MEDICATION

WIDESPREAD CONFUSION

> OBSTETRICS & GYNAECOLOGY
> PSYCHIATRY

A 31-year-old housewife and mother of three, Mrs F had coped with epilepsy for many years, taking carbamazepine and phenytoin to control her condition. When she became pregnant again in the mid 1990s, she experienced fits at least three times a week. Her neurologist adjusted her medication to phenobarbitone 60mg three times daily with phenytoin 400mg daily. Her epilepsy was reasonably well controlled on this regimen.

When Mrs F went into labour and attended her local hospital, the admitting doctor recorded and prescribed her medication incorrectly. Instead of phenytoin 400mg daily, he prescribed 400mg three times daily. Nobody noticed this error.

Mrs F had a healthy baby boy, but was kept in hospital due to an episode of fever. Two days later, the nursing notes record – 'Paranoid ideation – strange behaviour'. These notes also document a complaint of buzzing in the left ear since parturition. By that evening Mrs F was obviously distressed and agitated; her obstetric team asked the on-call psychiatrist to see her and advise.

The duty psychiatrist diagnosed puerperal psychosis and ordered an intramuscular injection of zuclopenthixol with oral anticholinergics and regular chlorpromazine. Dr T, a specialist in psychiatry, reviewed Mrs F the next day, noting auditory third-person hallucinations. He recorded ideas of reference and delusions concerning the newborn baby and his father, and noted an absence of confusion, disorientation or features of an organic brain syndrome.

Over the next few days Mrs F was reviewed by several members of the psychiatry team. As her symptoms were not improving, she was transferred to the local psychiatric unit, where she was treated with haloperidol and ECT.

The ECT failed to produce any epileptiform movements. Subsequently, Mrs F had slurred speech and visual hallucinations. Noting the presence of features consistent with an organic brain syndrome, Dr T recommended omitting the night-time chlorpromazine.

Over the next few days, Mrs F was restless and agitated. After a further ECT treatment where there were again absolutely no epileptiform movements, Dr T asked a neurologist to review Mrs F. The neurologist, who saw her the next day, identified the medication error and discontinued the phenytoin.

Mrs F recovered, but her coordination and walking were impaired, and she had difficulty regaining her original confidence; these symptoms took several months to resolve.

When Mrs F brought a claim against the hospital, we consulted an expert in psychiatry on behalf of Dr T. In the expert's opinion, although her original presentation was consistent with a diagnosis of puerperal psychosis, the failure to notice the error in the phenytoin dose and the evolving features of organic abnormality – namely the absence of any seizure activity during ECT and worsening clouding of consciousness – were indefensible. One expert in psychiatry questioned the use of ECT altogether in a case such as this. Liability was shared between the psychiatric and obstetric institutes at 25% and 75% respectively.

Learning notes

- The initial error in the prescription would probably have been brought to light sooner if (a) the doctors had reviewed the patient's notes more closely, (b) the doctors had reviewed the medication chart for possible causes of Mrs F's symptoms, and (c) the nurses administering thrice-daily doses of phenytoin had questioned the prescription.
- Phenytoin has zero-order metabolism, so toxicity is a possible cause of acute illness in a patient taking this drug. For a useful tutorial on phenytoin toxicity see www.emedicine.com/emerg/.
- Psychosis is a syndrome, not a diagnosis. Acute or chronic organic brain syndrome (delirium or dementia) can present with acute psychotic features.

SYSTEMS ERROR

MENINGIOMA PRESENTING AS TRANSIENT HEADACHE AND LEG WEAKNESS

> GENERAL PRACTICE

Mr G, a 29-year-old tiler, went to see his GP, Dr K, in 1999, complaining of occasional brief spells of feeling faint when returning from work. Dr K saw Mr G again for a full examination, but found no abnormality. He did not document the specifics of his neurological or cardiovascular examination.

Mr G returned a few months later complaining of regular, short-lived frontal headaches and episodic leg weakness. Dr K conducted a full examination and found no abnormality. Again, the specific negative findings were not documented. Dr K conducted some screening blood tests.

Mr G returned the next year with the same symptoms, which seemed to be triggered by moving his neck and so a cervical spine x-ray was ordered. It was normal.

Three months later, Mr G collapsed at work and was admitted to hospital in a coma. A CT scan showed a large occipital space-occupying lesion and temporal lobe coning.

Mr G did not recover and was declared brain dead. A biopsy showed the mass to be a non-malignant meningioma.

When Mr G's family alleged negligence, we asked a GP expert to examine the case notes. The expert found an ESR result from the screening blood tests. This was grossly elevated at 178 mm/hr. The result had been stamped by the practice and filed without any plan of action being indicated. In the light of this information, the expert felt that the management of Mr G from that point on could not be supported.

This abnormal result in a man with unusual and persistent symptoms should have prompted a referral for neurological or general medical advice.

However, the expert felt that the approach had been entirely appropriate up to this point, with frequent examination and screening bloods being done in a case that had presented quite unusually. Unfortunately, these tests were not acted upon; this was the fundamental error which led us to settle the case for a sum equivalent to £250,000, once we had expert neurosurgical opinion that earlier intervention could have saved Mr G's life.

Learning notes

- A reliable system for ensuring that test results are seen and acted upon is crucial.
- Diagnosing intracranial space-occupying lesions can be difficult. The rubric below is helpful for assessing patients that could have relevant symptoms.

1. Take and document a thorough history across the range of systems that might produce such symptoms.
2. Perform a brief screening examination of the cranial nerves and document what was examined. Documentation of fundoscopy is particularly important.
3. Check tone, power, co-ordination and reflexes in upper and lower limbs and document these findings.
4. Examine any other relevant systems and document the findings.
5. Consider what investigations might help and make sure the results are reviewed.
6. Seek specialist advice if unsure.
7. Be willing to reconsider your diagnosis in the light of evolving clinical information

- For further details on clinical features of raised intra-cranial pressure, see our previous article Diagnosing Acute Headache in *Casebook* August 2003, available at www.medicalprotection.org/assets/pdf/casebook/casebook_uk_2003_3.pdf.

MEDICATION

INADVERTENT INTRA-ARTERIAL INJECTION

> GENERAL PRACTICE
> NURSING

Mr D, a gardener in southern Africa, had substance misuse problems and had seen Dr W, his GP, several times about severe headaches which Dr W had treated with intravenous injections of pethidine and hydroxyzine into the dorsum of his right hand. Mr D had difficult veins and had previously had a localised reaction to the injections. Despite this, Dr W sent him to a venepuncture clinic to have blood taken to investigate his headaches, with a request to administer i-v pethidine and hydroxyzine.

Mr D arrived at the clinic with 100mg ampoules of pethidine and hydroxyzine dispensed by his local pharmacy; he was seen by two trained nurses. Sister F's attempt to find a suitable vein was unsuccessful, but Sister T managed to site a butterfly needle on the dorsum of Mr D's right wrist. On withdrawing a small amount of blood, she remarked that it appeared very light in colour and might be arterial, so she was not prepared to give an injection. Sister F, however, thought that the blood was not arterial and so gave an undiluted injection of pethidine 100mg mixed with hydroxyzine 100mg through the butterfly needle.

Mr D winced when he was given the injection and his hand became blotchy. Six hours later, he was admitted to hospital with an extremely swollen right hand. Compartment syndrome due to an intra-arterial injection was diagnosed. Despite surgery to decompress the right forearm and hand, and administration of local thrombolytic agents in the radial artery, amputation of the hand was eventually necessary. It later transpired that the ampoule containing the hydroxyzine was marked 'for intramuscular use only'.

Mr D started a legal claim against the clinic, which was indemnified by MPS. The experts we consulted were critical of both Dr W and Sister F – Dr W for his 'bizarre and bewildering' handling of the patient, and Sister F for ignoring the risk of a possible intra-arterial injection and failing to read the label on the hydroxyzine ampoule.

According to legal opinion, full liability for the incident lay with the clinic, as Sister F's employer. Although

DIAGNOSIS

A CRUSHING OVERSIGHT

> GENERAL MEDICINE
> GENERAL PRACTICE

In the late 1990s, Mr M, a stockbroker in his fifties, was referred by his GP to see Dr J, specialist physician and cardiologist. Mr M had been feeling generally 'not right'. He had suffered a transient left-sided loss of limb power a year earlier, diagnosed as a stroke. He was overweight, smoked and had high blood pressure, which his GP had found difficult to control, hence the referral for advice. Mr M also complained of generalised weakness, myalgia, poor stamina and recent erectile dysfunction.

Dr J noted Mr M's protuberant abdomen and florid complexion. He ordered blood tests, ECG, 24-hour BP monitoring, a carotid doppler ultrasound and a CXR. Mr M was advised to stop smoking.

Dr J's notes contain the thought – '?cortisol'. When Mr M came back for his results a couple of weeks later, the carotid doppler scan showed only minor atheroma and daytime BP on treatment averaged 135/89 mmHg. Mr M felt a little better and less weak, with some recovery of his sexual function.

Six months later, Mr M came back for review and told Dr J that he was depressed, his back was aching, and his legs felt weak again. He informed Dr J that his GP had ordered some further tests. Dr J wrote to the GP and asked to be copied into any results, commenting that 'obviously he has got creatine kinase and perhaps cortisol on the way'. He noted Mr M's propensity to bruising and documented an absence of limb girdle weakness, arranging an exercise ECG. This subsequently proved to be unremarkable and a further review was arranged for six months hence.

In the meantime, Mr M's GP wrote to Dr J suggesting Cushing's syndrome as a unifying diagnosis, asking whether or not a 24-hour urinary cortisol was advisable. Dr J replied that this diagnosis could explain the weakness and abdominal distension, adding, 'I felt that perhaps his alcohol consumption was more likely to be responsible at the time I saw him'. The urinary 24-hour cortisol and dexamethasone suppression tests were consistent with a diagnosis of Cushing's syndrome due to an adrenal adenoma, which was subsequently removed.

Unfortunately, Mr M suffered long-term sequelae due to his prolonged cortisol excess and was left with multiple vertebral crush fractures, irreversible skin changes, mood disturbance, muscle weakness and erectile dysfunction. Mr M sued Dr J, alleging that he had been negligent in failing to make the diagnosis initially, and in the length of time it had taken to make it.

Our experts in general medicine and endocrinology noted several opportunities for Dr J to make the diagnosis, as part of the screening tests one would conduct to investigate the cause of refractory hypertension, and because there was evidence of osteoporosis in the form of rib fractures on Mr M's initial x-rays. Because Dr J had raised the possibility of the diagnosis after his early consultations, it was thought that there were several clear clinical pointers to the diagnosis. Several opinions supported a direct causal link between the delay in Mr M's diagnosis and his subsequent problems and symptoms. We settled the case for a sum equivalent to £210,000.

Learning notes

- For a tutorial on the various causes of Cushing's syndrome and clinical and biochemical diagnostic advice, see www.emedicine.com/emerg/.
- When investigation results fail to support your initial assumptions, be open to alternative explanations.

Dr W's treatment had been unconventional and sub-standard, Sister F's intervening negligence, which he could not have foreseen, was the cause of the amputation. We settled the claim on behalf of the clinic.

Learning notes

- Always give clear, written instructions regarding dose and method of administration.
- If a patient has difficult veins, do not delegate administration of an i-v injection unless it is under supervision.
- Make sure that all your staff and technicians are aware of the risks of inadvertent intra-arterial injections and impress on them that they should never inject if they are in any doubt about the location of a needle.
- It is worth considering setting up an intravenous infusion of crystalloid solution and seeing that it is running smoothly before administering any drugs through a venous access device.
- Always read the label to check the drug name, strength, dose and route of administration before injecting medication.
- See Ghouri et al., Accidental Intra-Arterial Drug Injections via Intravascular Catheters Placed on the Dorsum of the Hand. *Anesth Analg* 95(2):487–91 (2002).

DIAGNOSIS

SECONDARY HYPERTENSION

> GENERAL PRACTICE
> ANAESTHETICS
> GENERAL MEDICINE

Mrs S, a 55-year-old secretary, had seen her GP, Dr E, with episodes of flushing attributed to the menopause and was treated with hormone replacement therapy. She also suffered bouts of dysuria, which were treated as suspected recurrent urinary tract infections with amoxycillin. Mrs S also consulted Dr E due to low mood, tearfulness, palpitations and insomnia. Dr E noted a resting tachycardia of 100 bpm. He arranged a CXR and ECG, which were unremarkable. Over the next 12 months he treated her with the maximum doses of a range of anti-hypertensive medications. Despite this, Mrs S's BP control remained variable.

Two years later, Mrs S was admitted for a left total-knee replacement. Her anaesthetist, Dr G, assessed her preoperatively. Blood tests showed hypokalaemia of 2.9 mmol/l (NR 3.5-5.0) and her BP was elevated at 165/90 mmHg.

Three days after an uneventful operation, Mrs S's serum potassium was 2.6 mmol/l, so she was started on oral potassium-replacement therapy. Five days later, she experienced dyspnoea and vomiting; she had difficulty taking her potassium tablets due to this. Plans were made to repeat her urea and electrolytes, but this appears not to have been done.

Two nights later, the nursing records note that Mrs S had not slept well due to 'asthma'. Later that morning she had a cardiac arrest and died. A postmortem showed her death was due to acute heart failure caused by a myocardial infarction and hypokalaemia, secondary to the presence of a large supra-renal phaeochromocytoma.

Mrs S's family sued Dr E for failing to check her urea, electrolytes and renal function, and for failing to investigate the cause of her hypertension when the treatment given proved ineffective. They sued the hospital for failing to act upon the low potassium found on admission, for continuing to administer bendrofluazide, and for failing to give intravenous rather than oral potassium supplements once Mrs S started vomiting. The claim also cited the failure to monitor Mrs S's potassium level adequately.

GP experts, whilst not expecting a GP to diagnose phaeochromocytoma, thought Dr E's failure to check urea and electrolytes in a patient taking an ACE inhibitor and bendrofluazide was inadequate. They criticised his failure to record examinations and said that a hypertensive patient with a poor response to long-term, high-dose anti-hypertensive therapy should have been investigated or referred.

'Mrs S's family sued Dr E for failing to check her urea, electrolytes and renal function, and for failing to investigate the cause of her hypertension when the treatment given proved ineffective.'

An expert physician thought it likely that referral to a physician would have led to investigations seeking a secondary cause for the hypertension, and very likely a definitive diagnosis and curative intervention. He thought that the high level of catecholamines secreted by the tumour had combined with the anaesthetic to cause Mrs S's death, although another expert physician disagreed. Both experts, however, felt that Mrs S's postoperative dyspnoea and hypokalaemia had been poorly managed.

Dr G's decision to proceed with anaesthesia in a significantly hypokalaemic patient with hypertension was criticised by an expert in anaesthesia. Most of the experts felt that the postoperative monitoring and treatment of the hypokalaemia fell below a reasonable standard. They were particularly critical of the continuing bendrofluazide therapy (known as a cause of hypokalaemia) and giving oral potassium to a nauseated patient. The claim was settled for a sum equivalent to £110,000, with liability shared equally between Dr E and the hospital team.

Learning notes

Postoperative care – Postoperative symptoms like nausea are common, but they can signify serious disease not directly linked to the surgery or anaesthetic. Therefore it is imperative that such symptoms are assessed objectively and not treated symptomatically and blindly.

Anaesthetic assessment – Hypertension and hypokalaemia should have signified a need for caution, and perhaps further investigation of their cause(s), before the administration of a general anaesthetic. For a concise tutorial on anaesthesia and adrenal disorders, including advice on anaesthetising patients with known phaeochromocytoma, and how one might detect undiagnosed phaeochromocytoma before anaesthesia, see www.4um.com/tutorial/anaesth/.

Guidelines for referring hypertensive patients in primary care – In the UK, the National Institute for Clinical Excellence (NICE) has issued primary care hypertension management guidelines that advise on when to investigate or refer patients. See www.nice.org.uk. Similar information is available in the British Hypertension Society's guidelines, at www.hyp.ac.uk.

Phaeochromocytoma and hypokalaemia – There is a known association between these conditions. For an interesting case report with self-assessment questions on the clinical features of phaeochromocytoma, see Smith JC et al., A Man Presenting with Limb Weakness and Electrolyte Imbalance. *Postgrad Med* J, 75(889):691–3 (1999). Available online at http://pmj.bmjjournals.com.

MEDICATION

LOOKING OUT FOR COMPLICATIONS

> OPHTHALMOLOGY
> GENERAL PRACTICE

From age 11, Ms V had suffered painful and inflamed eyes. She was diagnosed with atopic eye disease at an ophthalmology clinic and treated with topical sodium cromoglycate. However, her visual acuity was normal. Ms V's attendance at the clinic was sporadic.

By the time Ms V was 20, her vision had deteriorated to 6/18 (L) and 6/9 (R). Two ophthalmologists independently diagnosed keratoconus. She started treatment with sodium cromoglycate and prednisolone eye drops and had contact lenses fitted. At this time, her intraocular pressure (IOP) was 12mmHg bilaterally. Her attendance at follow-up appointments remained patchy.

Two years later, Ms V was referred by her optician to see Dr J, ophthalmology specialist. He measured Ms V's IOP at 20mmHg bilaterally. By now Ms V was using cromoglycate drops in both eyes and prednisolone drops in the left eye. The severity of her atopic conjunctivitis precluded her using contact lenses. Dr J increased the prednisolone dose and explained to Ms V that her intraocular pressure needed monitoring while she was using steroids. Ms V missed her initial follow-up appointment, but returned a couple of months later, when Dr J increased her prednisolone eye drops to four times daily (Predsol forte) in both eyes. IOPs were stable.

Dr J wrote to Ms V's GPs, advising that they continue a high-dose steroid and cromoglycate regimen and that he would keep them informed of her progress. Ms V's GPs issued a prescription for Predsol forte.

Dr J saw Ms V a few months later; her eyes were much better and he planned to wean her off the steroids. Her IOPs were unchanged. Dr J did not communicate his plans to Ms V's GPs, who continued to issue regular repeat prescriptions for Predsol forte.

At her next follow up with her hospital optician for a contact lenses fitting, Ms V also saw Dr J to get her intraocular pressure checked. Dr J advised Ms V again to tail off the steroids, adding that she would need to have her eye pressures checked regularly if she stayed on the drops. He asked her to register with his clinic, which she failed to do.

Neither the optician nor Dr J wrote to Ms V's GPs after this consultation. Over the next year she attended the hospital optician regularly, but did not see Dr J; she continued to receive repeat prescriptions from her GP practice for Predsol forte eye drops. At one point she had sore eyes and saw a member of the practice who noted bilateral conjunctivitis and prescribed chloramphenicol eye drops.

Eventually, Ms V went back to see Dr J due to deteriorating vision. Dr J arranged a second opinion. After this Ms V underwent bilateral trabeculectomies to treat severe chronic glaucoma secondary to prolonged topical prednisolone administration. Ms V was left effectively blind in her left eye, with vision in the right assessed at 6/9 for distance and N5 for near. The visual field in her right eye was markedly restricted.

'Dr J did not communicate his plans to Ms V's GPs, who continued to issue regular repeat prescriptions.'

Ms V sued Dr J, alleging inadequate monitoring of her use of prednisolone eye drops and for failing to warn her of the dangers of their prolonged use and the need to monitor intraocular pressure. She also alleged that her GPs were negligent for issuing repeat steroid eye drop prescriptions without ensuring proper ophthalmological review and for not recognising the dangers of their prolonged use.

We asked for opinions from GP and ophthalmology experts. From the GP viewpoint it was felt that the issuing of repeat prescriptions was justifiable after the initial opinion from Dr J, but that they should not have been continued in the long term without ophthalmological advice. The practice had a policy of dealing with steroid eye prescriptions separately in its computer system, which seemed to have been forgotten in this instance.

The ophthalmology expert felt that Dr J's failure to communicate his treatment plans with the GPs could not be defended. Had he communicated the advice to tail off the steroids, this would have prevented their prescription and resultant damage due to prolonged iatrogenic intraocular hypertension. No blame was attached to the optician, as monitoring of steroid eye drops and IOPs was not necessarily part of his role in these circumstances. We settled the claim for a sum equivalent to £125,000 plus costs, sharing liability between the GPs and Dr J.

Learning notes

- GPs – Seek help when prescribing treatments with potential complications that are usually supervised by a specialist.
- Specialists – Provide appropriate and clear advice to GPs in such circumstances.
- Educate patients about the dangers of long-term use of such treatments.
- For a tutorial on drug-induced glaucoma, including that caused by topical steroids, see www.emedicine.com/oph/topic124.htm.

MANAGEMENT AND INTERVENTION

WARNING SIGNS

> EMERGENCY MEDICINE
> CARDIOTHORACIC SURGERY
> GENERAL SURGERY

Mr Y, a 41-year-old father of five, was involved in a high-impact, head-on road crash late at night on a country road. He was wearing a seatbelt and was able to talk to the ambulance crew when they attended. On initial assessment, his BP was 140/90 mmHg with heart rate at 120 bpm.

He was taken to a rural A&E department, then referred to the on-call surgical registrar, Dr Q, at 11.30pm. Dr Q noted Mr Y's BP to be 90/50 mmHg. After a rapid crystalloid fluid infusion this came up to 140/90 mmHg. He remained tachycardic at 140 bpm. He was correctly orientated and had a GCS of 15/15 with good peripheral perfusion and pulses.

Mr Y complained of upper back pain and pain on inspiration. He had extensive thoracic and upper abdominal skin contusions. Dr Q noted tenderness along the whole length of the spine. Chest and cardiac auscultation were normal. Dr Q inserted a urinary catheter and arranged blood group and save, baseline routine blood tests and relevant x-rays.

Mr Y's initial haemoglobin (Hb) was 13.3 g/dl. An AP CXR, performed using a portable x-ray machine at 12.05am, showed widening of the mediastinum. There was no evidence of pneumothorax. The x-rays showed fractures of thoracic and lumbar vertebrae but no other major injuries. There was no on-call radiologist available, so Dr Q interpreted the films in isolation. No out-of-hours CT scanning facility was available at the hospital. Dr Q telephoned Dr V, general surgical specialist, for advice. The time of the call was not documented.

Dr Q reportedly told Dr V of the widened mediastinum, but added that the CXR was difficult to interpret because of the AP view and Mr Y's weight. Dr V thought Dr Q was not too concerned about Mr Y because of his good general condition and normal haemoglobin. He advised admitting Mr Y to the HDU on a spinal board for close observation, with repeat Hb estimation. Dr V did not come in to the hospital to review Mr Y, but asked to be informed of any change in his condition.

Mr Y's urine output was only 126ml overnight despite a fluid input of 7.5 l. When checked at 3.20am, his Hb was 8.9 g/dl; at 6.20am it was 11.5 g/dl. His blood pressure fluctuated between markedly hypertensive and borderline hypotensive. He remained tachycardic and tachypnoeic throughout the night.

Dr V reviewed Mr Y at 7.00am, and found him confused, disorientated and clearly unwell. Dr V noted clear widening of the mediastinal outline on the CXR and arranged an urgent CT scan of thorax. Unfortunately, Mr Y had a cardiac arrest whilst en route to the scanner and died.

Mr Y's family started a legal claim against Drs Q and V and the driver of the other car involved in the crash. Our experts in emergency medicine and cardiothoracic surgery agreed the CXR was grossly abnormal and should have prompted an urgent CT scan. If this was unavailable, then urgent transfer to the nearest hospital with CT scanning and cardiothoracic surgical facilities should have been arranged. They also thought the excessive fluid input may have induced hypertension, which may have made

SUCCESSFUL DEFENCE

AN ASPIRATION TO GAIN COMPENSATION

> ANAESTHETICS
> GENERAL MEDICINE

Mr W, a 30-year-old unemployed factory worker, was admitted to hospital for an elective intra-oral surgical procedure. His preoperative anaesthetic questionnaire was unremarkable except for a history of chronic mood disorder treated with carbamazepine and lithium. He was assessed preoperatively by Dr X, anaesthetic specialist, who noted that Mr W had recently recovered from a cold.

Dr X used a standard anaesthetic induction protocol utilising fentanyl, propofol and succinylcholine. To allow surgical access to the mouth he intubated Mr W with a naso-tracheal tube.

Immediately after intubation, Dr X had difficulty ventilating Mr W and found evidence of widespread bronchospasm. He was forced to use supra-normal inspired oxygen concentrations, but Mr W's pulse-oximetry SaO_2 remained low at 80-90%; his chest was 'wet-sounding' with decreased right-sided air entry.

Dr X thought the most likely problems were drug-related anaphylaxis, pneumothorax or pulmonary collapse due to inhaling a plug of mucus or blood. He exchanged the naso-tracheal tube for an oro-tracheal tube. An on-table CXR showed consolidation/collapse of the right middle lobe. In the absence of a rash or hypotension, anaphylaxis was thought unlikely. Dr X decided to abandon the procedure after Mr W started to cough forcefully; he extubated Mr W without difficulty.

Dr X immediately sought advice from Dr R, specialist physician. Dr R found scattered crackles with decreased air entry on the right side of Mr W's chest. Mr W was apyrexial but reported dyspnoea at rest and some chest discomfort on inspiration. Dr R considered aspiration pneumonitis as the most likely diagnosis and advised treatment with supplemental oxygen, chest physiotherapy, intravenous antibiotics and hydrocortisone.

Dr R reviewed Mr W later that day. He was coughing up some blood-stained sputum but felt and looked much better.

Mr W was discharged home with oral antibiotics three days after his adverse anaesthetic event. He appeared to have fully recovered and a CXR showed some minor non-specific shadowing at the right mid/basal zone, significantly improved compared to previous x-rays.

any aortic tear worse.

The emergency medicine expert commented: 'The management of the patient was substandard. Signs of grave physiological upset indicative of serious injury were overlooked.'

The cardiothoracic surgical expert stated: 'Dr V should have come in to review the x-ray and the patient. Dr Q should have re-contacted Dr V overnight to report the low urinary output, the subsequent haemoglobin results and the raised heart and respiratory rates.'

Given the above criticisms, both Dr Q and Dr V had probably acted negligently. Even so, it was uncertain whether or not early CT scanning and surgery would have saved Mr Y's life, as the logistics of transfer and work-up for theatre would have taken some hours. However, his relative youth, lack of significant co-morbidity and initial stability through the first night after his injuries indicated that he had at least an outside chance of survival. The case was settled for a sum equivalent to £325,000. The insurers of the at-fault driver paid 50% of the settlement. Drs V and Q were held to be 33% and 17% liable respectively.

Learning notes

- The use of protocols for managing trauma patients has been shown to improve outcomes.
- If you work in an emergency department, familiarise yourself with your local, current guidelines and protocols.
- For a useful overview of diagnostic and management issues in cases of chest trauma, see the following extract from the New York State University's Department of Surgery's trauma handbook at www.uhmc.sunysb.edu/surgery/edu.

Mr W sued Dr X, alleging that he had caused permanent damage to his trachea and lungs in the way he had used his equipment and that his suitability for anaesthesia had not been adequately assessed. Mr W also claimed that he was not adequately monitored during anaesthesia, that an anaesthetic was given when Dr X should have known that there was fluid or phlegm in his lungs, and that Dr X had given him insufficient oxygen.

An anaesthetic expert thought that Mr W had suffered aspiration of blood from the nasal passages or gastric contents into the right lung, or possibly atelectasis due to the presence of a mucus plug. The expert could find no evidence of negligent or sub-standard conduct by Dr X in the way that he had administered the anaesthetic or managed its complications. He had kept good records of the course of events, his actions, clinical thought processes and Mr W's physiological parameters. The expert thought it unlikely that Mr W had suffered any permanent injury as a result of the anaesthetic.

Similarly, an expert respiratory physician found no clinical, spirometric or radiological evidence of any harm to Mr W's respiratory system.

It was noted that Mr W complained of difficulties in breathing but it was felt that there was no organic physical cause for these symptoms. Rather, Mr W's mood and his weight disorder were more relevant to his symptoms. We rebutted the claim.

Mr W's solicitors continued to pursue the case, obtaining expert opinion suggesting that gastric intubation may have caused the problems. Our experts reviewed the x-rays and notes. They agreed that there was no evidence to support this hypothesis, and that such an event would still be entirely defensible given Dr X's management of events. Shortly afterwards Mr W and his legal representatives withdrew the claim before it went to court.

Learning notes

Defence – Dr X's rapid and appropriate management of the complication, his good documentation and timely involvement of a colleague from another specialty aided his defence.

Recent review papers – Asai T., Editorial II, Who Is at Increased Risk of Pulmonary Aspiration? *Br J Anaesth*, 93(4):497–50 (2004). Kalinowski CP and Kirsch JR. Strategies for Prophylaxis and Treatment for Aspiration. *Best Pract Res Clin Anaesthesiol*, 18(4):719–37 (2004).

MANAGEMENT AND INTERVENTION

MOOT MYELOPATHY

> ORTHOPAEDICS
> NEUROSURGERY

Mr U, a 50-year-old dentist, had suffered from symptoms of cervical myelopathy – due to spinal canal stenosis and intervertebral disc protrusion – for five years. The diagnosis had been confirmed by magnetic resonance imaging (MRI). He had chronic neck pain, numbness and weakness of the left arm and fingers, and a 'sluggish' left leg; these problems interfered with his ability to do his job.

His local orthopaedic surgeon sent him to a tertiary-referral centre in 1999 to see Dr R, a specialist in cervical spine surgery. Dr R agreed that Mr U's symptoms, signs and radiological findings were sufficient indications to operate.

Mr U received detailed counselling before consenting to undergo decompression and fusion of the relevant cervical disc space, using an iliac crest autograft and metal plate. The surgery took place without incident. Mr U received postoperative dexamethasone to minimise the chance of inflammatory pressure on the cord, given the pre-existing stenosis.

Postoperatively, Mr U complained of a drooping eyelid and global limb weakness. Dr R documented that Mr U's hand movements were stronger compared to preoperatively. His registrar noted some distal upper- and lower-limb weakness with hyperreflexia and an extensor plantar response on the left. Later that day, Mr U was seen by a neurophysiologist, who noted signs consistent with hemi-compression of the cord, one spinal level below the surgical site.

'The symptoms and signs continued, so Mr U had an MRI scan on the third postoperative day.'

The symptoms and signs continued, so Mr U had an MRI scan on the third postoperative day. This revealed that the bone graft used was protruding into the spinal canal and compressing the thecal contents. The disc at the level below the surgery was also causing a central cord compression.

Dr R took Mr U back to theatre and revised the procedure, removing the initial bone graft and fusing the spine one level below the original site, fashioning new grafts at each level. Mr U did well postoperatively and was discharged three days later. Postoperative MRI scans showed no evidence of cord compression, and a successful fusion procedure.

Mr U last saw Dr R three months after the surgery. He had engaged enthusiastically in physiotherapy and was doing well, but still had difficulty with fine movements in his hands and some weakness of the left leg. He was able to do a limited amount of dentistry.

Mr U started a legal claim against Dr R, alleging that fashioning a bone graft that protruded into the spinal canal, in a patient with known spinal canal stenosis, represented poor surgical technique. It was held that an intraoperative radiograph should have been done to ensure that the graft was correctly positioned. Mr U's legal team alleged that the three-day delay in imaging and revising the graft site had damaged Mr U's cervical cord and left him with disabling locomotor and sensory dysfunction.

We obtained a range of expert views from spinal surgery, radiology and neurology, as did the claimant. Some were largely supportive of Dr R, others less so. The areas of dispute concerned:

- The soundness of some of the operative techniques used (particularly the construction and measurement of the graft and the use of intraoperative radiological imaging).
- The significance of the protruding graft to any damage done to Mr U's cord.
- The contribution of the delay in imaging the operative site, and its

subsequent revision, to the long-term symptoms suffered by Mr U.

In light of this, a joint report for the claimant and MPS was obtained from an eminent academic surgeon working in this field.

He thought Mr U's cord had become permanently damaged, and that he was vulnerable to this, given the preoperative spinal canal stenosis and disc abnormalities. There was evidence to support damage occurring both before and after the surgery. It was impossible to distinguish what contribution to Mr U's symptoms these two sources of damage had caused. On balance of probabilities, however, the expert felt that the surgical damage had significantly affected Mr U and had caused his inability to be unable to carry out certain activities, 'perhaps ten years earlier than would have been the case if the operation had been an uncomplicated success'.

On this basis we decided to admit that Dr R may have been liable for a degree of Mr U's problems. Given the contentious evidence on the causal relationship between any breach of duty by Dr R and Mr U's symptoms, we were able to negotiate a limited out-of-court settlement. We paid Mr U a sum equivalent to £3,250 in compensation, and costs in excess of £5,000.

Learning notes

- This case demonstrates some of the difficulty in assessing and defending medico-legal claims. We rely on expert witnesses to give opinions on the merits of a case and the issues are often not straightforward. We do our best not to 'roll over' when claimants allege damage that may not be the clinician's fault, settling cases reasonably when it appears that a patient has been harmed by medical treatment.
- It is essential to take early action to investigate and, if necessary, treat any new symptom complex which occurs after a surgical or invasive procedure. Even if investigations reveal that nothing significant has changed, it is better to have this knowledge than delay any investigation and find out at a later stage that you should have been more decisive.

SUCCESSFUL DEFENCE

SCARRED FOR LIFE?

> ORTHOPAEDICS

Rachel broke her left clavicle when she was an infant. Unfortunately, it healed poorly and became a malunited pseudarthrosis. In 1997, aged 16, she was tired of the pain, swelling and unsightliness this caused her and she saw Mr C, specialist orthopaedic surgeon.

Mr C outlined the available treatment options, namely leaving things as they were or having surgery to correct the problem. He took care to explain to Rachel and her mother that an operation might leave her with a large scar and that a metal plate would have to be inserted to fix the bone. Rachel was advised the surgery was likely to improve her symptoms, but that Mr C could not guarantee that she would be symptom-free or entirely happy with the final appearance after surgery.

Rachel decided to have open reduction and internal fixation of her clavicular fracture; the surgery was uneventful. Mr C used subcuticular dexon sutures to minimise scarring. Postoperative x-rays showed the fracture had united satisfactorily with good alignment. During her follow up, Rachel complained of intermittent pain over her clavicle, but she had a full range of movement at the shoulder.

Eleven months after the original surgery, Mr C removed the plate to see if this would reduce the pain Rachel had been experiencing. At operation, the old fracture was seen to be firmly united and well-aligned. At her final review before discharge, six weeks after the revision surgery, Rachel was asymptomatic and appeared happy with the outcome.

Five years passed, then Rachel suddenly initiated legal proceedings against Mr C. She alleged he had failed to warn her of possible unsightly scarring, or that she would be left with a cosmetic appearance, shoulder dysfunction and pain worse than those caused by her original condition. She alleged this caused her life-impairing psychological problems for which she should be compensated.

We obtained an expert orthopaedic opinion, which was very supportive of Mr C's counselling and treatment of Rachel. The expert had no criticism of the techniques Mr C had used and felt that he had done all he could to minimise scarring. It was felt that any scarring was inevitable, as good as could be expected, and not due to any deficiency on Mr C's part. The expert was sceptical about the nature and severity of the symptoms of which the claimant complained.

We sought expert psychiatric opinion; he thought a large degree of the claimant's unhappiness and lack of confidence, allegedly due to her scar, was actually due to recent adverse life events she had suffered.

According to expert cosmetic-surgical advice, the scar was significant and noticeable, but not unusual for this area of skin. There was no keloid formation and the cosmetic surgeon thought the scar's appearance might improve over time, but that surgical intervention would be unlikely to help the situation. We rebutted the claim; it was discontinued shortly afterwards.

Learning notes

- Where surgery is being considered as a way of relieving chronic symptoms or improving a cosmetic appearance, it is important to ensure that the patient is fully aware of the range of possible outcomes and their likelihood. These discussions should be documented in the notes, along with a note on the use of any leaflets, photographs or other materials.
- We do our utmost to defend members from frivolous or unjustified claims, even where it incurs significant expenses. This is to protect the interests and reputation of members and to discourage unfounded claims.