Clinical Algorithms

Loss of vision

MICHAEL JAMIESON

A common and often major handicap, visual loss may be preventable and is often symptomatic of treatable intraocular and extraocular disease. Diagnosis of its underlying cause is often possible without specialist experience or equipment. Early diagnosis by a general practitioner or general physician may lead to early treatment and will be a useful guide to prompt and appropriate referral, avoiding time consuming and perhaps unnecessary referral to opticians and ophthalmologists. In particular, chronic glaucoma may be detected and treated early, refractive errors diagnosed and referred for retinoscopy, diabetic and hypertensive retinopathy found and appropriately managed, and disorders primarily of the central nervous system identified. This algorithm attempts to provide routes to diagnosis in cases where visual loss is the dominant symptom. Conditions in which pain predominates but where visual impairment may be present—for example, acute glaucoma, acute iritis—are not discussed. Likewise, conditions that are common world wide but rare in the United Kingdom—for example, vitamin A deficiency and infective diseases such as trachoma and onchocerciasis—are not considered further.

History

In all cases the history should take into account onset, progress, duration, and extent of visual loss. In particular, the clinician must distinguish between sudden and gradual loss, bearing in mind that gradual unilateral impairment may go unnoticed until vision becomes impaired (perhaps trivially) in the "good" eye. To distinguish between central and peripheral visual loss is also useful. Central visual loss presents as impairment of visual acuity and generally implies defective retinal image formation (through refractive error, lens, or vitreous opacity) or macular or optic nerve disease. Peripheral visual loss is more often silent but may present as an absolute or relative blind spot or as navigational difficulty (including night blindness and tendency to collide with unseen objects). Peripheral loss generally implies either diffuse non-macular retinal disease or a defect in the visual pathway (optic nerve, chiasm, radiation, and cortex). There are, however, areas of overlap as shown in the algorithm, and disorders may coexist.

The drug history may be relevant. Visual disorders that have been associated with drugs are listed in the table. The clinical importance and prevalence of these associations vary, and readers are referred to a more detailed discussion of the subject.[1] Few of these associations are predictable.

Visual disorders associated with drugs

Disorder	Drug
Corneal opacities	Chlorpropamide Chlorpromazine Hydroxychloroquine Indomethacin Vitamin D Amiodarone
Precipitation of acute narrow angle glaucoma	Antihistamines Tricyclics Mydriatic drops
Refractive changes	Thiazides
Lens opacities	Phenothiazines Corticosteroids
Retinopathy	Chloroquine/hydroxychloroquine Phenothiazines
Papilloedema (2° to benign intracranial hypertension)	Corticosteroids Tetracyclines Oral contraceptives
Optic neuropathy	Ethambutol Disulfiram Ethanol (Tobacco)

Examination

Examination should include assessment of visual acuity and visual fields, fundoscopy, and, when appropriate, pinhole testing and measurement of intraocular pressures. Near visual acuity is estimated with newsprint or, more formally, with British Faculty of Ophthalmologists reading type; distant acuity with Snellen or E charts. Confrontation testing will detect major defects in the visual field; smaller, relative, and colour defects are more likely to be missed, and formal mapping will be necessary if no field defect has been found to explain a volunteered blind spot.

Intraocular pressures should be assessed in older patients, ideally in all cases but particularly when symptoms (characteristically, "halos" seen around lights) or fundal findings suggest chronic glaucoma. With practice, digital estimation will detect a considerable increase in pressure but is unreliable in assessing smaller rises. If no tonometer is available referral to an ophthalmologist should be considered. The pinhole test is useful if refractive error is suspected: a pinhole roughly 1 mm in diameter held in front of the eye will improve acuity in those with refractive errors (but also in some patients with early corneal or lens opacities) but will tend to worsen the acuity of those with retinal or optic nerve lesions. The pupillary light reflexes are usually preserved in chronic visual loss, even in the face of severe impairment. Acutely absent ipsilateral direct and contralateral consensual reflexes suggest retinal arterial occlusion or injury to the optic nerve.

Management

Opportunities for management by the non-specialist are rather more limited, and most cases need appropriate referral. Chronic

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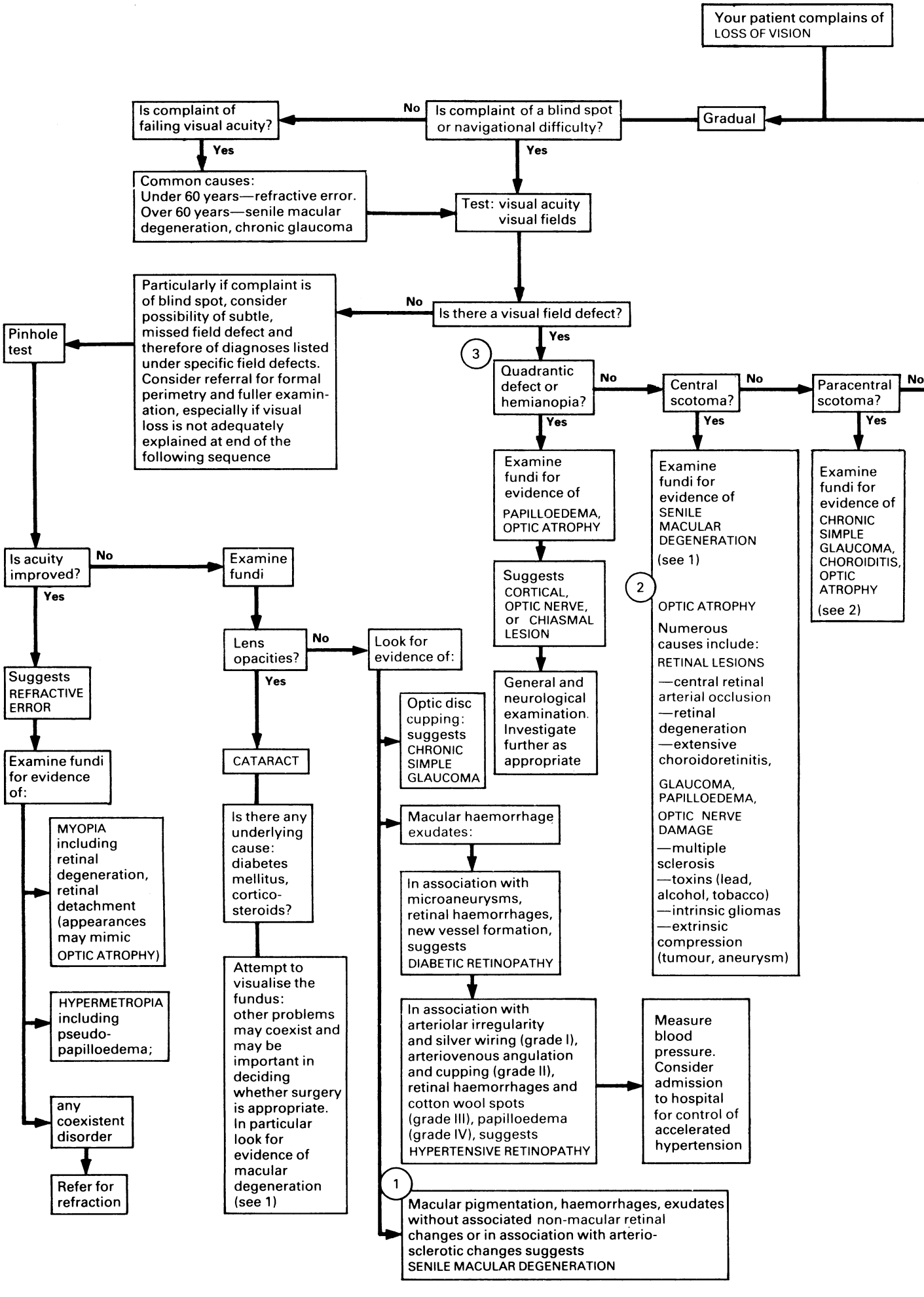
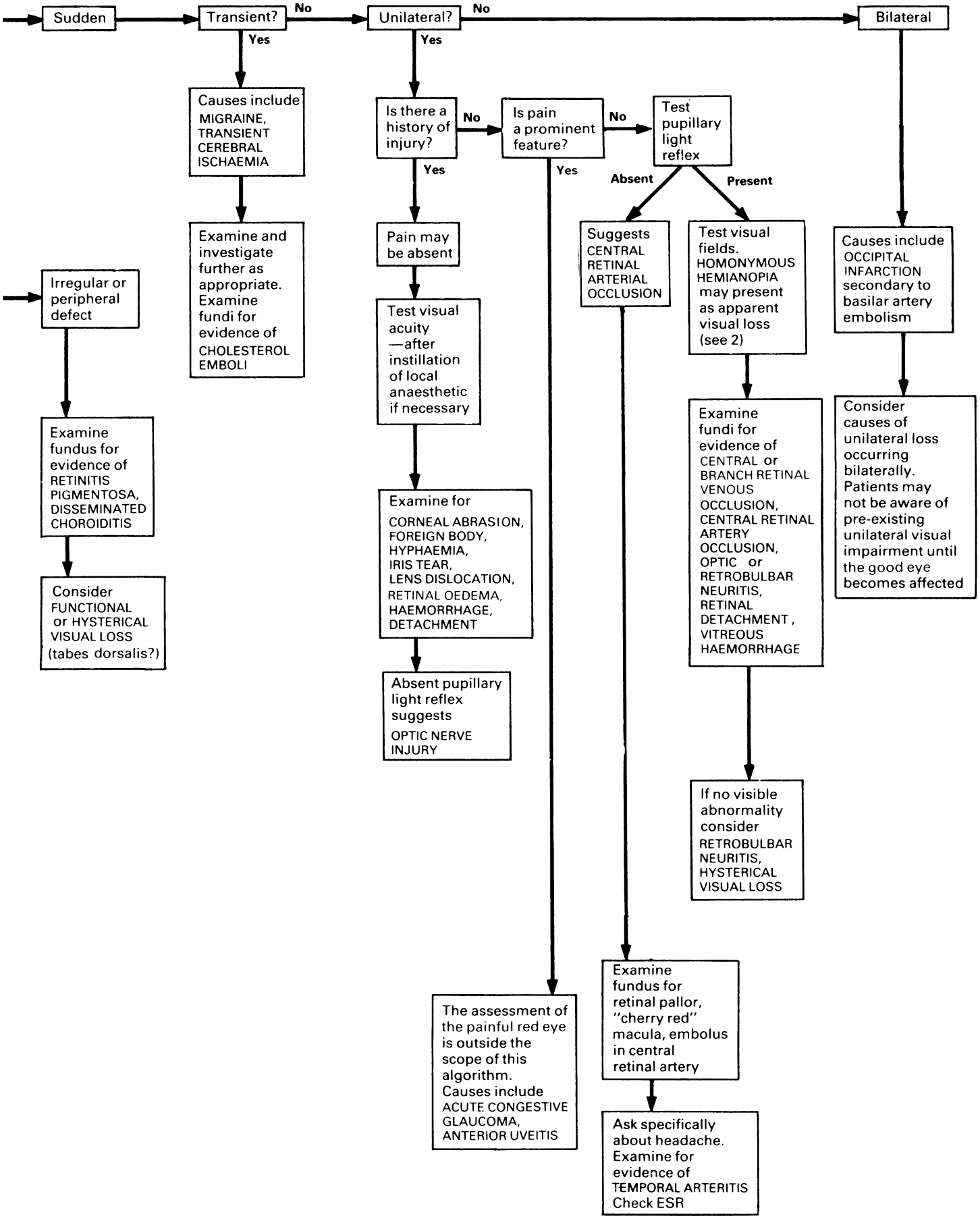


Loss of vision



simple glaucoma can be managed in general practice, preferably in conjunction with an ophthalmology clinic as regular measurements of intraocular pressure and visual field charting are necessary to assess adequacy of control. The drugs most commonly used in chronic simple glaucoma are timolol, adrenaline, pilocarpine, and guanethidine (with or without adrenaline) eye drops and oral acetazolamide (roughly in descending order of popularity). Surgery should be considered if these fail to control intraocular pressure.

Vision may be saved after central retinal arterial occlusion if effective treatment is given within three or four hours of onset and especially if the occlusion is embolic. Lie the patient flat and attempt to lower intraocular pressure by brief intermittent massage of the globe. Using both index fingers apply firm pressure—ask the patient to look down—for about 20 seconds and repeat this after an interval. (More prolonged compression may increase intraocular pressure and further impair retinal perfusion). If giant cell arteritis is a possibility systemic steroids should be given in high dose. Unfortunately vision is often lost, particularly because most patients present late, and emphasis should therefore be placed on preventing blindness in the other eye. Patients should be warned to report immediately if any symptoms develop in the "good" eye.

[1] Crombie AL. Eye disorders. In: Davies DM, ed. *Textbook of adverse drug reactions*. 2nd edition. Oxford: Oxford University Press, 1981:449-61.

Lesson of the Week

Necrobacillosis: a forgotten disease

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Abstract

Over four years five previously healthy young adults developed necrobacillosis, a severe septicaemic illness caused by Fusobacterium necrophorum. The infections were characterised by sore throat followed by rigors and the formation of metastatic abscesses and all caused considerable diagnostic confusion.

Necrobacillosis should be considered in previously healthy patients who develop severe septicaemia and pulmonary symptoms after an initial sore throat

Introduction

Necrobacillosis—infection with *Fusobacterium necrophorum*—is a severe suppurative illness that before the advent of antibiotics was often fatal. The presenting features are usually characteristic and were described in detail in 1936 by Lemierre,[1] who suggested that the diagnosis may be strongly suspected on clinical grounds alone. Despite the intense interest in anaerobic infections over the past decade *F necrophorum* is seldom specifically mentioned in reports and necrobacillosis is now rarely, if ever, diagnosed. Over four years we saw five patients with this illness, all of whom showed the classic features of the disease.

Case reports

Case 1—An 18 year old man developed a severe sore throat and over three days became very ill with rigors, pain in the back and shoulders, pleuritic chest pain, haemoptysis, and dyspnoea. On admission he had cervical lymphadenopathy and jaundice. A chest *x* ray film showed bilateral abscesses and a pleural effusion. The white cell count was $21{\cdot}4\times10^9$/l, blood urea concentration was raised at 13·9 mmol/l (83 mg/100 ml), and bilirubin concentration was 38 μmol/l (2·2 mg/100 ml). Initial treatment with intravenous erythromycin and cefuroxime produced no clinical improvement, and a week later shadowing was visible in the chest *x* ray film, which gallium scanning and computed tomography suggested was a paravertebral abscess. Metronidazole was added empirically to his treatment, and a few days later *F necrophorum* was isolated from two of the eight blood cultures taken on admission. Metronidazole was continued for two months, during which time he made a slow but complete recovery.

Case 2—A 20 year old woman developed a severe sore throat and over one week became very ill with fever, cough, abdominal pain, and dyspnoea. On admission she had bilateral cervical lymphadenopathy and jaundice. A chest *x* ray film showed extensive bilateral consolidation, which subsequently cavitated. The white cell count was raised at $17{\cdot}0\times10^9$/l, blood urea concentration raised at 38·3 mmol/l (230 mg/100 ml), and serum bilirubin concentration raised at 178 μmol/l (10·4 mg/100 ml). Treatment was initially with intravenous gentamicin, metronidazole, and ampicillin. Her abdominal pain worsened and the results of ultrasound scanning and computed tomography were highly suggestive of a perinephric abscess, but the result of laparotomy was entirely normal. Cultures of blood and pleural fluid taken on admission subsequently grew *F necrophorum* and a microaerophilic streptococcus. Metronidazole and benzylpenicillin, and then oral metronidazole and amoxycillin, were administered during her lengthy and severe illness, which was further complicated by joint effusions in hip and knee and an episode of obstruction of the small bowel. She eventually recovered completely.

Case 3—A 21 year old man developed a sore throat and over one week became very ill with high fever, pleuritic chest pain, haemoptysis, and severe dyspnoea. On admission cervical lymphadenopathy and haematuria were noted. A chest *x* ray film showed extensive bilateral consolidation and effusions. The white cell count was raised at $23{\cdot}7\times10^9$/l, blood urea concentration was normal, and serum bilirubin concentration was raised at 50 μmol/l (2·9 mg/100 ml). He improved after initial treatment with intravenous erythromycin but after a further five days again developed a fever. At this stage *F necrophorum* was

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