110 mm. Hg. She was confused with paranoid trends and exhibited hallucinations at times. Depression had also been noted by her relatives before she was admitted to hospital.

Physical examination showed osteoarthritis of both knee joints and small joints of the hands, confirmed by x-ray. Intravenous pyelogram showed normal appearance of both kidneys. Her haemoglobin was 6 g./100 ml. and the blood picture was hypochromic and microcytic, which improved after iron therapy. Blood sugar, electrolytes, proteins, and cholesterol were normal. There was no glycosuria, and urinary 17-ketosteroid estimation was within normal limits. X-ray of the skull showed well-marked hyperostosis frontalis interna (Fig. 1). As seen in Figs. 2 and 3, obesity was mainly limited to the upper arms and thighs.

Association of hirsutism, obesity, and hyperostosis frontalis interna has been observed under the title of "Stewart–Morel Syndrome" or "Morgagni–Morel Syndrome." Morel reported 17 cases and Stewart reported a few cases.[1] Michaux described a case in 1959 presenting mental symptoms along with this syndrome.[2] Aubertin described a patient with arterial hypertension.[3] All say that the case is obscure. The newly formed bone is deposited in the inner aspect of the frontal bones with considerable thicken-

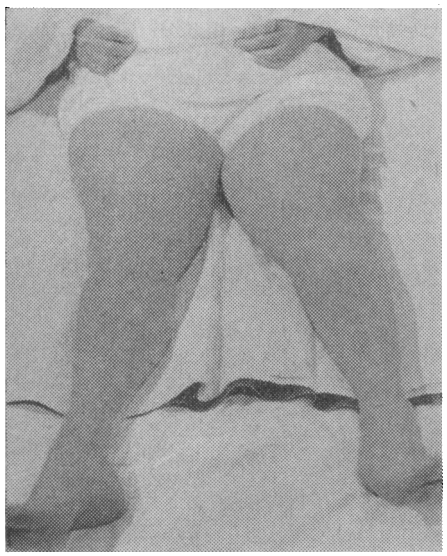

FIG. 3

ing—resulting in atrophy of frontal lobes and pituitary dysfunction—and has almost always been recorded in women.

Our patient presented all features of the syndrome—that is, hyperostosis frontalis interna, hirsutism, obesity, mental and nervous symptoms, and hypertension.—We are, etc.,

I. P. ROWLANDS.
B. C. DAS.

General Hospital, Warrington, Lancs.

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Hypogammaglobulinaemia in Adults

SIR,—Your special correspondent's account of my lecture on hypogammaglobulinaemia in adults (5 August, p. 362), given at the conference on disorders of protein metabolism at the Royal College of Physicians, London, contains some misquotations.

Hypogammaglobulinaemia in adults is of two types: primary, in which the deficiency of gammaglobulin is of unknown origin, and secondary, in which it is due to protein loss from the gut or kidneys or results from the effect of neoplasms upon immunoglobulin production. I did not say that the disease could be due to the toxic effects of drugs, and there is no evidence that hypogammaglobulinaemia can be caused in this way.

The eighteen patients whom I reported had all been diagnosed as having hypogammaglobulinaemia in adult life. The onset of the disease as judged by the history of recurrent infections was not, as stated in your report, always in adult life. In four of these adults the first onset of recurrent infections had occurred in childhood.—I am, etc.,

Brompton Hospital, London S.W.3. K. M. CITRON.

Cardiac Resuscitation

SIR,—As a lecturer and examiner in first aid for both Red Cross and St. John Ambulance Societies over the past nine years, I can substantiate Dr. J. D. Barrett's feelings (12 August, p. 437) that the indications for external cardiac compression are not understood by the majority of first-aiders who have only attended one or two courses. Often candidates state at examination that if the casualty does not respond by spontaneous breathing after the first four to six inflations of the lungs then external cardiac compression should be started. There is no thought of palpating the neck for pulsation, of observing the colour of the lips, or of noting the condition of the pupils. I feel that many casualties would be better with only basic first aid—that is, arrest of haemorrhage, ventilation of the lungs, and the treatment of cuts, burns, and fractures. I suggest that external cardiac compression should be taught to recognized "life-savers" who have passed at least two first-aid examinations and have shown aptitude in diagnosis, and not to those who have only just started.—I am, etc.,

Public Health Office, Swanage, Dorset. W. E. HADDEN.

Insulinotropic Action of Secretin

SIR,—Drs. D. R. Boyns, R. J. Jarrett, and H. Keen (10 June, p. 676) have confirmed the insulin releasing action of secretin in man but report evidence which they suggest throws doubt on the possible physiological role of this hormone in the regulation of insulin secretion. Some of our observations in studies related to this question and in studies of the effects of pancreozymin are at variance with theirs. In the earlier experiments of Boyns, Jarrett, and Keen with intraduodenal infusions of citric acid hypertonic solutions of glucose were delivered into the duodenum as a means of raising the blood glucose concentration.[1] With this procedure possible effects of the hypertonic solution on secretin release might obscure further effects of acidification of the mucosa. However, it is clear from the results of the three experiments reported in their recent paper that the infusion of 2.5 mEq of citric acid into the duodenum did not significantly modify the response to intravenous infusion of glucose.

We are in the course of a series of experiments in which hydrochloric acid is infused in the duodenum for a period of 20–40 minutes to a total dose of 30 mEq while glucose is given intravenously. This dose of acid does not exceed estimates of the normal acid secretion in response to a mixed meal.[2] In two out of three experiments distinctly higher levels of serum immunoreactive insulin were observed while the acid was infused into the duodenum; and in the third a difference in the same direction was recorded. Moreover, we have found that the intravenous infusion of synthetic human gastrin in six normal subjects modifies the response to intravenous glucose in the same manner as secretin, and it seems that this effect may be mediated by stimulation of acid secretion.

Our findings with a highly purified preparation of pancreozymin of verified exocrine activity differ from those of Boyns, Jarrett, and Keen. This preparation administered to eight subjects in doses of 25 to 50 μg. has consistently enhanced the rise in serum insulin associated with intravenous infusion of glucose and has accelerated glucose disposal. In fasting subjects little or no change in peripheral serum immunoreactive insulin is obtained when pancreozymin is administered intravenously, but a large transient rise in portal serum immunoreactive insulin has been observed. The same preparation of pancreozymin enhanced the change in serum insulin concentration associated with intravenous infusion of arginine in eight normal subjects. We believe that all preparations of secretin or pancreozymin must be tested for exocrine activity after use in experiments yielding negative results. Our findings will be presented at the forthcoming meeting of the International Diabetes Federation.

It is unlikely that duodenal infusion of isotonic glucose causes secretion of pancreozymin in man, and it has been shown by Dr. R. Preshaw at McGill[3] that such infusions do not produce exocrine effects of secretin in man. This procedure was used by McIntyre and his colleagues[4] to demonstrate enhanced insulin secretion in man during intestinal absorption of glucose. It appears, therefore, that an insulinotropic hormone other than secretin or pancreozymin is secreted when glucose is absorbed from the small intestine. However, the response to ingestion of protein together with carbohydrate suggests to us that stimulation of the endocrine pancreas is further potentiated to an extent not fully accounted for by the direct effect of circulating nutrients. The digestive secretagogues may be responsible for this potentiation.—We are, etc.,

J. DUPRÉ.
J. D. CURTIS.
J. C. BECK.

Fraser Laboratory for Research in Diabetes, Royal Victoria Hospital, Montreal, Canada.

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Mortality of the Ambulance Ride

SIR,—The letter from Mr. C. H. Cullen and others (12 August, p. 438) inadvertently underlines the great importance of taking seriously injured patients to the proper centre