# Correspondence

*Because of heavy pressure on our space, correspondents are asked to keep their letters short.*

## Transient Focal Cerebral Ischaemia

Sir,—I hope I may be allowed to comment on the provocative paper of Drs. R. E. Kendell and J. Marshall on the genesis of transient focal cerebral ischaemic attacks (August 10, p. 344). Because these authors were unable to reproduce the patient's focal symptoms as a primary manifestation by the production of falls of blood-pressure by tilt-table with infusion of hexamethonium they conclude that hypotension is not normally a causative factor in the genesis of such attacks and disparage our hypothesis of focal cerebral haemodynamic crisis. Yet focal symptoms were produced in 20 of the 37 cases, and in 12 of these were relevant to those of spontaneous attacks. The authors decline to attribute such signs to a localized haemodynamic crisis because subjective symptoms of oncoming faintness, deep respiration, clammy skin, etc., occurred first, and were attributed to general cerebral ischaemia. They appear to be unaware that such pre-syncopal symptoms commonly accompany any severe local cerebral haemodynamic crisis occurring spontaneously as a result of blood loss, cardiac arrhythmia, or shock, and relate to the steepness of fall of blood-pressure. Such symptoms do not of themselves necessarily indicate cerebral ischaemia, being commonly produced directly by carotid sinus stimulation, but in any case the first paralytic symptom clearly associated with ischaemia comes from the vulnerable focal cerebral arterial area. This was our original contention, which the findings of these investigations in no way deny.

I have elsewhere[1] discussed the effect of established infarct on such attacks, and would consider the cases listed under group III by Drs. Kendell and Marshall as irrelevant to the general problem. In their groups I and II 12 of 23 developed focal signs with hypotension, six of them signs of the kind which occur spontaneously. The remainder developed no focal sign before syncope or near syncope. I would maintain that in these 11 the absence of reproducibility of attack means either that cerebral ischaemia had no part in their causation or that any ischaemic factor underlying the attacks was no longer operative. In the absence of any information as to how recently the spontaneous attacks had occurred it is not possible to resolve this alternative. Nevertheless the prime criterion of haemodynamic crisis (reproducibility of attack) was absent, so that by our definition that physiological state is excluded in this group. There are clearly many causes of recurrent cerebral symptoms. On general principles we doubt that cerebral ischaemia, whether focal or general, can occur as frequently as 200 to 1,000 times without catastrophe, as in five of the patients who were cited. The transient disorders of extreme hypertension, as in the one case cited in detail, are also irrelevant to the problem, so that I question the validity of the word "ischaemic" in the title of the paper.

Finally, since "general cerebral ischaemia" to the point of syncope (with generalized convulsions in three) was produced by these authors at blood-pressures as high as 95 mm. Hg in one patient, 90 mm. Hg in three, and 70–80 mm. Hg in nine, I challenge the statement of these authors that "hypotension is remarkably well tolerated" in hypertensive states. For all these reasons I submit that the findings do not warrant the sweeping generalizations that are made in relation both to the complex problems of cerebrovascular stenosis and to the equally potentially catastrophic states of hypertension.—I am, etc.,

D. Denny-Brown.

Department of Neurology,
Harvard University Medical School,
Cambridge, Mass., U.S.A.

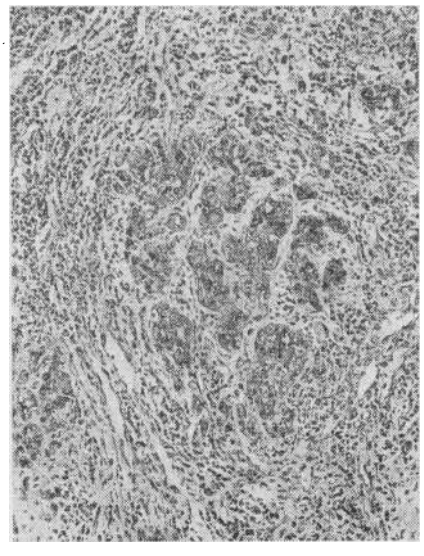
References

[1] Denny-Brown, D., *Arch. Neurol. (Chic.)*, 1960, 2, 194.

## Spontaneous Regression of Cancer

Sir,—Your annotation (September 21, p. 700) prompts us to put on record a striking case of unexpected survival after generalized cancer.

The patient was a man of 22 who presented in 1948 with a swelling 5 cm. in diameter in the right scapular region. He said it had been growing slowly for two years and was becoming painful. At operation the mass was found to be in the *right* latissimus dorsi muscle and section showed undifferentiated carcinoma (see Fig). There were no other clinical findings, and whilst investigations were proceeding for a primary focus he developed a mass of lymph nodes in the *left* axilla. One month after the first operation this mass of glands, 6 cm. in diameter, was removed and showed a pathological picture similar to the previous one. His general health and functions were normal and all investigations were negative. The only pointer to a primary focus was a 3-cm. scar in the middle of his back from which an innocent-looking mole had been removed by his doctor abroad three years previously and no histology had been performed.



As can be seen from the pathology, the prognosis seemed hopeless and a difficult decision had to be made as regards his disposal, as he had come to London from abroad to study and had just commenced a three-year course. He was seen in consultation with Sir Daniel Davies and Sir Stanford Cade and it was decided to let him carry on with his studies while a course of radiotherapy was given to the upper half of the trunk as an out-patient at Westminster Hospital. He has remained well ever since, 15 years after the removal of these malignant deposits.

This case also demonstrates well the tremendous advantage to a patient of complete ignorance of his disease. This man had absolute faith in his medical advisers and never asked any questions (all the more remarkable as he was studying law and is now in active practice as a barrister). Supposing he had insisted on knowing the truth? In view of the pathology he would have had to be told that he had barely a few months to live. Surely the moral is to avoid telling a patient the truth if it is really unpleasant. —We are, etc.,

Isobel P. Beswick.
George Qvist.

London W.1.

## Jejunal Biopsy in Childhood

Sir,—I read with great interest and pleasure Professor D. Hubble's article on Coeliac Disease in Childhood (September 21, p. 701) and in general would agree with his comments on the value of the diagnostic procedures now in common use.

Experience at this hospital, however, would lead me to give a more encouraging account of the technique of obtaining a jejunal biopsy using the Crosby capsule. In the past year 110 biopsies have been obtained with this instrument and there have been no complications. Screening time rarely exceeds five minutes. In one child the attempt to obtain a biopsy was abandoned when the capsule had not passed the pylorus after a screening time of 15 minutes. There have been no other complete failures although in some cases more than one attempt was necessary.

However, I find myself in disagreement with the view quoted by Professor Hubble that the capsule is too large to pass the pylorus of infants under the age of 18 months. Twenty-nine of the patients in our series were between the ages of 6 months and 18 months. Adequate biopsies were obtained without any great difficulty. It would be particularly unfortunate if the use of this simple technique was discouraged in these young infants, since it is at this age that accurate urine and faecal collections, on which one would