serotype *canicola* in the same way; and this aspect is under continuous review.—We are, etc.,

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Syringomyelia

SIR,—Your leading article on syringomyelia and cavities in the cord (27 December, p. 759) neglects, I think, some important points. The use of the term "syringomyelia" throughout medical literature is confused by a lack of a clear definition because of lack of understanding of the pathological processes concerned. Perhaps the inclusion of many conditions under the general heading of "syringomyelia" is justified in the present state of knowledge. There is, however, certainly a case for differentiating from the heterogeneous accumulation of diseases which have at various times been called "syringomyelia" at least one specific and clear-cut syndrome, which I have called "communicating syringomyelia."[1]

The presence of a communication between the syrinx and the fourth ventricle has long been known and the great frequency and importance of this communication in pathogenesis have been stressed.[2-7]

This communication is invariably accompanied by an obstruction or partial obstruction in the subarachnoid pathways linking the intracranial cerebrospinal fluid compartments with the spinal subarachnoid space. It seems probable that intracranial pulsation is thereby directed down the communication and produces a transmural pressure gradient across the wall of the cord. This would account for the gradual and progressive destruction of cord tissue in this disease.

There is therefore a well-defined triad recognizable and worthy of redefinition. This consists of: (1) clinical evidence of intramedullary cord destruction with a cavity; (2) partial blockage of the cerebrospinal fluid pathways between the intracranial compartments and the spinal subarachnoid space; and (3) a communication between the fourth ventricle and the cord cavity.

I think that it is unwise to claim this syndrome as "true syringomyelia," since this would demand a very radical reclassification of books, articles, specimens, case-records, and, perhaps most important, the thoughts of men. Clearly this is responsible for a great deal of what has been called "syringomyelia" in the past. I suggest the term "communicating syringomyelia" as being clear and preserving the historical associations of the noun.

To define "syringomyelia" as Ellertsson[8] attempts to do on the basis of *x*-ray findings seems to be unsatisfactory. All the cases which he defines as "syringomyelia" seem to be "communicating syringomyelia." It is the communication which allows the cysts to become slack when the patient is not performing movements of venous distension—for example, when relaxed on the *x*-ray table. It is the communication also which allows the pressure to build up again after the aspiration of fluid from the syrinx, thus giving rise to the phenomenon which was commented upon in your leader—that is, that aspiration of such cases is not beneficial. Not mentioned in your leading article was another publication by Ellertsson and Greitz[8] in which they deliberately demonstrated the existence of the communication in the same series of patients by isotopic studies with injection of radioactive iodine-labelled albumin.

I would disagree with your last paragraph in which you state that the aetiology of syringomyelia remains obscure. The communicating variety has been demonstrated and treated in neurosurgical clinics all over the world, and though it may well be true that some cystic cavities have an obscure aetiology some of them have a mechanical and remediable cause. It is vitally important that persons having the care of such patients should refer them for neurosurgical investigation and treatment.—I am, etc.,

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Wigs and Waste

SIR,—We also have the same problem as Dr. Anne E. McCandless (24 January, p. 235) in obtaining from the National Health Service, temporary wigs for children.

During the last twelve months we have treated at least eight children with either chemotherapy for leukaemia or radiotherapy for primary brain tumours or metastases who have subsequently lost their hair. Our aim is to get these children home as soon as possible and back to a normal life, possibly even back to school. This is rendered impossible if the child is bald. Some of these children may only have a few more months of symptom-free existence left, but one aims to make these as normal and as happy as possible. Not only does an N.H.S. wig cost between £35 and £40, but it also takes up to three months to obtain one. A perfectly satisfactory acrylic wig can be bought the same day for under £5, but the N.H.S. will not at present pay for this. The Department of Health and Social Security hopefully suggests that "free monies" may be available. Surely such a practical, economical, and time-saving alternative to the expensive wigs at present provided at the taxpayers' expense should be provided under the N.H.S.

This does not only apply to children having treatment. The delay in getting an N.H.S. wig for an adult often means that by the time the wig arrives the hair is already growing again and the wig is hardly used.—I am, etc.,

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Proteins and Insulin Release

SIR,—The experiments of Dr. R. J. Jarrett and his colleagues reported recently in your journal (6 December, p. 598) show that exogenous secretin or pancreozymin enhances the increase in blood insulin in response to intravenous infusion of amino-acids in man, an effect which has already been amply demonstrated.[1 2] Their observation that the concurrent administration of both hormones together with amino-acids has more than an additive effect is only evident in one of their two subjects (Fig. 2) and must await confirmation.

Changes in glucagon-like immunoreactivity in the previous studies suggested that stimulation of glucagon-release by pancreozymin is associated with prolonged enhancement of insulin secretion. Changes in the blood glucose suggested that maintenance of the glycaemic stimulus to insulin secretion by the glycogenolytic action of glucagon was involved. Similar observations in the dog have led to the same conclusions.[3 4] It is therefore of interest that the insulin responses to pancreozymin with or without secretin in the subjects of Jarrett and his colleagues were not apparently related to changes in the blood glucose, but it is difficult to interpret the data in the absence of information about the blood amino-acid levels.

The physiological importance of an alimentary stimulus to insulin during the absorption of amino-acids from the intestine in man has been inferred from the effects of intravenous or enteric infusions of arginine.[5 2] In these studies the blood levels of glucose and amino-acids were matched, but only the enteric infusions provoked maintained stimulation of insulin secretion, and it was suggested that pancreozymin may mediate this effect.

With respect to the role of gut hormones in the release of insulin after ingestion of glucose, evidence has been reported which conflicts with that cited by Jarrett and his colleagues. Enhancement of the insulin response to intravenous glucose has been provoked by acidification of the duodenum in two further studies in man, and reasons for the absence of this effect in experiments with achlorhydric subjects have been put forward.[6 2] The validity of pancreatic exocrine responses as indexes of release of secretin or pancreozymin in all circumstances must be questioned. Studies with a radioimmunoassay for secretin have shown that glucose taken by mouth provokes an increase in blood levels of this hormone