CORRESPONDENCE

Correspondents are asked to be brief

Epiglottitis in Adults J. R. May, F.R.C.PATH., and A. Ingold, A.I.S.T.	Accident and Emergency Services D. W. Bracey, F.R.C.S., and R. E. Loder, F.F.A. R.C.S.; G. Rigby-Jones, F.R.C.S.ED.	Use of English F. Langford, F.R.C.S.
Bowlby's Disease? M. J. Mitchinson, M.D., M.R.C.PATH.	Prognosis in Industrial Dermatitis B. Bentley-Phillips, M.D.	Experience versus Examination Results H. de Glanville, M.B.
Mental Health Aspects of Shoplifting P. R. V. Tomson, M.B.	Malignant Hyperpyrexia N. P. Keaney, M.B., and F. R. Ellis, PH.D., F.F.A. R.C.S.	Dissect J. N. Matthews, D.P.H.
Interpretation of Arteriography J. W. D. Bull, F.R.C.P.	The New F.F.R. G. A. Medhurst, M.R.C.S., D.M.R.D.	Hospital Laboratory Services J. Tregillus, F.R.C.PATH.
Starch Granulomatosis of the Peritoneum J. F. Colin, F.R.C.S.ED., and C. Wastell, F.R.C.S.; G. Hyman, F.R.C.S.	Cyclophosphamide and the Bladder F. R. Glover, D.P.H.	Voluntary Services in Hospitals Chrystal King.
Deglycyrrhizinized Liquorice in Duodenal Ulcer B. Whiting, M.R.C.P., and T. J. Thomson, F.R.C.P.	Reflux and Hernia Surgeon Captain T. L. Cleave, M.R.C.P.	Junior Staff and Private Patients D. R. Redman, M.R.C.P.
Treatment of Opium Addiction R. A. F. Jack, M.B.	Mental Deficiency Nursing Jean Patey, F.R.C.S.ED.	Job Hunting—Why Not Emigrate? B. S. Sengupta, F.R.C.S.ED.
Maternal Oestrogen Levels during Penicillin Treatment M. Pulkkinen, M.D., and K. Willman, M.D.	Practicalities of Nursing Dame Isabel Graham Bryce, M.A.	Flatulence C. F. Hawkins, F.R.C.P.
	Queen Alexandra and the Tate A. H. R. Hewett-Clarke, L.M.S.S.A.	Psychogeriatric Care J. F. Donovan, D.P.M., and others.
	Threadworm Cystitis J. C. Linley-Adams, M.D.	Pensions and the Abatement Rule D. Lamont, F.R.C.S.

Epiglottitis in Adults

SIR,—In your leading article "Epiglottitis in Adults" (24 July, p. 204) you state that the causative organism of acute epiglottitis, in adults or children, is usually *Haemophilus influenzae* type B and that ampicillin is the antibiotic most naturally chosen for treatment. You add that "it may be wise to supplement this with cloxacillin in case the condition is caused (our italics) by a penicillinase-producing staphylococcus. . . ." This statement raises a point which we believe to be important, but inadequately appreciated, concerning the management of infections in which penicillinase-producing organisms are involved.

While agreeing with your recommendation that it may be wise to give cloxacillin with ampicillin, because of your use of the word "caused" we think our reasons may differ. Can *Staph. aureus* really cause acute epiglottitis? Although there are occasional reports in the literature of the isolation of this organism from the pharynx or larynx of patients with acute epiglottitis, there seems to be none of its isolation from a blood culture in this condition. In contrast, blood cultures positive for *H. influenzae* type B have been commonly reported.^{1,2}

There must, therefore, be doubts about the causative association between *Staph. aureus* and acute epiglottitis but a case can be made, nevertheless, for adding cloxacillin to the treatment of any acute infection caused by a penicillin- or ampicillin-sensitive organism in a site where penicillinase-producers may be present.

The possibility that penicillinase-producing organisms, though not themselves pathogenic in a given situation, might interfere with penicillin therapy directed against the true pathogen was predicted as long ago as 1946 by Fleming,³ and a preliminary communication from our laboratory reported six ex-

amples of such interference, by enterobacteria, with ampicillin therapy of chronic *H. influenzae* bronchial infections.⁴ We have subsequently studied this phenomenon in many more patients and the findings will be reported in due course.

Cloxacillin and methicillin are inhibitors of β -lactamase (the commonest type of penicillinase); they bind with it, but, being split only very slowly, release it again equally slowly. This property may be used therapeutically to prevent the destruction, by coincident penicillinase-producers, of penicillin or ampicillin directed against the true pathogen, and examples of patients in which this approach was adopted have been given in the report mentioned above.⁴ Thus the addition of cloxacillin to ampicillin therapy of the type of infection mentioned in your leading article may be efficacious not because of the suppression of the (non-pathogenic?) *Staph. aureus*, but because of the inhibition of its penicillinase, which would otherwise destroy the ampicillin needed to suppress an underlying infection with *H. influenzae*. It is important to recognize the existence of such a mechanism, since one can envisage a situation in which penicillinase-producing *Staph. aureus* is isolated from a patient with, say, acute epiglottitis, leading to therapy with cloxacillin alone, which would of course have no effect on *H. influenzae*, if this were the true pathogen, and might have disastrous consequences.

The phenomenon of interference by one bacterial species with chemotherapy directed against infection by another does not seem to have been widely reported, but experience gained in this department of the management of chronic non-tuberculous respiratory infections suggests that it may occur quite commonly when more than one bacterial

species is present at the site of infection. In addition to the patients mentioned above, in whom penicillinase-producers interfered with ampicillin therapy, we have seen a chronic bronchitic patient harbouring a strain of *Pseudomonas aeruginosa* which apparently destroyed chloramphenicol directed against an underlying haemophilus infection, and we believe that sometimes tetracycline can be destroyed by enterobacteria in vivo, although we have been unable so far to confirm this in vitro.—We are, etc.,

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¹ Turk, D. C., and May, J. R., *Haemophilus influenzae—its Clinical Importance*. London, English Universities Press, 1967.

² Gorfinkel, H. J., Brown, R., and Kabins, S. A., *Annals of Internal Medicine*, 1969, **70**, 289.

³ Fleming, A., ed., *Penicillin*, p. 88. London, Butterworth, 1946.

⁴ Maddocks, J. L., and May, J. R., *Lancet*, 1969, **i**, 793.

Bowlby's Disease?

SIR,—The interesting case described by Mr. R. A. W. McDowall (31 July, p. 290) with coexistent idiopathic cervical and mediastinal fibrosis raises once again the enigma of the association of those two rare diseases "Riedel's thyroiditis" and idiopathic mediastinal and retroperitoneal fibrosis. The association has been reported several times.¹⁻³

The fibrosis around the thyroid is not usually contiguous with the mediastinal fibrosis, but those interested in this problem may wish to read an old case report by Bowlby.⁴ The account includes a necropsy report. A "stony hard," infiltrative mass involved the thyroid and other structures in the neck and was contiguous with similar tissue throughout the mediastinum. Though its wide spread suggested to Bowlby a

sarcoma, the histological appearances were those of a "hard fibroma," and the long history and complete lack of metastases do not suggest a malignant neoplasm. In retrospect this was almost certainly the first account of the disease described some 10 years later by Riedel, and incidentally, of its association (and contiguity) with idiopathic mediastinal fibrosis.

Perhaps this tantalizing group of diseases, with its cumbersome and variable nomenclature, should be called "Bowlby's disease".—I am, etc.,

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1 Comings, D. E., Skubi, K. B., Van Eyes, J., and Motulsky, A. G., *Annals of Internal Medicine*, 1967, **66**, 884.

2 Mulvaney, W. P., Gordon, L. Z., and Gritti, E. J., *Journal of Urology*, 1968, **99**, 417.

3 Ullmann, A. S., and Dacso, M. R., *Diseases of the Chest*, 1968, **53**, 656.

4 Bowlby, A. A., *Transactions of the Pathological Society of London*, 1885, **36**, 420.

Mental Health Aspects of Shoplifting

SIR,—The paper on the mental aspects of shoplifting (11 September, p. 612) was of interest to magistrates as well as doctors.

It might be preferable if the older women were not brought to court. If there is a "not guilty" plea the bench has to decide on guilt before the probation officer's report is read. The usual defence is that there was no intent to steal, and that the defendant was unaware of her actions. The store detective's evidence often describes the furtive way articles are transferred to the private shopping bag, and it becomes difficult not to convict the defendant. It may be that the behaviour is a cry for recognition or help, but a crime seems to have been committed. Psychiatrically, it may be naive to discuss whether she is "aware" of what she is doing and whether she has an "intent to steal," but the magistrate has to answer these questions.

Having found her guilty, what are the effects of sentence? Those usually considered are a straight fine, perhaps coupled with advice to see her doctor: this has the advantage of completing the affair rapidly, as the fine is usually able to be paid in seven days; or probation; or an absolute or conditional discharge, which may be contrary to local policy because of the lack of deterrent value. Has punishment any therapeutic value?

Is it the appearance in court, the conviction, or the sentence that has the traumatic effect, and have the authors any information about the results of different sentences on the individuals?—I am, etc.,

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Interpretation of Arteriography

SIR,—May I make one comment on your leading article "Fibromuscular Hyperplasia of the Internal Carotid Artery" (11 September, p. 598)? In an otherwise clear exposition, with no less than 23 references from English and American literature, you say, in referring to the classic arteriographic picture of a "string of beads": "However, this angiographic appearance is insufficient evidence to

allow a firm diagnosis of the disease to be made, for a similar picture may result from spasm of the carotid artery, particularly when related to a difficult puncture of the artery during arteriography. Surgical inspection and pathological examination of the vessel is the only absolute way of making the diagnosis." What do you mean by this? If an appearance is "classic," it is "classic." You do not quote any of your 23 authorities substantiating your caveat.

The arteriographic picture of spasm of the artery is quite different from the "string of beads" sign, which I would agree is not only classic but pathognomonic. What do you imply by a "difficult picture of the artery"? The puncture may certainly be difficult and three possibilities may ensue: (1) It is so difficult that it fails, in which case there is no arteriographic appearance. (2) If successful, the classic and pathognomonic sign will be revealed. (3) If the contrast substance enters the wall rather than the lumen of the artery, another pathognomonic sign will appear which closely simulates complete occlusion of the vessel, a now well-known artefactual appearance.¹

I would strongly contest the statement that "surgical inspection" is "an absolute way of making the diagnosis." The surgeon cannot see the wall layers or shape of the lumen of the artery without making an incision. Certainly pathological examination is "an absolute way."

Where does the scepticism of some medical writers end in regard to radiology? It would be a *reductio ad absurdum* to ask a surgeon or pathologist to confirm or refute the presence of a fracture revealed on an x-ray film in order to make an "absolute diagnosis," but how does this example differ in principle from the condition under discussion?—I am, etc.,

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¹ Newton, T. H., and Couch, R. S. C., *Radiology*,

Starch Granulomatosis of the Peritoneum

SIR,—May we, through your columns, report two cases of starch powder granulomatosis of the peritoneum similar to those reported by Mr. J. Neely and Dr. J. D. Davies (11 September, p. 625).

Both patients developed symptoms of malaise and ill-defined abdominal pain with pyrexia three weeks after laparotomy. Both were reoperated on within three days of each other and there were exactly similar intra-abdominal findings in each case. There was free intraperitoneal fluid, the greater omentum appeared inflamed, and there were many dense adhesions. Histological examination of biopsy specimens of the greater omentum showed there to be an intense inflammatory reaction with eosinophils and foreign body giant cells. The intraperitoneal fluid was sterile on culture in both cases and no acid-fast bacilli could be detected either by Ziehl-Neelsen staining or by guinea pig inoculation. The reaction appeared to be directed towards anisotropic periodic-acid-Schiff-positive material, which was likely to be starch powder granules.

The first case was a 43-year-old woman who had a cholecystectomy and operative

cholangiogram for chronic cholecystitis and cholelithiasis on 26 February 1971. Twenty days later she developed right-sided abdominal pain and pyrexia and was readmitted to hospital. On examination she was tender on the whole of the right side of the abdomen, and there was some induration around the lower half of the cholecystectomy incision. It was thought that she had a wound infection. However, the white cell count was consistently normal, serum amylase normal, blood culture normal, and there was no evidence of a subphrenic abscess. There was a urinary tract infection with *Proteus* spp. and *Str. faecalis* and the aspartate transaminase and alanine transaminase were slightly raised. She was observed for 11 days, during which time the pulse gradually rose to 120 beats per minute, the temperature to 102°F (38.9°C), and she became quite ill. Laparotomy was performed on 2 April and 3 litres of clear sterile slightly green fluid were aspirated from the peritoneal cavity. There was no evidence of a subphrenic abscess or acute pancreatitis, but the greater omentum was inflamed and stuck to all of the surrounding organs. Biopsies of the greater omentum were taken and the wound closed with drainage. Postoperatively she developed pulmonary oedema and a collapsed right lower lobe; she became very ill and exhausted and required intermittent positive pressure respiration for three days. Eventually she made a satisfactory recovery.

The second case was a 28-year-old man who underwent a selective proximal vagotomy for an active duodenal ulcer on 9 March 1971. Twenty-two days later he became unwell with a loss of appetite, malaise, and abdominal pains. He was admitted to hospital three days later when he was found to have a swinging temperature with a normal white cell count, and again it was thought he had a wound infection. The abdominal pain increased, and so 27 days after his original laparotomy reoperation was performed. The peritoneal cavity contained 1 litre of straw coloured fluid, which was sterile, the greater omentum was inflamed, and there were many dense intra-abdominal adhesions. Biopsies were taken for histological examination and the wound closed with drainage. He continued to have a hectic fever for the following 13 days but eventually made a satisfactory recovery.

We feel it is worth recording these two cases because both patients were ill and on general grounds required exploration. Had they not had a previous operation and simply presented de novo the diagnosis would have been of serious peritoneal inflammatory disease.—We are, etc.,

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SIR,—For the past three years I have used dry surgical gloves without finding it necessary to add extra powder lubricant.

The main glove manufacturers in England, who incidentally have a large export trade, have told me that so far as they know surgeons in Britain are practically the only ones using extra powder. Each glove already has a fine coating of powder and the makers are at present trying to eliminate it as far as possible from the outer surface. I would