It would be interesting to know more precisely what were the criteria of "failure" cases.—I am, etc.,

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Management of Acute Salicylate Poisoning

SIR,—Unexpected sudden death has been a recognized feature of acute salicylate poisoning, and it still occurs. An early acidosis is the cause. Would not a blood pH done in casualty probably be life-saving? The result can be obtained in 15 minutes—far quicker than the plasma-salicylate level.—I am, etc.,

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R. C. MAC KEITH.

Unusual Mycoplasma pneumoniae

SIR,—We wish to report a case of *Mycoplasma pneumoniae* infection with an unusual presentation.

The patient was a 5-year-old boy who five days before admission developed a maculopapular, mildly pruritic erythematous rash. The rash appeared first on his upper arms and legs, and over the next three days spread up his trunk to involve the face. He was pyrexial during this time and had commenced a course of penicillin at home after the onset of the rash. He also had a mild irritant cough. There was no known contact with infectious or other disease. He had had rubella and chicken-pox and had been vaccinated against measles one year previously. No drugs had been taken prior to onset of the rash. A budgerigar was the only pet in close contact with the child.

On examination his temperature was 100.4° F. (38° C.). There was a generalized maculopapular rash tending to be confluent in the axillae and involving the palms and soles. The face was only mildly affected. A few petechial spots were noted on the hard palate. Generalized lymphadenopathy was present. His spleen was palpable 5 cm. below the left costal margin; the liver was palpable 3 cm. below the right costal margin. Lung fields were clinically normal. No other abnormality was detected.

A clinical diagnosis of glandular fever was made. However, his Paul-Bunnell test was negative on two occasions, and white blood count was 6,700/cu. mm., with slight neutrophilia and no abnormal cells. Further investigations excluded toxoplasmosis, cytomegalic inclusion disease, brucellosis, and enteric fever. E.S.R. 21 mm. at 1 hour. Haemoglobin 11.3 g./100 ml. No virus was isolated from throat swabs, blood, urine, or stools. Blood cultures were sterile. His temperature settled in 24 hours without treatment, and during the next five days his rash began to fade and the spleen and lymph nodes became smaller. His cough became more prominent, and on the fifth day after admission he developed signs of left upper lobe consolidation, confirmed by chest x-ray examination. He was given a seven-day course of ampicillin, but despite this the signs in his chest spread with crepitations heard in all areas of both lungs. His temperature rose to 101° F. (38.3° C.) in

the first 72 hours of the pneumonia, but he remained surprisingly well. The diffuse signs in the lungs persisted for 12 days, but after this time his chest was clear clinically and radiologically. There was some skin staining in the distribution of the rash, and his spleen was just palpable. His E.S.R. had risen to 62 mm. at 1 hour and his white blood count was 8,800/cu. mm., with 7% eosinophils, a total eosinophil count of 550/cu. mm.

The diagnosis was made retrospectively. Specimens of blood for virus antibodies were collected on the seventh day and the 28th day after the onset of the illness. Significant complement fixation titres were found only to *Mycoplasma pneumoniae* as follows:

7th day	...	1:4
28th day	...	1:64 (convalescent)

No cold agglutinins were detected on the 28th day. When seen as an outpatient at that time he was completely well. The spleen and liver were no longer palpable, and his E.S.R. was 8 mm. at 1 hour.

The rise in complement fixation titre to mycoplasma is regarded as significant. A positive titre is often not obtained until the tenth day of the illness.¹ Rashes are uncommon in *Mycoplasma pneumoniae* infections, but this organism has been implicated in a number of cases of erythema multiforme. In one series complement fixation antibodies to mycoplasma were found in five consecutive cases.² Pityriasis rosea and erythema nodosum have also been reported during mycoplasmal pneumonia.³ Splenomegaly is said to be rare in mycoplasmal pneumonia.⁴ We suggest that *Mycoplasma pneumoniae* infection should be considered when a clinical diagnosis of "glandular fever" is made, unsupported by haematological findings and with a negative Paul-Bunnell, specially when rash and pneumonitis are prominent.

We would like to thank Dr. J. F. Warin, consultant in infectious diseases, for permission to report this case.

—We are, etc.,

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ALISON LEACH.
B. W. LEWIS.

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Hiatus Hernia

SIR,—As one of the writers who criticized the final paragraph of your leading article on hiatus hernia (21 September, p. 691) I would like to comment on the recent remarks made by Mr. W. Silber (4 January, p. 53).

In the past surgery for the repair of hiatus hernia has had a bad name because it was said that there was a high degree of recurrence. During the last few years, when much has been learnt about the physiology of the gastro-oesophageal junction, the surgical results have improved greatly, and a very low incidence of recurrence follows operations such as the Belsey operation. During the year which has just finished, I have operated on 72 patients with hiatus hernia, and a further 40 have had their names added to the waiting-list. These patients have had distressing symptoms of reflux oesophagitis for an average of 10 years, and an appreciable

percentage have suffered serious complications during that time, such as anaemia of varying degrees, and repeated spillover pneumonitis. A much smaller number had developed organic strictures, judged at oesophagoscopy or thoracotomy, and two of the 72 patients had developed carcinomas after many years of reflux oesophagitis.

There is indeed an incidence of recurrence, as after all hernia surgery. In the case of hiatus hernia, however, this incidence is low, and, because a few of these patients suffer a recurrence, surely this is no reason for denying the great majority lasting relief from their most unpleasant symptom. Of all the thoracic surgical procedures, the successful repair of a hiatus hernia produces some of the most grateful patients.—I am, etc.,

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Treatment of Skin Fissures

SIR,—Lieutenant-Colonel H. C. M. Walker (4 January, p. 53) omits an essential part of the treatment of skin fissures. Healing will not occur in the presence of hard, thickened skin, and the fissure edges must be adequately trimmed before an occlusive dressing is applied. Healing is then certain and permanent. As he says, detergents must be avoided.—I am, etc.,

J. R. MASON.

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Rickettsial Endocarditis

SIR,—In view of recent reports in the *B.M.J.*—the clinicopathological conference (5 October, p. 40), Dr. H. Williams and others (9 November, p. 387), Dr. J. H. Walters (21 December, p. 770), and Dr. D. Meyers (21 December, p. 771)—we wish to describe briefly the apparently successful use of lincomycin, initially alone and subsequently with tetracycline, over a period of 15 months in a patient with endocarditis due to *Coxiella burnetii*. It seems particularly important to describe those cases where antibiotic therapy appears to have been successful since the publication by Kristinsson and Bental¹ who reported survival in five of six patients in four of whom valve replacement had been used. Prior to this all published cases managed conservatively had been fatal.

A 43-year-old farm manager's wife felt unwell in March 1966, with intermittent rigors and unusual tiredness. She was first seen in this hospital in July 1967, having been referred for investigation in the belief that she had a urinary infection. She was subsequently admitted to this hospital in September 1967. At the time of admission she was pyrexial, had finger clubbing, splenomegaly, and clinical and radiological evidence of mitral stenosis. There was a moderate anaemia (haemoglobin 11.4 g./100 ml.) and an E.S.R. of 45 mm. in the first hour. A palmar erythema was accompanied by a disturbance of liver function tests. There was also thrombocytopenia (platelets 32,000/cu. mm.) without purpura. In the belief that she had subacute bacterial endocarditis 15 samples of blood were taken during the first 48 hours for blood culture, and all of these proved subsequently to be sterile. Treatment was instituted initially with penicillin and streptomycin, and the pyrexia be-

came less. Subsequently she developed a very marked sensitivity rash, and this treatment was therefore stopped after two weeks. Oral erythromycin was then used, but only for 24 hours because of severe vomiting and diarrhoea. Oral lincomycin, 1.5 g. six hourly, was then given and effected complete remission of the fever; she has remained apyrexial since. After lincomycin therapy had been started complement fixation tests for *Coxiella burnetii* revealed 1:128 (phase I) and 1:4096 (phase II). Subsequent tests showed a rise of phase I titres to 1:2048. When the diagnosis of rickettsial endocarditis seemed in this way to be established, and two weeks after the institution of lincomycin, tetracycline 2 g. daily was added in the light of published data up to that time. The patient has continued with lincomycin and tetracycline in the same doses up to the time of reporting, and it is interesting to note that at no time has she experienced any diarrhoea or other untoward side-effects. The cost of this treatment is of course very considerable.

At the end of October 1967 she suffered a left-sided hemiplegia, a few days after developing a mitral systolic murmur, but over the past 14 months she has been making a slow but steady improvement. Currently there are the signs of minor mitral stenosis and regurgitation, but there is neither finger clubbing nor palpable splenomegaly. In September 1968 the E.S.R. and liver function tests were normal, and the complement fixation titres were for phase I 1:512 and for phase II 1:1024.

The fever was controlled by lincomycin for the first time; under prolonged therapy with lincomycin and tetracycline there has been improvement in the clinical state with parallel improvement in the laboratory findings.

Dr. R. E. Smith (9 November, p. 387) suggests that the Q fever organism should be called *Rickettsia burnetii* in preference to *Coxiella burnetii*, citing Derrick's homage to Burnet in 1937 in support of his contention. However, more recently Derrick himself² has acknowledged that, since differences between *burnetii* and other rickettsiae might call for a different generic name, *Coxiella* might be the more fitting; he also pleads that the name should remain uncomplicated as "*burnetii*" rather than "*burnetii*."—We are, etc.,

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Vesical Atony

SIR,—Mr. T. Moore (7 December, p. 646) raises the question of the ultimate residual urine after bladder capacity reduction operations. This is of particular significance in the treatment of bladder-neck sclerosis.¹

In 1946 Mr. Terence Millin demonstrated to me the Marion operation of "capitonnage," liberally translating this as "reefing the bladder mainsail." During the intervening 23 years, 11 cases have been so treated in my service. When biopsy of the vault at the time of operation has shown muscle tissue to be still present the preoperative residual urine has entirely disappeared, but it has seemed probable that recovery would have been complete in such cases without capacity reduction. When complete muscle atrophy of the vault has been demonstrated the residual urine has been diminished postoperatively but has never

entirely disappeared. In such cases subtotal cystectomy, removing the atrophic vault, is probably more logical in that it restores a completely muscularized organ. Preoperative cystometrograms have given no useful information in arriving at a decision, so it is probable that very few real indications for the operation will present in a urological lifetime.—I am, etc.,

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REFERENCE

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Pneumomediastinum and Diabetic Hyperpnoea

SIR,—We were interested to read the Medical Memorandum by Professor B. McNicholl and others (23 November, p. 493) describing pneumomediastinum in association with diabetic hyperpnoea. We have recently seen a similar case at this hospital.

The patient was a 20-year-old married woman, a 'diabetic of 10 years' standing, who had been well controlled by 80 units of lente insulin daily for the previous 18 months. When admitted on 4 November she was semicomatose, dehydrated, and hyperventilating grossly. No clinical signs of infection were found, but a blood count showed 32,000 white cells with a neutrophil leucocytosis. Blood glucose was 416 mg./100 ml. and serum bicarbonate 6 mEq/l. A radiograph of the chest showed widespread free air in the mediastinum and soft tissues of the neck, and inflammatory changes at the left lung base.

She responded well to treatment, though traces of mediastinal air were still present eight days after admission—a Gastrografin (sodium diatrizoate) swallow earlier had excluded oesophageal perforation. When able to give a history the patient described increasing difficulty in breathing for some days, culminating in an odd retrosternal ache, worse on inspiration, on the day before admission. She had had no cough or sputum. At no time during her illness was Hamman's sign detected.

The complication of pneumomediastinum in diabetic ketoacidosis had not previously been encountered by any of us, though J. W. Pierce¹ has noted the association on several occasions in his radiological experience at St. Thomas's and the Brompton Hospital. It seems somehow to have been overlooked in the standard texts on medicine and radiology, and deserves wider recognition.—We are, etc.,

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REFERENCE

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Chlorpromazine

SIR,—Professor W. A. Cramond recommends (23 November, p. 497) chlorpromazine for the treatment of acute confusional states "in doses of 100 mg. intramuscularly immediately, repeated in two to three hours if required for two to three more occasions." Later he states, "Elderly and debilitated patients should be given half the recommended daily dose."

I think it should be emphasized that a single intramuscular dose of chlorpromazine of 100 mg. in an elderly or debilitated patient is potentially quite dangerous. There is great potential for postural hypotension with brain softening, myocardial infarction, syncope with trauma, and even irreversible shock and death. Chlorpromazine 100 mg. by injection is about equivalent to 400–500 mg. in a single oral dose in its capacity to produce sedation and hypotension. Thus, not only the total daily dosage, but especially the maximum quantity of doses, must be halved in the elderly and infirm.

Professor Cramond rightly warns about the dangers of habituation and toxicity of barbiturates used to promote sleep. He adds, "Chloral hydrate and glutethimide are better drugs." It is hard to see why, since both in equivalent doses also produce habituation and toxic confusion in patients with organic psychoses.—I am, etc.,

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Tetracycline and Nystatin

SIR,—There is much argument about the use of a combination of an antibacterial with an antifungal antibiotic. It is important, therefore, that published evidence for or against such a combination should be convincing. This is not the case with the British Tuberculosis Association report (16 November, p. 411), which compared tetracycline with Mysteclin (tetracycline and nystatin).

Fully conscious of my amateur status as a statistician, may I venture the following comments?

(1) Because of the small number of patients in the trial it seems unlikely that statistically significant results could have been obtained. The incidence of the relevant symptoms mentioned is about 20–30% with tetracycline. For Mysteclin to give results of clinical importance it should reduce the symptom incidence to about 10–15%. If the rate with tetracycline were 20% and with Mysteclin 10%, it appears that 195 patients would be needed in each group to have an 80% chance of significance on a two-sided test at the 5% level. Yet the total number of patients in the trial is only 111—barely more than one-fourth of the required number.

(2) As I understand it, the manufacturer's claim for Mysteclin that it protects against *Candida* overgrowth in the gut and the associated symptoms, not that it cures them. Surely, therefore, patients with initial symptoms and initial *Candida* should be excluded. This would reduce the total number in the trial to 67, and would correspondingly increase the probability of a negative result.

(3) In Table IV the results before and after treatment are amalgamated so that each patient appears twice. Clearly the results on the same patient before and after treatment are not independent. Surely, however, the chi-squared test can only be applied if all entries in the Table are independent. Conclusions about the significance of the data on Table IV therefore appear to be invalid.

(4) In the symptomatology symptoms are included which do not seem necessarily to be related to the presence or absence of yeasts—e.g., flatus, abdominal pain, heartburn, nausea, difficulty in swallowing. These merely cloud the issue.

(5) Surely 10 days' treatment are too few to assess the value of Mysteclin even in a previously untreated, symptom-free and *Candida*-free group. In the miscellaneous group under trial they are even more inadequate. None the