immediately treated. The Denis Browne splint offers an easy and entirely acceptable approach to the problem of keeping the child in the corrected position. It is light; easily manageable by the mother; the child can be picked up and carried as in a portable crib; the child seemingly enjoys his position and placement in this splint; and there is no problem such is found in placing these children into a cast or even a Milwaukee brace.

Our approach, therefore, to the management of the child with idiopathic or other forms of scoliosis is to use our best judgment and avail ourselves of all the modalities of treatment which have been found to be useful. I place the Denis Browne splint in this category, and I would further say that if there is any form of treatment that should be carried out routinely on any infant with postural or structural curvature of the spine the Denis Browne splint offers the only logical means of handling this problem. While in many cases the treatment may be superfluous and unnecessary it certainly does no harm either to the emotions of the mother, the child, or to anyone else. In scoliosis I have the strong feeling that it is better to overtreat rather than to undertreat. I therefore agree with Sir Denis Browne's concepts of proper management of the infantile scoliotic, even though in many instances it might be postural and spontaneously resolving.—I am, etc.,

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Prevention of Heart Disease

SIR,—May I comment on the paper by Dr. M. F. Oliver and Professor C. H. Stuart-Harris (20 November, p. 1203) with regard to rapid gain in weight and hypercholesterolaemia as risk factors in ischaemic heart disease.

In April this year four members of a Dublin cricket club, all males between the ages of 29 and 37, expressed their desire to lose weight by way of a "crash diet." A diet providing 500 Cal./day had been devised for them for a period of 10 days. Although the importance of exercise was pointed out, they could not make themselves free for more than two occasions during the 10 days, when it was decided to "jog" around the club grounds eight times (total distance about two miles—3.2 kilometres).

Only two of the four were able to go through with the programme of diet and exercise as planned; subject No. 1, a 29-year-old man who put on 18 lb. (8.1 kg.) during the previous winter, and subject No. 2, a 34-year-old man who gained 15 lb. (6.8 kg.) in the same time. Both were moderately overweight according to the Metropolitan Life Insurance Company standard weights and height classification.

Serum-cholesterol levels were estimated before the start of the diet and thereafter every other day for the whole 10 days. The first subject lost 9 lb. (4.1 kg.) and the second 9.5 lb. (4.3 kg.). The series of cholesterol levels (mg./100 ml.) were as follows:

Subject No. 1: 195, 193, 218, 247, 286, 289.
Subject No. 2: 180, 182, 215, 264, 278, 273.

Two weeks later, when they were proceeding with a normal but still somewhat carbohydrate-reduced diet, the readings were as follows: No. 1—190; No. 2—187.

Without trying to draw generalized conclusions from such a limited study, could it be possible that a rapid weight loss by way of fat mobilization might supply even more lipids for arterial deposition than gain in weight?—I am, etc.,

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Heart Failure in Old Age

SIR,—I read with interest Dr. A. K. Thould's paper on "Coronary Thrombosis in the Aged" (6 November, p. 1089) and also your editorial "Heart Failure in Old Age" (p. 1076) of the same issue.

The following figures from a survey of 1,713 post-mortems in patients aged 70 and over during the years 1950–63 show that cardiovascular disease accounted for 543 deaths, of which 138 were caused by coronary thrombosis (females 63, males 75). Ruptured heart occurred in 12 cases, the oldest being a male aged 93, and one was syphilitic in origin. Seventeen patients in this total of 138 were diabetics, and 22 had carcinomata of various sites.

Haematemesis and melaena from gastric and duodenal ulcer was probably the precipitating cause in 5 cases, while 71 other major pathological findings were recorded amongst the remainder. Pomerance[1] draws attention to the multiplicity of pathological heart conditions in the aged, and among a variety of lesions found in my group there were 14 cases of subacute bacterial endocarditis.

With reference to Dr. Thould's remarks on the frequency of coronary thrombosis in relation to cerebrovascular accidents as a reason for admission, when it comes to post-mortem this series shows 152 cases of cerebrovascular accidents (male 45, female 107), which agree closely with those of Howell,[2] and Howell and Piggot,[3] and Stewart Smith.[4]

May I suggest that when considering diseases of old age post-mortem findings perhaps give more consistently accurate statistical estimates than clinical impressions.—I am, etc.,

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[1] Pomerance, A., *Brit. Heart J.*, 1965, **27**, 697.
[2] Howell, T. H., *Geriatrics*, 1949, **4**, 281.
[3] —— and Piggot, A. P., ibid., 1950, **5**, 90.
[4] Smith, G. S., *Lancet*, 1950, **1**, 24.

Cardiac Arrest in Myocardial Infarction

SIR,—We read with interest your editorial on cardiac arrest following acute myocardial infarction (13 November, p. 1135). Since the introduction of an intensive-therapy unit at this hospital we have been attempting to classify the severity of each case of myocardial infarction on admission. Each case had been assessed according to the prognostic index of Peel *et al.*,[1] and also had an arterialized venous sample of blood estimated for biochemical estimation of base deficit.[2] In a series submitted for publication 50 consecutive cases were analysed. Of these 20 cases had a prognostic index of less than 13 with six deaths, and the remaining 30 cases had a prognostic index of greater than 13 with seven deaths. No case with a prognostic index of less than eight died.

When we analysed the arterial sample for base deficit 33 cases had a significant base deficit in which there were 12 deaths. Only one case who failed to show a significant base deficit died (this is significant to 5%).

It would appear, therefore, that by the estimation of base deficit we have a rapid, easy, and accurate method of ascertaining the severity of cases suffering from myocardial infarction and, therefore, those which are more likely to benefit from intensive therapy.—We are, etc.,

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[1] Peel, A. A. F., Semple, T., Wang, I., Lanchester, W. M., and Dall, J. L. G., *Brit. Heart J.*, 1962, **24**, 745.
[2] Astrup, P., Jorgensen, K., Andersen, O. S., and Engel, K., *Lancet*, 1960, **1**, 1035.

Evanescent Parotitis in Diabetes

SIR,—A temporary condition of painful parotitis has occurred in some 20 of my diabetics. As I see only diabetics I do not know if this also occurs in non-diabetics, but I have noted no reference to such a condition in the general medical literature nor is there mention of it in the 10th edition of Joslin's book.[1]

The patients usually complain of earache, sometimes unilateral but usually bilateral, with an obvious swollen and tender parotid gland. In those who develop the condition it is usually brought on by some stimulating taste, such as sucking a lemon, and exacerbated by strong-tasting food. I remember a public-school boy who had his holidays spoiled by the better-tasting food at home, which kept his parotid painful. He has now grown out of the condition, as do all the sufferers ultimately. There has been no apparent sepsis in the mouths of these patients nor in the juice extracted from the duct of the parotid gland. I cannot explain the condition. Frequently, in my absence, the patients have been referred to an ear surgeon with supposed ear trouble.—I am, etc.,

London W.1. R. D. LAWRENCE.

REFERENCE

[1] Joslin, E. P., Root, H. F., White, P., and Marble, A., *Treatment of Diabetes Mellitus*, 1959, 10th ed. Lea and Febiger, New York.

Hypoglycaemia Due to Isolated Corticotrophin-deficiency

SIR,—We were interested in Dr. J. Gordon Sprunt's comments (30 October, p. 1064) on the use of methopyrapone in the assessment of pituitary-corticotrophin reserve. Unfortunately, plasma cortisol was not measured in our patient during the administration of methopyrapone. However, in common with the 17-ketogenic steroids, urinary total 17-hydroxycorticosteroids failed to increase either during or after the administration of methopyrapone.

Dr. Sprunt states that many would consider inadequate the dose of methopyrapone administered to our patient. We would then refer them to the paper by Buus *et al.*,[1] in which it was recommended that methopyrapone be administered two-hourly owing to its short biologic half-life; 3 g. of methopyrapone given as 250 μg. two-hourly beginning at noon was shown to produce