demonstrated until 1971 when Andrén and Eiken[3] showed that radio-opaque fluid injected into the wrist under pressure often flowed into the ganglion, whereas injection into the ganglion itself never caused fluid to flow in the other direction. More recently Angelides and Wallace[4] have demonstrated that the dorsal wrist ganglion arises from the scapholunate joint, from which fluid is pushed into the overlying capsule where it passes through a tortuous duct which acts as a one-way valve.

The valve mechanism explains all the various characteristics of ganglia—namely, their occurrence at certain sites over joints and tendon sheaths, their aggravation by movement and relief by rest, their tendency to ramify and burrow into tendons, nerves, and even bone, and the high recurrence rate from simple excision without removal of the valve mechanism. But this mechanism of ganglion formation also explains the tendency for ganglia to resolve spontaneously or to disappear after rupture or after incomplete removal. This is presumably due to the fact that a Bunsen valve may cease to operate once pressure has been reduced on both sides of the valve.

A 50% spontaneous recovery rate certainly justifies an expectant policy with symptomless ganglia and a modest remission rate after rupture justifies this as the initial treatment in many cases. However, I do not agree with the suggestion that the doctor should try "simple pressure" on the ganglion. This is painful and often fails to rupture the ganglion. Provided the ganglion is of a reasonable size and can be made tense, then dispersal is nearly always possible using a sharp blow with a mallet. The mallet should be concealed in the pocket of the operator, who stands with his back to the patient while delivering a single sharp blow over the top of the ganglion.

Failing this, surgery is indicated in symptomatic ganglia. Provided that the operation is done under good conditions with an exsanguinating tourniquet so that the underlying valve mechanism can be located and excised, then recurrences will be reduced to vanishing point.[5]

A W FOWLER

Bridgend General Hospital,
Bridgend, Mid-Glamorgan

[1] Adams, R, *Dublin Journal of Medical Science*, 1840, **17**, 520.
[2] Jayson, M I V, and Dixon, A St J, *Annals of Rheumatic Diseases*, 1970, **29**, 415.
[3] Andrén, L, and Eiken, O, *Journal of Bone and Joint Surgery*, 1971, **53A**, 299.
[4] Angelides, A C, and Wallace, P F, *Journal of Hand Surgery*, 1976, **1**, 228.
[5] Fowler, A W, *British Medical Journal*, 1971, **4**, 558.

Sterilisation and its reversal

SIR,—I was concerned to learn from Mr G J Hughes's article on sterilisation failure (19 November, p 1337) that he recommends that "as much tube as possible should be destroyed if the failure rate is to be further reduced." He further states in his last sentence that the encouraging drop in the pregnancy rate after sterilisation "can be further reduced . . . if more of the tube is destroyed. . . ."

Mr R M L Winston, in his article "Why 103 women asked for reversal of sterilisation" (30 July, p 305), pointed out that "sterilisation seems to be performed at an increasingly early age, particularly in England and America." He and others have suggested that sterilisation under the age of 30 is particularly associated with the request for reversal. Unipolar diathermy sterilisation through the laparoscope is notoriously dificult to reverse because the tubal damage may be so extensive. I believe that many women, after adequate counselling, would accept the slightly higher risk of subsequent pregnancy after less extensive tubal damage (preferably by the clip or Falope ring methods) in preference to the almost total loss of all opportunity for reversal.

JOHN GUILLEBAUD

Nuffield Department of
Obstetrics and Gynaecology,
John Radcliffe Hospital,
Oxford

Thalidomide and cranial nerve abnormalities

SIR,—A recent short report by Drs R Murphy and P Mohr (5 November, p 1191) describes a patient with two cranial nerve abnormalities caused by thalidomide. Judging by the content of this report and of the references quoted it would appear that the authors are not familiar with some of the work of German authors who have more experience of thalidomide embryopathy than anyone else. Stephenson[1] does mention briefly some of the German conclusions but does not say how these were reached. Moreover, Rapin and Ruben[2] in a recent comprehensive publication on anomalies with malformed ears make no reference to any of the investigations by these authors. We therefore think it worth summarising some of the findings and conclusions of German authors with regard to the aetiology of congenital cranial nerve lesions.

Congenital palsies of the abducens and facial nerves (often labelled the Duane and Moebius syndromes) are sometimes found in association with ear abnormalities. However, when large series of congenital ear deformities were described in the wake of the thalidomide disaster this association became common. In one series of 70 patients with deformed ears[3] 23 had bilateral abducens nerve palsies, while 41 had facial paralysis that was complete and bilateral in seven. There was much controversy at this time as to whether the facial paralysis was due to narrowing of the fallopian canal or to an underdevelopment or aplasia of the brainstem nuclei. Besides the analogy with the Moebius syndrome there are several points in favour of the nuclear theory. Radiologically the fallopian canal is normal or abnormally wide in many cases of complete paralysis[1]; it is also our experience that the suggested narrowing of the canal is not shown. The occasional association with palatal paralysis also suggests a lesion above the geniculate ganglion.[5] The most surprising and important findings, however, involved a later series of 43 thalidomide children with facial palsy and ear abnormalities, 29 of whom also had abducens palsies.[5] Normal indirect electrical excitability was found in 32 cases and even in complete congenital facial paralysis the facial nerve was found to be able to function, with a normal excitability and conduction time. It had been pointed out previously[6] that in thalidomide facial palsy, as in the Moebius syndrome, it is mainly the upper branches of the nerve that are involved, and Nessel and Suwalek found no electromyographic activity in the forehead muscles but active action potentials of the muscles around the eyes and mouth. Fibrillation potentials were absent. Electromyography in a small child under sedation is not always reliable, though measurement of conduction time probably is. The coincidence of electroencephalographic abnormalities with cranial nerve paralysis and ear malformations is also strong evidence in favour of lesions of the brainstem.[1 5]

The often-quoted spontaneous regression of these thalidomide palsies was limited to cases with incomplete paralysis and was probably due to late development of the medullary nuclei. Its effect was also exaggerated, and of their 43 cases Nessel and Suwalek[5] could find no obvious improvement in 28; the improvement was slight in 14 and convincing in only 3. This discrepancy between electrophysiological findings and the degree of facial paralysis may well be explained by the phenomenon of *Gewohnheitslähmung* or habitual paralysis whereby the memory for the execution of a particular action is lost during a long-lasting paralysis. In a congenital paralysis a similar situation would apply if the process of initiating action by the late-developing cranial nuclei has not been "learnt." These findings and considerations suggest the necessity for electrophysiological examination of all patients with facial paralysis due to congenital dysplasias.

In a survey of congenital ear deformities seen at Oxford one of us (PDP) reported 19 cases of seventh cranial nerve lesions and 18 cases of sixth nerve lesions,[7] nearly all of which were considered to be due to thalidomide. No comprehensive electrophysiological investigation or follow-up survey has been made on these children, but in one non-thalidomide patient, now aged 13, there has been a significant improvement in facial nerve function.

P D PHELPS
P E ROLAND

Walsgrave Hospital,
Coventry

[1] Stephenson, J P B, *Developmental Medicine and Child Neurology*, 1976, **18**, 189.
[2] Rapin, I, and Ruben, R J, *Laryngoscope*, 1976, **86**, 1469.
[3] Kleinsasser, O, and Schlothane, R, *Zeitschrift für Laryngologie, Rhinologie, Otologie und ihre Grenzgebiete*, 1964, **43**, 344.
[4] Terrahe, K, *Archiv für klinische und experimentelle Ohren-, Nasen- und Kehlkopfheilkunde*, 1972, **202**, 85.
[5] Nessel, E, and Suwelack, K, *Archiv für klinische und experimentelle Ohren-, Nasen- und Kehlkopfheilkunde*, 1967, **189**, 98.
[6] Miehlke, A, and Partsch, C J, *Archiv für Ohren-, Nasen- und Kehlkopfheilkunde und Zeitschrift für Hals-, Nasen- und Ohrenheilkunde*, 1963, **181**, 154.
[7] Phelps, P D, *Archives of Otolaryngology*, 1974, **100**, 11.

Pathogenesis of acute appendicitis

SIR,—Obstruction of the appendicular lumen plays an important part in the pathogenesis of appendicitis.[1 2] It is thought that as a consequence of high intraluminal pressure the blood supply to the appendix mucosa is compromised and there is invasion of the appendix wall by bowel organisms leading to inflammation. If this is so, in the early stages of appendicitis there should be a well-defined area of inflammation distal to the obstruction.[3 4] We have recently analysed the distribution of inflammatory changes in acute appendicitis by means of histological examination of multiple longitudinal sections. Appendices were opened longitudinally when fresh and pinned out flat for fixation. In 42 out of 44 cases inflammation was either sharply confined to the distal part of the appendix or involved the whole organ. In only two cases was there inflammation confined either to the proximal part of the organ or to the central portion.

The cause of the obstruction has been variously ascribed to fibrous bands, kinks, faecoliths in the lumen, or lymphoid hyperplasia. Faecoliths have been found in 67% and 89% of acutely inflamed appendices.[2 1] In this study faecoliths were present in only 9% and in 25% the lumen was entirely empty. In the remaining 66% the lumen contained a mixture of soft faeces and purulent material. No fibrous bands or kinks were identified. Lymphoid tissue in the appendix reaches its maximum development in the first decade. The lumen reaches its smallest size relative to wall thickness and theoretically is at its most

susceptible to obstruction as a result of lymphoid hyperplasia until involution of this lymphoid tissue occurs. This relationship would account for the characteristic age distribution of acute appendicitis.[5] On this basis lymphoid hyperplasia occurring during viral exanthemata and bacterial infections could lead to appendicitis. However, one study has shown that there is no greater incidence of acute appendicitis in children with these infections than there is in children who are otherwise well.[6] An alternative explanation is that obstruction is due to muscular spasm in the appendix associated with a low-fibre diet, as is thought to occur in the colon, and this would account for the geographical distribution of the disease.[3 4]

L W L HORTON

Department of Surgical Pathology,
St Thomas's Hospital,
London SE1

[1] Wagensteen, O H, and Bowers, W F, *Archives of Surgery*, **34**, 496.
[2] Bowers, W F, *Archives of Surgery*, 1939, **39**, 362.
[3] Burkitt, D P, *British Journal of Surgery*, 1971, **58**, 695.
[4] Burkitt, D P, in *Refined Carbohydrate Foods and Disease*, ed D P Burkitt and H C Trowell, p 87. New York, Academic Press, 1975.
[5] Bohrod, M G, *American Journal of Clinical Pathology*, 1946, **16**, 752.
[6] Lawrence, K B, and Waring, G W, *New England Journal of Medicine*, 1949, **241**, 1.

Richter's hernia at site of insertion of laparoscope

SIR,—Mr J B Bourke's suggestion (26 November, p 1393) that this complication might be eliminated if smaller-diameter laparoscopes were used cannot be allowed to pass without comment. It is surprising that a surgeon (rather than a gynaecologist) should ever accept an unsutured 12 mm defect in the linea alba. This defect is easily closed with a figure-of-eight suture using a heavy taper-cut or cutting needle and the problem is eliminated. Smaller defects caused by smaller laparoscopes should be similarly sutured.

At present the general surgeon is usually called to deal with the complications of gynaecological laparoscopy. Natural reluctance to embarrass colleagues results in little comment being made in these circumstances and the reported complications are almost certainly only the "tip of the iceberg." As laparoscopy is used increasingly in general surgery and the surgeon treats complications of his own making I anticipate further reports of Richter's hernia if the defect in the linea alba is not sutured.

DOUGLAS STEWART

Royal Infirmary,
Edinburgh

Labetalol and urinary catecholamines

SIR,—We have recently learnt that the levels of free urinary catecholamines are considerably increased in some patients following oral labetalol and that this effect may persist for up to 10 days after stopping the drug. The demonstration of excess urinary catecholamines is often used as a criterion for the diagnosis of phaeochromocytoma, but patients suspected of having this condition are not normally investigated until all antihypertensive treatment has been stopped. Nevertheless, we have recently heard of a labetalol-treated patient in France in whom phaeochromocytoma was wrongly diagnosed and an unnecessary operation subsequently carried out. It is to avoid similar mistakes that we have written this letter.

We are carrying out experiments to determine the mechanisms whereby labetalol increases urinary catecholamines Increased sympathetic activity in response to the fall in blood pressure caused by labetalol is, of course, a probable explanation for the excess urinary catecholamines. This hypothesis and others are now being investigated in our laboratories and the results obtained will be published as soon as possible. In the meantime we wish to reassure physicians that rebound hypertension did not occur in patients in whom labetalol treatment was deliberately stopped abruptly and has not been reported in any other patient.

DAVID HARRIS
Medical Director,
D A RICHARDS
Head of Medical Department,
Allen and Hanburys Research Ltd

Ware, Herts

Shadow over Maltese medicine

SIR,—Mr J Z Adami (17 December, p 1604) says that he can provide proof that the aid offered by the BMA to Maltese students goes only to students who take an anti-Maltese Government stand and he therefore accuses the BMA of being a political organisation. The fact is that help is offered, in so far as it is possible to offer help, to all Maltese students irrespective of their political allegiances, which are of no concern to us. I have, however, made inquiries as a result of which I am informed that two students who support the Maltese Government are in this country. The first is Mr Adami himself, and he has not so far called at BMA House, nor has he requested assistance. The other student called at BMA House to request our help in obtaining a locum. He was given several addresses and I understand that he has now obtained a preregistration job in the London area, though we have not heard from him since. I believe that there are two Maltese students apart from Mr Adami who have not so far visited us at BMA House, but we are not aware of their political views. Needless to say, we shall be very pleased to see them if they would care to call.

One student who, we are told, supports the Maltese Government has sought financial help from us. Unfortunately we were unable to help him, as the fund from which we provide financial assistance is limited to Maltese nationals and this student is not Maltese. It had nothing to do with his political views, of which we were unaware at the time.

E GREY-TURNER
Secretary, BMA

BMA House,
London WC1

Prevalence of communicable diseases

SIR,—In your recent leading article on schistosomiasis in China (19 November, p 1304), the unqualified statement that "Schistosomiasis is the only communicable disease in the world whose prevalence is increasing" begs several questions. How far is it justifiable to consider the prevalence of a communicable disease as global when most such diseases occur in their own separate environments? Furthermore, those who have intimate knowledge of the occurrence of communicable diseases in the field know the difficulties in interpreting official notifications.

Are we not in our seventh pandemic of cholera? Are viral jaundices on the decline? Is malaria control equally effective in all parts of the tropics? Is not the "epidemic" of Bancroftian filariasis in the Far East still under way? Although transmission of onchocerciasis (associated with river blindness) may have been controlled in the Haute Volta, what is happening in the rest of Africa? And is the control of sleeping sickness effective in all parts of Africa?

In sum, sir, the increase in the prevalence of communicable diseases may by no means be confined to schistosomiasis.

WILLIAM KERSHAW

Hesketh Bank,
Preston, Lancs

Emergency in emergency departments

SIR,—I would like to endorse wholeheartedly Mr C C Slack's anxiety regarding the future of staffing of accident and emergency departments throughout the country (19 November, p 1359). In his letter he raises one or two important points regarding the staffing of such departments.

(1) He points out that the present junior hospital doctors' contract has made the work in an accident and emergency department less attractive than before. I am sure that he does not state this point forcefully enough, as most departments operate a 40-hour week, creating a situation in that doctors moving to an accident department job from a surgical specialty will often suffer a cut in salary of up to 30%.

(2) He raises the point of subsequent default and alleges that some hospitals adopt a policy that work in the accident and emergency department must be the first undertaken in any surgical rotation. This is not our practice in Hull, but it is certainly accepted both by the senior house officers and by the employing consultants that one reason for senior house officers wishing to work in the department is that they use it as a time for taking examinations, as a consequence of which up to a month of the six months spent in the department may be taken as holiday, study leave, and leave for examinations.

(3) Many doctors entering Britain now have little option but to take jobs in accident and emergency departments, as few surgical specialties will accept senior house officers unless they have already passed their primary fellowship examination. This is a blueprint for disaster, as accident and emergency departments are fraught with dangers of diagnosis, medicolegal problems, hazards with communication, etc, and it is of course the last place that a doctor new to the country should elect to work.

Having now had experience of managing an accident and emergency department for several years I would like to propose (1) that it is possible to run both a training programme and a career grade structure of doctors parallel to one another in the same department; and (2) that the junior staffing levels of a department should be based on the number of patients seen per annum in that department. It is accepted now that the specialty of accident and emergency work has come to stay and it therefore follows that all major accident