steroids,[5] and there is good evidence that they do increase the muscle mass in castrates of both sexes and in most patients with a high rate of protein catabolism, but critical studies confirming these effects in normal men on an optimal protein and calorie intake are lacking. In a carefully controlled trial W. M. Fowler and colleagues[6] failed to find any effect of an anabolic steroid on the physical performance of young healthy men, even in a group taking extra physical exercise along with the drug. It might be argued that the physical exercise used in this study was not severe enough to compare with that undertaken by athletes preparing for international competition. If that were so it is possible that anabolic steroids combined with a high-protein diet might have an anticatabolic effect under conditions of severe muscular exercise, and hence produce an increase in physical performance.

It is difficult to detect whether a patient is taking these preparations, because the doses used might be small, and in any case adequate methods for detecting these drugs and their metabolites in blood and urine have not been fully evaluated. Since any effect they may have on muscular development will be prolonged, athletes can stop taking the drugs several weeks before a competition, so that any chance of detection then would be negligible. Secondary effects of the anabolic steroids on serum enzyme levels or on the level of thyroxine-binding globulin,[7] with lowering of the plasma protein-bound iodine, might be more prolonged, but further studies are required to clarify their duration. It is to be hoped that all the governing bodies of sports will take a stand against the use of such methods of training.

Mismanagement of Non-disease

Some two years ago C. K. Meador[1] wrote an amusing but important article entitled "The Art and Science of Non-disease," drawing attention to common errors of diagnosis in the endocrine field, with resulting treatment of nonexistent disease. Some of the mistakes were due to laboratory errors. "Laboratory error syndromes," he wrote, "are the delight of the alert non-disease clinician; one must add, however, only before therapy has been initiated. In this regard they are probably the most responsive to treatment of all entities. One merely has to repeat the test once to see astounding results; for example, in what other non-disease or disease can one see a rise in the haemoglobin level from 6 to 14 g./100 ml. in two hours with only one iron tablet?"

Recently A. B. Bergman and S. J. Stamm[2] have discussed morbidity of cardiac non-disease in schoolchildren. They studied 93 children who were said to have had rheumatic fever or to have "something wrong with the heart." Seventy-five (81%) had nothing wrong with them—and thirty of these had been subjected to severe restrictions on the instruction of their doctor. Eighteen had organic heart disease, and six of them had been put under severe but unnecessary restrictions. They wrote that "Restriction of children with innocent murmurs is only slightly more absurd than restriction of the vast majority of children with an organic heart defect"; and "The diagnosis of heart disease may cause profound changes in the functioning of a child and his family, far beyond the immediate physical effects." On the basis of their study of these 93 children they concluded that "The amount of disability from cardiac non-disease in children is estimated to be greater than that due to actual heart disease." They rightly criticize the restrictions imposed on children with congenital or acquired heart disease, advocating that they should be left to find their own level of activity of which they are capable. Parents fear that their child will drop dead if allowed to cry or to exert himself in other ways. In fact, as the writers state, sudden death is rare except in children with aortic stenosis.

Functional murmurs are exceedingly common in children. R. W. Quinn and E. S. Campbell[3] found them in 44% of 4,039 Nashville schoolchildren. Unfortunately it is not easy to describe in words the difference between a functional murmur and the murmur of organic disease. The distinction is a matter of experience. In general, the functional murmur is louder when the child is lying down, it is increased by exertion, and it has a musical, twanging nature, or is very soft. Murmurs in the newborn period frequently suggest serious congenital heart disease to the unwary, only to disappear in a few days. The venous hum, the to-and-fro murmur beneath the clavicle, can easily lead the doctor towards the diagnosis of a patent ductus arteriosus, while the true nature of the murmur could be readily established by causing its disappearance on rotating the head or by pressure on the vessels of the neck. The tachycardia of excitement or nervousness and the raised systolic blood pressure under the circumstances of the doctor's examination can likewise lead to the wrong diagnosis of cardiac disease.

Now R. S. Illingworth,[4] of Sheffield, has tried to dispel "Fallacies in Four Fevers." Discussing measles, German measles, chicken-pox, and mumps, he questions the value of some of the time-honoured methods of managing children with these infections and quotes with approval N. L. Browse's remark that "the bed is often a sign of our therapeutic inadequacy."[5] Certainly many doctors as well as parents would agree with Illingworth that too long confinement to bed of children with these fevers can do actual harm and give no benefit. He is likewise critical of the isolation and medication of these patients on principles that may be long accepted but have escaped careful scrutiny. When prescribing treatment, he says, one should always ask three questions: What good may it do? What harm may it do? What harm may be done by not giving this treatment?

Oesophageal Function in Diabetes

Among the better-known neurological complications of long-standing diabetes are peripheral neuropathy, impotence, diarrhoea, and postural hypotension. The exact nature of the neurogenic defect is still a matter of debate, and the effects are often complex and are not necessarily clinically apparent. In the gastrointestinal tract, for example,[1 2] symptomless gastric atony can result in a radiological appearance similar to that produced by vagotomy,[3] whereas diarrhoea in some patients may be associated with steatorrhoea or with changes in bowel flora.[4]

[1] Meador, C. K., *New Engl. J. Med.*, 1965, **272**, 92.
[2] Bergman, A. B., and Stamm, S. J., ibid., 1967, **276**, 1008.
[3] Quinn, R. W., and Campbell, E. S., *Yale J. Biol. Med.*, 1962, **34**, 370.
[4] Illingworth, R. S., *Brit. med. J.*, 1967, **3**, 41.
[5] Browse, N. L., *The Physiology and Pathology of Bed Rest*, 1965. Springfield, Ill.

[1] Ellenberg, M., *Adv. intern. Med.*, 1964, **12**, 11.
[2] Katz, L. A., and Spiro, H. M., *New Engl. J. Med.*, 1966, **275**, 1350.
[3] Wooten, R. L., and Meriwether, T. W., *J. Amer. med. Ass.*, 1961, **176**, 1082.
[4] Malins, J. M., and French, J. M., *Quart. J. Med.*, 1957, **26**, 467.
[5] Rundles, W. R., *Medicine*, 1945, **24**, 111.
[6] Mandelstam, P., and Lieber, A., *J. Amer. med. Ass.*, 1967, **201**, 582.

Rather surprisingly, little attention has been paid to the oesophagus. W. R. Rundles,[5] in a classic review of diabetic neuropathy, mentioned the finding of degenerated nerve plexuses in the oesophagus of two patients, but there has been no detailed investigation of oesophageal function. A recent report[6] shows that oesophageal abnormalities may in fact be fairly common. Fourteen patients with evidence of diabetic neuropathy were examined by cineradiography, and 12 had evidence of dysfunction. Peristalsis was reduced or absent, emptying was delayed, the oesophagus was dilated, and tertiary contractions were seen. Two patients had oesophageal reflux and hiatus hernia. No abnormalities were found in 14 matched control subjects. In spite of the frequency of radiological findings, only three patients had symptoms, consisting of heartburn in one and dysphagia in two.

These results are similar to previous investigations of other parts of the gastrointestinal tract, in which a high incidence of radiological abnormalities contrasts with the paucity of clinical symptoms. In addition, there was no correlation with the degree of diabetic control, with other evidence of visceral neuropathy, or with other complications of the disease. One patient with severe dysphagia died, but the oesophageal nerve plexuses appeared normal. The enigmatic nature of diabetic neuropathy, in which disorders of function may be found without either recognizable pathological changes or the production of symptoms, presents a challenge to the physician. In the case of the oesophagus further insight might be provided by the use of intraluminal manometry.

Ventricular Aneurysm

For the past quarter of a century radiologists have recognized that after myocardial infarction there are commonly abnormalities of ventricular contraction. These have been reported in as many as 70% of an unselected group of patients with both old and fresh myocardial infarcts,[1-3] and may take the form of paradoxical pulsation of a localized segment of the cardiac silhouette or of diminished or absent pulsation. Serial kymographic studies have shown that in some patients these abnormalities disappear or become less marked with healing of the infarct.

The advent of cineventriculography has made it easier to study disturbances of ventricular contraction. Using this technique R. Gorlin and his colleagues[4] found abnormalities of contraction in about 20% to 25% of patients with coronary artery disease who were being assessed before the operation of implantation of an internal mammary pedicle. In Gorlin's study the term ventricular aneurysm was used to describe abnormalities of local movement of the ventricular wall, whether or not these were associated with thinning or fibrosis. This definition of aneurysm is mechanical rather than anatomical and is probably too great an extension of the traditional meaning of the word. A more acceptable definition is that of J. Schlichter and his colleagues,[5] who defined a cardiac aneurysm as a localized outpouching of the cavity of a cardiac chamber with or without outward bulging of the external surface—in either case a large transmural infarct leads to thinning of the left ventricular wall and consequent paradoxical pulsation. If Schlichter's definition of aneurysm is accepted a different term will be needed for the reversible abnormalities of ventricular contraction without a full thickness infarct described by Gorlin and his coworkers.

Abnormalities of ventricular contraction after myocardial infarction can usually be recognized at the bedside. Careful palpation of the cardiac impulse will detect the development of a sustained abnormal impulse appearing during the first week after acute cardiac infarction in about one-fifth of patients.[6-8] Recognition of this simple physical sign can be made without the aid of graphic recording by simultaneous palpation and auscultation. If a sustained outward impulse is felt, lasting up to the time of the second heart sound, this is abnormal. Where this sign persists after acute cardiac infarction strong presumptive evidence of a ventricular aneurysm exists, provided there was not already left ventricular hypertrophy due to co-existing hypertension or aortic valve disease. The site of greatest excursion of the abnormal impulse varies with the site of the aneurysm. If it is anterior the impulse is usually greatest somewhere over the midpraecordium. If, however, the aneurysm is postero-lateral then the abnormal impulse is usually confined to the apex. The surest evidence that the sustained impulse is related to the infarction rather than to previous left ventricular hypertrophy is observation of its development during the first week after acute infarction. Graphic records confirm the abnormally sustained nature of the impulse and usually show a double hump due to a large palpable preceding atrial beat. Apex cardiography is an unreliable method of recording late systolic events, as it is influenced by the pressure with which the pick-up head is applied to the moving chest wall; but absolute displacement techniques such as impulse cardiography[9] and kinetocardiography[10] give faithful records. When the diagnosis of cardiac aneurysm has been suggested by the presence of an abnormal cardiac impulse, it can be confirmed by fluoroscopy and electrocardiography. Angiocardiography is of value in detailed preoperative investigation.

D. L. Abrams and his colleagues[11] in a review of 65 consecutive cases of ventricular aneurysm at necropsy recommended a conservative approach to treatment. Their main indications for ventriculoplasty were progressive heart failure refractory to medical therapy, where coronary arteriography showed the blood supply to the rest of the ventricle was adequate; and repeated systemic embolization that could not be controlled by treatment with anticoagulants. The highest mortality from cardiac aneurysm is within the first year, and is caused by intractable heart failure. If patients survive this period the prognosis does not appear to be very different from that of ischaemic heart disease in general.

[1] Dack, S., Sussman, M. L., and Master, A. M., *Amer. Heart J.*, 1940, **19**, 464.
[2] Master, A. M., Gubner, R., Dack, S., and Jaffe, H. L., ibid., 1940, **20**, 475.
[3] Dack, S., *Dis. Chest*, 1955, **27**, 282.
[4] Gorlin, R., Klein, M. D., and Sullivan, J. M., *Amer. J. Med.*, 1967, **42**, 512.
[5] Schlichter, J., Hellerstein, H. K., and Katz, L. N., *Medicine (Baltimore)*, 1954, **33**, 43.
[6] Mourdjinis, A., Olsen, E., Taubman, J., and Mounsey, J. P. D., *Brit. Heart J.*, 1966, **28**, 430.
[7] Mounsey, J. P. D. *Cardiologia (Basel)*, 1966, **48**, 203.
[8] Mourdjinis, A., Olsen, E., Raphael, M. J., and Mounsey, J. P. D., in press.
[9] Beilin, L., and Mounsey, J. P. D., *Brit. Heart J.*, 1962, **24**, 409.
[10] Eddleman, E. E., Willis, K., Reeves, T. J., and Harrison, T. R., *Circulation*, 1953, **8**, 269.
[11] Abrams, D. L., Edelist, A., Luria, M. H., and Miller, A. J., ibid., 1963, **27**, 164.

The next session of the General Medical Council will open on Tuesday, 28 November, at 2.15 p.m., when the president, the Rt. Hon. Lord Cohen of Birkenhead, will take the chair and will deliver an address.