instances of subarachnoid haemorrhage and of unexplained deaths.

This case, which will be presented in detail, supports Mr. Barabas's contention that there may exist an "arterial" type of syndrome. It provides a particularly florid example, with a similar illness occurring in milder form in a brother. Although usually regarded as an autosomal dominant trait,[1] the possibility of different modes of inheritance in a heterogeneous syndrome has always to be considered, and one might speculate that some cases of the "arterial" form might be transmitted as autosomal recessives. If this were so parents would not be expected to show the trait, and absence of family history could be compatible with this mode of inheritance, as in the case of Rubinstein and Cohen,[2] another of our own,[3] and number 27 of Barabas.

The features of systemic illness (fever, etc.) in our patient are not well explained and it may be noteworthy that they had occurred in milder form earlier in this patient's history. We wonder whether others have had similar experiences and whether more acute episodes of arterial damage may occur in the course of Ehlers–Danlos syndrome.—We are, etc.,

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Warning of Massive Pulmonary Embolism

SIR,—Dr. W. G. Smith (5 August, p. 370) rightly stresses the importance of early recognition and treatment of "warning" pulmonary embolus. This may not, however, be quite as usual as he suggests.

Coon and Coller[1] reported warning emboli in 25% of their cases of fatal pulmonary embolism, and Little, Lowenthal, and Mills[2] found that 7 of their 37 cases of fatal pulmonary embolism had evidence of warning embolus, an incidence of 19%.

A review of St. Thomas's Hospital records for the years 1959–64 shows a total of 76 cases of fatal pulmonary embolism. A definite warning embolus was recorded in 13 (17%) of these cases. In a further 16 cases symptoms of dyspnoea or chest pain were not considered sufficient for a definite diagnosis to be made, and in the remaining 47 cases sudden and profound collapse of the patient was apparently unheralded.

These figures suggest that massive fatal pulmonary embolism usually occurs without any warning. It is therefore all the more important to regard any symptom or sign of pulmonary embolus, however slight, as a potential "warning," and to treat it with the seriousness it deserves.—I am, etc.,

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Chemical Treatment of Skin Cancer

SIR,—Your leading article (15 July, p. 125) on this subject made good sense. On several occasions I have witnessed Dr. Frederick Mohs at work in his clinic, and have been deeply impressed by both the logic of his chemosurgical method of therapy and the care with which he carries it out. The success he achieves in cases of advanced skin cancer and cases of cancer which have recurred after previous radiotherapy or surgery is unequalled by that attainable by any other method.

Dr. G. M. King (19 August, p. 495) states that his results from treatment with 5% 5-fluorouracil (5-FU) ointment, though it is much too early to assess the long-term success rate, have been "quite impressive." This statement can carry little weight, firstly because it is unsupported by figures which can be interpreted by others, and secondly because it is easy to obtain "quite impressive" results in the treatment of skin cancer by a wide variety of methods. The results would certainly have to be far more impressive than those obtained by E. Klein and his colleagues[1] with 20% 5-FU to justify the widespread adoption of this method of treatment. The possible value of 5-FU in the treatment of solar or arsenical keratoses is in my view a matter for separate evaluation.—I am, etc.,

Chester Beatty Research Institute, FRANCIS J. C. ROE.
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REFERENCE

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Hazard from Nylon

SIR,—I was interested to read of the case of ischaemia of the finger-tip of a small infant reported by Dr. G. G. Noott (5 August, p. 370). A similar case came under my care not long ago in which a loop of fibre inside the baby's glove had strangulated the finger-tip, resulting in gangrene of the part.

This is a hazard I had not come across before, and it seems obvious that mitts made of material would be much safer than knitted gloves or mittens on tiny babies.—I am, etc.,

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Epsom, Surrey.

Treatment of Mendelson's Syndrome

SIR,—Dr. G. Taylor's letter (5 August, p. 368) outlines a very comprehensive regimen of treatment when aspiration of gastric contents has occurred, and states that "vomiting and regurgitation of stomach contents . . . should be regarded as preventable." However, apart from the excellent recognition that all women in labour must be suspected of having a full stomach, irrespective of their last oral intake of food or drink, and the advice on giving oral antacids before anaesthesia, he omits emphasis of the use of various techniques available at induction of anaesthesia for prevention of inhalation of gastric contents. Presumably these are taken for granted, but in such an important subject, where prevention is considerabiy superior to cure, any mention of prevention (points (1) and (2)) should stress the importance of a well-organized technique of induction, maintenance, and termination of anaesthesia.

Several methods are available for induction of anaesthesia: varying the position of the patient, general anaesthetic agents used,[1–3] use of cricoid pressure,[4] or use of regional analgesia.[5–8]

No method can guarantee 100% safety, and, although we are grateful for any new methods of treating Mendelson's syndrome when it has occurred, we should strive our utmost to reduce the incidence of this complication by taking all possible steps to avoid its occurrence.—I am, etc.,

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Stewart–Morel Syndrome

SIR,—A woman, 77 years old, was admitted to our unit complaining of headache, vertigo, exertional dyspnoea, and some joint pains. It was noted that she exhibited hirsutism, and she told us that it has been necessary for her to shave each day for about 20 years. She did not remember when she started menstruation, but her periods were regular and she had had the menopause at about the age of 45 years.

On examination there was pallor, slight pitting oedema of both ankles, and lipomatosis of both arms and thighs. Her blood pressure was 220/

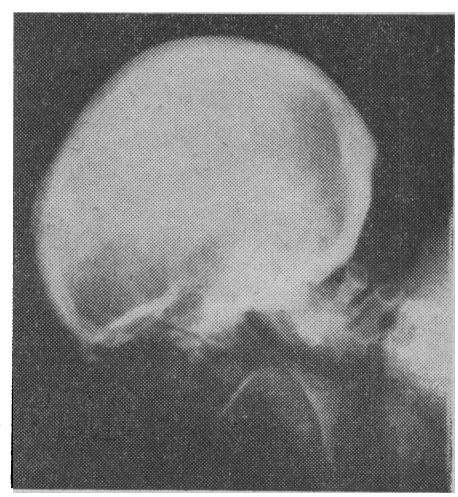

FIG. 1

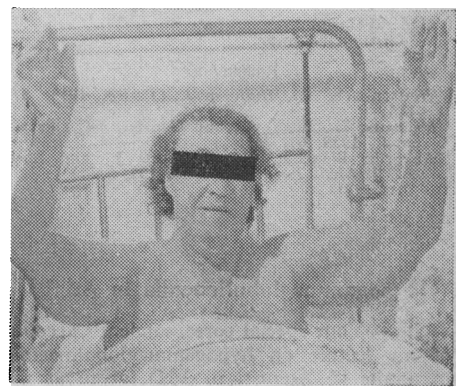

FIG. 2

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-

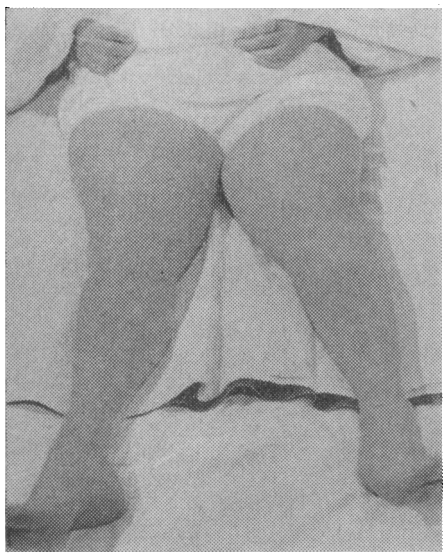

FIG. 3

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.,

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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.,

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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.,

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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.,

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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