today.

In the sixteenth and seventeenth centuries the islands were appropriated by European powers—the English, French, Dutch, and Spanish. They became sugar islands, devoted to sugar plantations worked by negro slaves captured in West Africa and carried across the Atlantic in the dreadful circumstances of the middle passage. After emancipation in the nineteenth century the sugar industry declined because the ex-slaves hated the sight of the sugar cane, which was being grown in increasing quantities in other parts of the tropics, and the sugar beet, efficiently cultivated in the temperate zone, began to rival the cane as a source of sucrose. During a long period of economic depression the Caribbean territories faded out of history, becoming of little importance to their European owners. Partial economic recovery came only recently, with the development of new industries such as bauxite manufacture, and the discovery, by wealthy Canadians and Americans, that the Caribbean offers an escape from winter blizzards and has lovely scenery. The tourist industry has boomed. During the last decade many of the Caribbean territories have become independent members of the United Nations.

This, roughly, is the setting of the Caribbean Food and Nutrition Institute, established in 1967. The institute has centres in Jamaica and Trinidad, each located in the campus of the University of the West Indies. At present its activities

[1] Sorsby, A., and Leary, G. A., *Medical Research Council. Special Report Series*, 1970, No. 309.
[2] Sorsby, A., Sheridan, M., and Leary, G. A., *Medical Research Council. Special Report Series*, 1962, No. 303.
[3] Morgan, O., *British Medical Journal*, 1970, **1**, 175.
[4] Gilkes, M. J., *British Medical Journal*, 1970, **1**, 758.

[1] *Journal of Tropical Pediatrics*, 1968, **14**, 52.
[2] Waterlow, J. C., Cravioto, J., and Stephen, J. M. L., *Advances in Protein Chemistry*, 1969, **15**, 131.