thought to be *Haemophilus influenzae*, or possibly a coliform, it grew well on nutrient agar in the absence of X and V factors, and did not grow on McConkey's agar. It was finally proved to be *Pasteurella multocida*, a mouse dying 24 hours after inoculation with a culture of the organism. This organism was sensitive in vitro to penicillin, chloramphenicol, tetracycline, erythromycin, and cephaloridine. The white blood cell count was only 9,000/cu.mm. on admission, 85% of these cells being neutrophils. Discharge from the right ear produced a growth of *Staphylococcus aureus*, but no other organism was identified.

The patient was treated initially with full doses of penicillin, chloramphenicol, and sulphadimidine. Chloramphenicol was discontinued after one week, and the dose of sulphadimidine was reduced. After a further week the penicillin was replaced by a short oral course of cloxacillin, since the staphylococcus grown from the middle ear was penicillin-resistant. A second C.S.F. specimen obtained 10 days after the first was sterile, but still contained 22 leucocytes (mainly lymphocytes) per cu. mm. Satisfactory progress was maintained, and the patient was discharged home after one month in hospital. No contact with dogs or cats was admitted on direct questioning, and no symptoms of bronchitis had been noticed.

Pasteurella multocida is not uncommonly present in infected wounds, particularly in those caused by dog or cat bites, and is also found in the respiratory tree. It is usually not difficult to culture, although confusion may well arise between *Pasteurella multocida* and *Haemophilus influenzae* and possibly even with bacteria of the *Neisseria* group. This is especially true when a direct smear taken from the C.S.F. deposit is examined as an emergency procedure, as in this case. *P. multocida* is usually sensitive in vitro and in vivo to penicillin, and this would seem to be the drug of choice. However, as *Haemophilus influenzae* infection was considered likely, three different antibiotics were given to the patient reported here, and it is probably wise to give more than one antibiotic to patients with *P. multocida* meningitis in view of the poor prognosis.

No history of an animal bite was obtained, nor was there any evidence of bronchitis, both of which might have afforded a possible portal of entry for the organism. Since the patient had had a biopsy of the pituitary in the past it is possible that the organism entered through the nose, and even though the organism was not grown from the aural discharge this does not rule out the possibility of an infected middle ear being the portal of entry to the meninges.—We are, etc.,

J. A. EASTON.

J. LISTER.

C. PRAKASH.

Wexham Park Hospital,
Slough, Bucks.

REFERENCE

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Obese Non-diabetic Patients

SIR,—We would like to raise several points regarding the paper by Drs. A. Hart and H. Cohen (3 January, p. 22) on their trial of phenformin for obese non-diabetic patients.

For instance, in their discussion they state that they took the precaution of giving phenformin only in therapeutic dosage, for "in this way there was less danger that weight reduction could have been due to anorexia produced by the drug or interference with intestinal absorption." By "therapeutic dosage" they presumably mean the type of dosage usually required to lower the blood sugar level. If by "therapeutic dosage" they mean to reduce body weight, then the average amount required would be considerably greater in obese diabetics. By the purposeful avoidance of such doses they seem to have predetermined the negative results of their trial with regard to weight loss in non-diabetic obese subjects, for there is increasing evidence to show that the weight loss induced by biguanides is effected both by anorexia and malabsorption, in both diabetics and non-diabetics.^{1,3}

Further on in their discussion they also say, "Our experience leads to the conclusion that there exists in obese patients with diabetes a metabolic disturbance which is favourably modified by phenformin to effect weight reduction. There are obese non-diabetic subjects who do not have this metabolic disorder and therefore do not lose weight as a result of phenformin administration." The first part of this statement is so vague as to render the second part meaningless.—We are, etc.,

J. M. STOWERS.

P. D. BEWSHER.

Diabetic Outpatient Department,
Royal Infirmary,
Aberdeen.

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Allergy to Iprindole (Prondol) with Hepatotoxicity

SIR,—Side-effects with iprindole are uncommon, and so far as I know no cases of hepatotoxicity have been published.

The patient was a 42-year-old woman who was pregnant for the fifth time, with three children surviving. She had had no miscarriages. This pregnancy was uneventful, except for a single episode of pain in the left chest, and tenderness over the left kidney at 13 weeks. The only treatment she received for this was a few tablets of paracetamol. The pain cleared completely after four days. The only other drugs she received during pregnancy were iron (as Ferromyn-S) one three times a day, and folic acid 5 mg. daily.

At her post-natal examination on 30 April she mentioned that she was feeling "flat" and depressed, which I explained to her in terms of post-puerperal depression, and prescribed iprindole 15 mg. three times a day. I next saw her two weeks later, when she told me she had stopped taking the tablets. When questioned further it appeared that she had had a generalized rash after taking only five tablets, with giant urticaria, dermatographia, and weal formation. This rash and irritation stopped after about two days. After a further five days she started taking the tablets again, but after approximately five days she again stopped taking them because her rash recurred. She was vomiting bile and felt ill. Her urine was dark and gave a positive reaction to presence of bile (Ictotest). At no time was she clinically jaundiced.

Laboratory investigations on the same day

showed a raised E.S.R. 40 mm./hr.; W.B.C. 4,600/cu.mm. (normal differential); total protein 7.4 g./l.; electrophoresis albumin within normal limits, slight increase in alpha₂ globulin (otherwise normal distribution); serum bilirubin 1.3 mg./100 ml.; direct van den Bergh positive, alkaline phosphatase 25 K-A/units; thymol turbidity 1 unit; G-P transaminase 120 units. The pathologist's comment on the liver function tests was "consistent with hepatotoxicity." Two weeks later the E.S.R. had fallen to 16mm. in one hour and liver function tests were normal except for a slightly elevated alkaline phosphatase at 15 units. Urine bile test was negat.ve.

Iprindole is a fairly new drug, for which is claimed a lower incidence of side-effects when compared with other antidepressants. I feel this patient showed an allergic reaction to iprindole, as the symptoms recurred after the patient's own resumption of the drug after initial withdrawal. She also had a mild degree of hepatic involvement from which she made a complete recovery.

I should like to thank Dr. J. Lister, consultant physician to the Windsor Group, and Dr. T. V. A. Harry, of the medical department of John Wyeth and Brother Ltd., for their help with the presentation of this case.

—I am, etc.,

Bracknell, Berks.

P. J. W. YOUNG.

Organic or Psychogenic Stupor

SIR,—The differentiation of organic from psychogenic stupors receives but scant attention in the medical literature. A patient was recently admitted in stupor who had a past history of both cerebrovascular disease and depression. The definitive diagnosis was made only after the use of intravenous sodium amylobarbitone.

The patient, a 44-year-old woman, was hypertensive and had suffered a left hemiplegia eight weeks before her first admission to a psychiatric hospital with a depressive illness. The depression responded well to amitriptyline. Shortly after admission she had a sweating attack, and subsequent E.C.G.s. suggested a small myocardial infarction. In view of this and persisting hypertension she was transferred to a general hospital for review of her cardiovascular state.

Shortly after her transfer she became suddenly withdrawn and unresponsive and was readmitted to a psychiatric hospital. She was now mute, almost akinetic, but followed ward activities with her eyes and on occasions would respond to some verbal commands. She was incontinent and had to be fed. A diagnosis of stupor of either depressive or organic origin was made.

Ward observations were unhelpful, so a trial of intravenous sodium amylobarbitone was performed. Over five minutes 250 mg. of a 5% solution of sodium amylobarbitone was injected slowly. Following this the patient became more talkative, the content being entirely depressive. She also became more mobile, walking about the ward and eating her meals unaided.

Thus it became obvious that the stupor was of a depressive nature, and a course of electroconvulsive therapy was given with beneficial effect.—I am, etc.,

W. A. SAUNDERS.

Barrow Hospital,
near Bristol.