

CASE REPORTS

Childhood asthma predisposes to spontaneous pneumomediastinum

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Spontaneous pneumomediastinum is a rare condition for which potentially life threatening differential diagnoses must be excluded. A case is presented of a young man with a history of severe childhood asthma, who was successfully treated conservatively.

The radiographic presentation of air within the mediastinal planes is usually an indication of serious breach of an intrathoracic luminal structure. The unprovoked appearance of pneumomediastinum demands exclusion of such serious pathology before a course of conservative management can be safely pursued. A case of spontaneous pneumomediastinum in a patient with a history of asthma in childhood is presented.

CASE REPORT

A 22 year old man presented to the accident and emergency department with progressive dyspnea and neck pain and a two day history of non-productive cough and malaise, associated with cold sweats but no vomiting. He also had increasing dull chest pain, exacerbated by coughing, talking, breathing, and swallowing. He had multiple previous stays in hospital for childhood asthma but was not taking current medication and denied recreational drug use. He was afebrile and examination revealed a resting tachycardia with distant heart sounds but a normal blood pressure. There was no evidence of Hamman's crunch. Respiratory excursions and air entry were globally decreased but additional breath sounds were absent. No clinical signs of chest infection were found.

Haematology and biochemistry tests were normal but chest radiography revealed a pneumomediastinum with no associated pneumothorax. Gastrografin swallow and computed chest tomography (fig 1) showed extensive pneumomediastinum but no evidence of contrast leak or pleural effusion. The patient was treated conservatively with intravenous fluids and antibiotics and denied oral intake. He subsequently developed cervical surgical emphysema but vital signs remained stable and he tolerated a light diet the following day. He was discharged after three days. At one week's outpatient follow up, the patient was well, with no further chest pain or dyspnea. His chest was clinically clear and chest radiography was normal.

COMMENT

Spontaneous pneumomediastinum is reportedly rare but may be an under-diagnosed condition. Chest pain and dyspnoea are the usual presentations.¹ Subcutaneous cervico-facial emphysema and Hamman's auscultatory sign, named after

the author who first described the condition, are common findings.

Manual straining, coughing bouts,^{1,2} sneezing,³ and inhalational drug use⁴ are prelude to spontaneous pneumomediastinum development. The mechanism in common is sudden increased intrathoracic pressure induced by an effected Valsalva manoeuvre.⁵ Consequent rupture of marginal pulmonary alveoli, allowing bubbles of air to dissect along the vascular sheaths and connective tissue planes to the mediastinum has been shown by perfluorocarbon radiographic imaging.⁶ Although our patient did not have acute bronchospasm, his history of severe childhood asthma suggests airway hyper-reactivity, previously described to be associated with spontaneous pneumomediastinum.⁷ Peripheral alveolar air trapping and air pressure surges with coughing are suspected to have lead to alveolar rupture.

On its own, spontaneous pneumomediastinum is usually a benign, self limiting condition and the treatment is conservative.² Rarely, life threatening tension pneumopericardium may occur. Importantly, conditions carrying high mortality and morbidity such as bronchial or oesophageal rupture must be excluded by endoscopy and contrast radiography. Associated tension pneumothorax must also be excluded. Recurrence is uncommon.²

In conclusion, spontaneous pneumomediastinum should be suspected in young patients with a history of asthma presenting with chest pain. It is largely a benign condition best treated expectantly, but serious pathology such as bronchial or oesophageal rupture or associated tension pneumothorax or pneumopericardium must be excluded.



Figure 1 Computed tomography showing spontaneous pneumomediastinum.

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REFERENCES

- 1 Halperin AK, Deichman RE. Spontaneous pneumomediastinum: a report of ten cases and review of the literature. *N C Med J* 1985;46:21-3.

- 2 Abolnik I, Lossos IS, Breuer R. Spontaneous pneumomediastinum. A report of 25 cases. *Chest* 1991;100:93-5.
- 3 Dechambre S, d'Odemont JP, Cornelis JP, et al. Spontaneous pneumomediastinum after sneezing. *Ann Thorac Surg* 1995;60:1457.
- 4 Mattox KL. Pneumomediastinum in heroin and marijuana users. *JACEP* 1976;5:26-8.
- 5 Shyamsunder AK, Gyaw SM. Pneumomediastinum: the Valsalva crunch. *Md Med J* 199;48:299-302.
- 6 Jamadar DA, Kazerooni EA, Hirschl RB. Pneumomediastinum: elucidation of the anatomic pathway by liquid ventilation. *J Comput Assist Tomogr* 199;20:309-11.
- 7 Dekel B, Paret G, Szeinberg A, et al. Spontaneous pneumomediastinum in children: clinical and natural history. *Eur J Pediatr* 1996;155:695-7.

Face value?

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A 13 year old boy presented to accident and emergency with his mother. He claimed he had fallen, hitting his nose on the pavement. His initial epistaxis settled and he was left with a small laceration on the side of his right nostril (see fig 1). There was no swelling or trismus and the general examination was normal. The attending doctor was called away and on return noticed a swelling over his left cheek, which was now mildly tender. Radiographs were performed at this point (fig 2A and B). The boy still denied any other history despite being shown the radiographs. Eventually the mother was persuaded to leave the room and it was only then that the child admitted to being shot in the

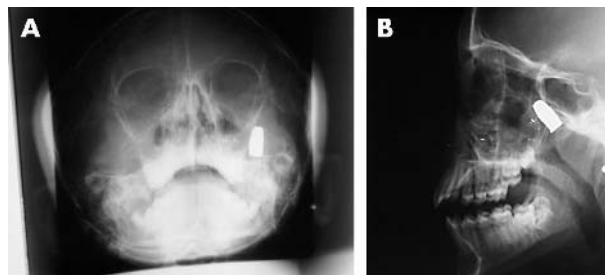


Figure 2 (A) AP and (B) lateral radiographs.

face with an altered blank bullet, while playing with his friends.

A subsequent CT scan showed the bullet lying superomedial to the cornoid process of the left mandible. It had passed through the nasal septum and both walls of the left maxilla. His symptoms settled completely and his lead levels remained constant, so the decision was to leave the bullet in place as it often is.¹

The main lesson to draw from the case was, despite the seriousness of the incident, young adolescents often withhold crucial information when a parent or guardian is present. Thus in some situations a more accurate history can be obtained with the child alone.²

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REFERENCES

- 1 Demetrides D, Chahan S, Gomez H, et al. Initial evaluation and management of gunshot wounds to the face. *J Trauma* 1998;45:39-41.
- 2 Purcell JS, Hergenroeder AC, Kozinetz C, et al. Interviewing techniques with adolescents in primary care. *J Adolesc Health* 1997;20:300-5.

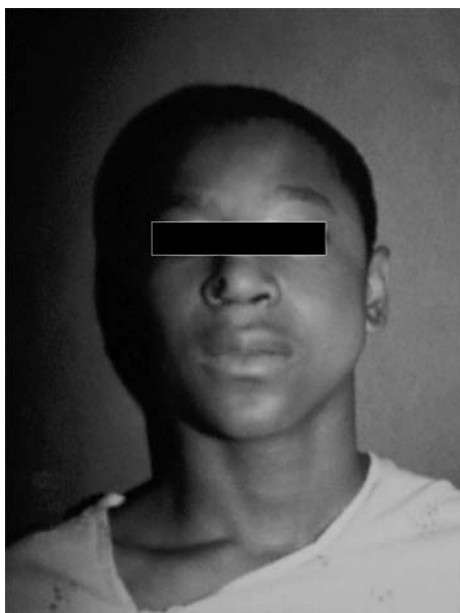


Figure 1 Photography one day after injury.

Propofol combined with lorazepam for severe poly substance misuse and withdrawal states in intensive care unit: a case series and review

K Subramaniam, R M Gowda, K Jani, W Zewedie, R Ute

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A safe, rapid, and effective way to detoxify patients from substance misuse is important in facilitating further treatment of their psychiatric or substance use disorder. This paper discusses the treatment of acute withdrawal from polysubstance misuse in three patients in the intensive care unit setting using combined sedation with a benzodiazepine, lorazepam, and a general anaesthetic, propofol. Lorazepam alone was not effective in massive doses in these cases. The advantages and mechanism of action of using multiple agents to control refractory symptoms is discussed.

Substance misuse remains a major health problem in the United States. In a study of the economic costs to society of alcohol and drug misuse, the National Institute on Drug Abuse and the National Institute on Alcohol Abuse and Alcoholism of the National Institutes of Health estimate the cost to be \$246 billion in 1992, the most recent year for which sufficient data were available. Alcohol misuse and alcoholism generated about 60% of the estimated costs (\$148 billion), while drug misuse and dependence accounted for the remaining 40% (\$98 billion).¹ Among the drugs, heroin and cocaine were the largest contributors.

Several pharmacological approaches for managing withdrawal have been suggested based on the substance used. Benzodiazepines remain the gold standard for alcohol withdrawal delirium and methadone is often used to prevent opioid withdrawal syndrome.² Propofol, an intravenous anaesthetic agent, used for sedation in the intensive care unit is easily titrated to the effect and consciousness returns rapidly after withdrawal of the drug with minimal residual central system effects.³ The use of multiple agents acting on different neurotransmitter systems reduces the required dose and side effects of one drug while blocking the symptoms of withdrawal effectively. In this paper, we describe our experience of combination therapy with propofol and lorazepam in three patients presented to the intensive care unit with poly substance misuse and severe withdrawal symptoms, which were not controlled by large doses of lorazepam alone.

CASE REPORTS

Case 1

A 42 year old man was admitted to the intensive care unit (ICU) because he was found unresponsive after an attack of tonic clonic seizures. He had a history of chronic alcoholism and his last alcohol intake was two days before admission. Physical examination was significant for asterixis and an enlarged liver. The alcohol concentration on admission was less than 1 g/l. Other significant laboratory findings were a low magnesium, phosphorus and moderate increase of

transaminase activities. He was given lorazepam (Ativan, Wyeth-Ayerst Laboratories, USA) 2 mg boluses for agitation as needed. After about an hour of presentation he woke up but was restless, agitated, and extremely tremulous. He was tachycardic, tachypnoeic, and hypertensive. He continued to deteriorate and he developed delirium tremens (DTs). He needed deep sedation with lorazepam and was electively intubated for airway protection. The rate of infusion was titrated using a 5 point sedation score (4—completely awake and open eyes, 3—drowsy, closed eyes; 2—asleep but responds to verbal commands; 1—asleep but responds to touch or pain; 0—does not respond). He required progressively escalating doses of lorazepam and the DTs remain uncontrolled despite lorazepam given at the rate of 15 mg/h. After 24 hours, lorazepam infusion was reduced to 2 mg/h and propofol (Diprivan, AstraZeneca Pharmaceuticals, USA) infusion was given at a rate of 10 µg/kg/min. Propofol was titrated up to achieve the symptom control and he required a maximum dose of up to 50 µg/kg/min. With this regimen, we could maintain tranquility and stable haemodynamics for 24 hours. In the morning every day, the propofol infusion was stopped for half hour and the patient was assessed for agitation, tremors, sweating, hallucinations, and orientation/clouding of consciousness. On the third day of propofol infusion, the patient remained calm, opened his eyes, oriented, and was properly responding to verbal commands. It took only 15 minutes for the patient to wake up after stopping propofol infusion. He remained haemodynamically stable and was successfully weaned off from the mechanical ventilation and extubated. The patient's ICU stay was complicated because he developed pneumonia, which was treated with empirical antibiotics. Once the patient started taking orally lorazepam infusion was stopped and oral chlordiazepoxide 25 mg every eight hours was started and the patient was transferred to the general medical floor.

Case 2

A 36 year old man with history of psychotic illness attributed to longstanding alcoholic and intravenous drug misuse came to the emergency room with right leg cellulitis. His alcohol concentration on admission was 265 mg% and his urine for opioid was positive. He was admitted to the general medical floor and treated with antibiotics and haloperidol 10 mg twice daily. He also received methadone 50 mg every day and was given chlordiazepoxide 24 hours after admission at 75 mg every six hours (alcohol concentrations less than 10 mg%) to prevent alcohol withdrawal symptoms. He became increasingly agitated requiring 100 mg chlordiazepoxide every hour and was transferred to the ICU. In the unit, he was given a lorazepam infusion and the patient was symptomatic with doses more than 10 mg/h. He was

Abbreviations: ICU, intensive care unit; DT, delirium tremens

intubated for airway protection. Propofol infusion was started immediately after intubation (first ICU day and 72 hours after admission). Lorazepam was reduced to 2 mg/h and propofol infusion was titrated up to control agitation and achieve the appropriate level of sedation. Propofol was continued for four days at the maximum dose of 100 µg/kg/min. On the fifth day, the patient started responding appropriately to verbal commands without agitation or tremors after stopping propofol infusion. He was extubated and observed in the ICU for an additional eight hours. Lorazepam was stopped and he was then transferred to the general medical floor where he was given chlordiazepoxide, haloperidol, and methadone. He was referred to substance misuse rehabilitation programme. He had transient increase in hepatic transaminases during his ICU stay. Hepatitis profile and abdominal ultrasound were negative. The enzymes returned to normal baseline values upon discharge from intensive care unit.

Case 3

A 39 year old woman with a history of moderate persistent asthma came to the emergency room with acute onset of respiratory distress and received inhaled bronchodilators and corticosteroids. However, she did not respond very well and her arterial blood gas pressure showed uncompensated respiratory acidosis. She was persistently tachypnoeic, tachycardic, and in severe distress with minimal air entry to the chest. She was intubated and mechanically ventilated. She received lorazepam 2 mg bolus and given an infusion titrated to sedation. Her bronchospasm subsided but she continued to be diaphoretic, tachycardic, agitated, and restless. Urine toxicology was positive for cocaine, opioids, and methadone. We suspected drug withdrawal syndrome. She required increasing doses of lorazepam up to 13 mg/h. We gave the patient clonidine 0.1 mg three times a day and methadone 30 mg/day. This regimen did not affect the patient's agitation. We decided to give propofol in low doses 10 µg/kg/min. We continued lorazepam at 2 mg/h and propofol was required up to 60 µg/kg/min for 36 hours. The patient was weaned off from the ventilator and sedation and extubated at this time. After extubation she was observed for 24 hours before being discharged with bronchodilators She was referred to a substance misuse programme.

DISCUSSION

Symptoms of alcohol withdrawal typically begin within 4 to 12 hours after cessation or reduction of alcohol use, peak in intensity during the second day of abstinence, and generally resolve within four to five days. Symptoms of alcohol withdrawal include tremors, autonomic dysfunction (tachycardia, tachypnoea, fever, and sweating), insomnia, restlessness, agitation, anxiety, panic attacks, and gastrointestinal upset. Fewer than 5% of patients withdrawing from alcohol progress to delirium tremens; a condition that has significant morbidity and mortality.⁴ Delirium tremens is a state of confusion accompanied by visual, tactile, and auditory hallucinations. Cocaine withdrawal symptoms are mild and no specific pharmacological treatment is indicated on a regular basis. Although the untreated opioid addict experiences significant anxiety and discomfort, the process itself presents no serious risks.² The classic symptomatology and the typical onset and duration of symptoms of alcohol withdrawal may be modified by the concurrent misuse of other substances such as heroin and cocaine.

Benzodiazepines are the mainstay of treatment for alcohol withdrawal states. Alcohol withdrawal delirium may require large doses of benzodiazepines (such as 1000 mg diazepam).^{5,6} There are reports in which massive doses of benzodiazepines failed to prevent or shorten the duration of

DTs.^{7,8} Benzodiazepines bind at the GABA benzodiazepine receptor in the central nervous system and when these receptors are saturated additional drug cannot bind. These patients may tolerate these exceedingly higher doses but not necessarily benefit from them.⁶ Residual sedation and prolonged rehabilitation may increase the hospital stay in these patients because benzodiazepines are sequestered in fat stores after high dose therapy.⁹ Propylene glycol toxicity marked by acidosis, tubular necrosis leading to acute renal failure has been reported with higher doses of lorazepam or prolonged infusion of the drug.^{10,11} Considering these disadvantages, there is a need for an alternate and additive drug in the treatment of these patients.

Coomes *et al* first reported the successful use of propofol in DT in a patient refractory to massive doses of benzodiazepines.¹² McGowan *et al* concluded in their case series that propofol should be considered as a therapeutic option in patients with refractory DT.⁸ All of their patients had improved symptom control with propofol. Benzodiazepines act only through GABA receptors. Propofol is synergistic with lorazepam at GABA receptors. In addition, propofol inhibits NMDA subtype of glutamate receptors, which explains its superiority over benzodiazepines.¹³ NMDA-receptors seem to play a central part in alcohol and other substance dependence, withdrawal states, and alcohol induced neurological disorders.^{14,15} The mechanism of action of propofol in opioid withdrawal states may also be related to NMDA receptors or this could just be a general anaesthetic effect. Propofol has been successfully used for sedation during rapid opioid detoxification.^{16,17} There is no pharmacological evidence for propofol acting at opioid receptors.

In our case series, we added propofol and reduced the dose of lorazepam once we realised the resistance of symptoms to high dose lorazepam. Lorazepam and chlordiazepoxide were preferred to midazolam because of their intermediate half life and benefit in the prevention of seizures and a much more smoother withdrawal course in these patient populations with less breakthrough symptoms after short acting propofol has been stopped.¹⁸ Carrasco *et al* have shown the synergistic effects of co-administration of midazolam and propofol for sedation after coronary artery bypass surgery. Combination therapy provided safe and effective sedation with advantages over conventional regimen with propofol or midazolam administered as sole agents, such as absence of haemodynamic impairment, >68% reduction in maintenance dose, and lower pharmaceutical cost.¹⁹ Reduction in dose and duration of propofol infusion with combined sedation will avoid problems of long term high dose propofol sedation such as higher cost, hypertriglyceridaemia,²⁰ pancreatitis,²¹ severe protracted metabolic acidosis,²² haemodynamic impairment and delayed seizures.²³ Currier *et al* described a patient who developed alcohol withdrawal syndrome in the postoperative period after internal fixation of ankle fracture.²⁴ The patient's symptoms were not controlled by midazolam or propofol and required muscle relaxation and mechanical ventilation. The authors explained the refractoriness of withdrawal symptoms by the acute tachyphylaxis to propofol because the patient initially responded to small doses of propofol and failed to respond later on even with 1000 µg/kg/h of propofol infusion. We did not encounter propofol related complications in any of the three patients.

Various scales have been described to evaluate the severity of withdrawal symptoms such as the objective opiate withdrawal scale, the subjective opiate withdrawal scale,²⁵ and the revised clinical institute withdrawal assessment for alcohol scale.²⁶ These scales may be useful in patients with mild withdrawal symptoms in the general medical floor but were very difficult to use in ICU patients with severe symptoms requiring very deep sedation, endotracheal intubation, and

mechanical ventilation. These scales also will not be useful in patients with psychotic disorders such as case 2. However, we used sedation scoring, which is simple to use by our nursing staff to titrate the drugs to control the symptoms such as agitation, restlessness, hallucinations, sweating, and tremors. It was aimed to achieve symptom control with a sedation score of 2 in all the patients. None of the patients responded and sedation was deepened to score 1.

All patients became awake and responding after 15–20 minutes of stopping propofol infusion. This is an advantage over the other drugs studied in the treatment of alcohol withdrawal syndrome such as flunitrazepam, chlor-methiazole, haloperidol, and clonidine.²⁷ Haloperidol can aggravate delirium and lower seizure threshold in these patients.^{28, 29} In combination with benzodiazepines, the duration of mechanical ventilation may be prolonged and a small percentage of patients may develop neuroleptic malignant syndrome.²⁷ Clonidine is less effective in suppressing hallucinations and may increase the cardiovascular complications.²⁷ We used haloperidol in case 2 because of his psychiatric history and clonidine was used in case 3 to suppress autonomic signs.

In conclusion, propofol added to lorazepam is safe and effective in controlling the symptoms of acute withdrawal syndrome associated with poly substance misuse. Multimodal therapy using benzodiazepines, propofol, haloperidol, clonidine, and methadone should be tried in these patients to reduce the required dose and side effects of any individual drug. Further well controlled clinical studies are recommended with various combinations of drugs to find out an ideal cost effective regimen to treat these patients requiring ICU admission.

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REFERENCES

- Holland P, Mushinski M. Costs of alcohol and drug abuse in the United States, 1992. Alcohol/Drugs COI Study Team. *Stat Bull Metrop Insur Co* 1999;**80**:2–9.
- Fingerwood MI. Illicit and therapeutic use of drugs with abuse liability. In: Barker LR, Burton JR, Zieve PD, eds. *Principles of ambulatory medicine*. Maryland: Williams and Wilkins, 1999:264–88.
- Smith I, White PF, Nathanson M, et al. Propofol: an update on its clinical use. *Anesthesiology* 1994;**81**:1005–45.
- Yost DA. Alcohol withdrawal syndrome. *Am Fam Physician* 1996;**54**:657–64.
- Wolf KM, Shaughnessy AF, Middleton DB. Prolonged delirium tremens requiring massive doses of medication. *J Am Board Fam Pract* 1993;**6**:502–4.
- Nolop KB, Natow A. Unprecedented sedative requirements during delirium tremens. *Crit Care Med* 1985;**13**:246–7.
- Miller FT. Protracted alcohol withdrawal delirium. *Subst Abuse Treat* 1994;**11**:127–30.
- McCowan C, Marik P. Refractory delirium tremens treated with propofol: a case series. *Crit Care Med* 2000;**28**:1781–4.
- Mirski MA, Muffelman B, Ulatowski JA, et al. Sedation for the critically ill neurologic patient. *Crit Care Med* 1995;**23**:2038–53.
- Laine GA, Hossain SM, Solis RT, et al. Polyethylene glycol nephrotoxicity secondary to prolonged high-dose intravenous lorazepam. *Ann Pharmacother* 1995;**29**:1110–14.
- Cawley MJ. Short-term lorazepam infusion and concern for propylene glycol toxicity: case report and review. *Pharmacotherapy* 2001;**21**:1140–4.
- Coomes TR, Smith SW. Successful use of propofol in refractory delirium tremens. *Ann Emerg Med* 1997;**30**:825–8.
- Orser BA, Bertlik M, Wang LY, et al. Inhibition of propofol of the NMDA subtype of glutamate receptor in cultured hippocampal neurons. *Br J Pharmacol* 1995;**116**:1761–8.
- Davis KM, Wu JY. Role of glutamatergic and GABAergic systems in alcoholism. *J Biomed Sci* 2001;**8**:7–19.
- Bisaga A, Popik P, Beshpalov AY, et al. Therapeutic potential of NMDA receptor antagonists in the treatment of alcohol and substance use disorders. *Expert Opin Investig Drugs* 2000;**9**:2233–48.
- Hensel M, Kox WJ. Safety, efficacy, and long-term results of a modified regimen of rapid opiate detoxification under general anaesthesia: a prospective study in methadone, heroin, codeine and morphine addicts. *Acta Anaesthesiol Scand* 2000;**44**:326–33.
- Demaria PA Jr, Rodgers C, Braccia G. Propofol for sedation during rapid opiate detoxification. [Letter]. *Am J Psychiatry* 1997;**154**:290–1.
- Ritson B, Chick J. Comparison of two benzodiazepines in the treatment of alcohol withdrawal. *Drug Alcohol Depend* 1986;**18**:329–34.
- Carrasco G, Cabre L, Sobrepere G, et al. Synergistic sedation with propofol and midazolam in intensive care patients after coronary artery bypass grafting. *Crit Care Med* 1998;**26**:844–51.
- Carrasco G, Molina R, Costa J, et al. Propofol and midazolam in short-, medium-, and long-term sedation of critically ill patients. *Chest* 1993;**103**:557–64.
- Possidente CJ, Rogers FB, Osler TM, et al. Elevated pancreatic enzymes after extended propofol therapy. *Pharmacotherapy* 1998;**18**:653–5.
- Perrier ND, Baerga-Varela Y, Murray MJ. Death related to propofol use in an adult patient. *Crit Care Med* 2000;**28**:3071–4.
- Islander G, Vinge E. Severe neuroexcitatory symptoms after anaesthesia with focus on propofol anaesthesia. *Acta Anaesthesiol Scand* 2000;**44**:144–9.
- Currier DS, Bevacqua BK. Acute tachyphylaxis to propofol sedation during ethanol withdrawal. *J Clin Anesth* 1997;**9**:420–3.
- Hendelsman L, Cochrane KJ, Aronson MJ, et al. Two new rating scales for opiate withdrawal. *Am J Drug Alcohol Abuse* 1987;**38**:159–66.
- Sullivan JT, Sykora K, Schneiderman J, et al. Assessment of alcohol withdrawal: the revised clinical institute withdrawal assessment for alcohol scale (CIWA-Ar). *Br J Addiction* 1989;**84**:1353–7.
- Spies CD, Dubisz N, Neumann T, et al. Therapy of alcohol withdrawal syndrome in intensive care unit patients following trauma: results of a prospective, randomized trial. *Crit Care Med* 1996;**24**:414–22.
- Lohr RH. Treatment of alcohol withdrawal in hospitalized patients. *Mayo Clin Proc* 1995;**70**:777–82.
- Crippen DW. Strategies for managing delirium tremens in the ICU. *J Crit Illness* 1997;**12**:140–9.

Air gun injury

M J Shepherd

A 25 year old man felt a sudden pain in his chest while walking in the park. He observed two youths with an air rifle in the near vicinity. Two days later he presented to the accident and emergency department with continuing chest pains and two episodes of haemoptysis. Examination showed a 5 mm healing left chest wound in the 4th intercostals space. Respiratory and cardiovascular examinations were unremarkable.

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A chest radiograph (figs 1 and 2) showed a foreign body within the thoracic cavity but no pneumo/haemothorax.

After admission under the cardiothoracic surgeons a CT scan (fig 3) showed the pellet to be lodged between the aorta and a large pulmonary vein. He was treated conservatively and after three days of observation discharged home.



Figure 1 AP chest radiograph showing foreign body left mid-zone.



Figure 2 Lateral chest radiograph showing foreign body mid thoracic cavity overlying cardiac shadow.

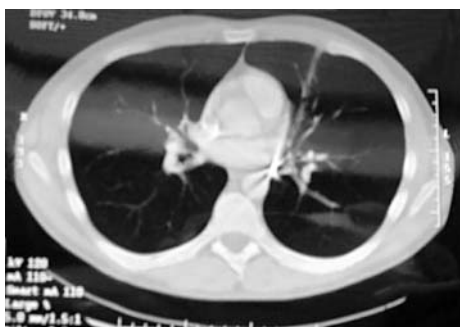


Figure 3 Computed tomogram of the thorax showing pellet lodged between aorta and large pulmonary vein.

This case further illustrates that perceived “low velocity” weapons such as air rifles potentially produce life threatening penetrating injuries in adults as well as children.¹

Patients may at presentation appear haemodynamically normal with minimal indication of underlying injury.²

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REFERENCES

- 1 Hudson AJ, Wyatt JP. Cardiac air gun pellet injury. *J Accid Emerg Med* 2001;**18**:519.
- 2 Wascher RA, Gwinn BC. Air rifle pellet injury to the heart with retrograde caval migration. *J Trauma* 1995;**38**:379–81.

Occult knee dislocation: the importance of secondary survey

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A 26 year old man was admitted after a single vehicle road traffic accident. He was an unrestrained driver travelling at approximately 50 mph and was ejected from his vehicle. His initial hospital management followed standard Advanced Trauma and Life Support guidelines. He had no loss of consciousness and complained only of severe left thigh pain. On examination, there was gross angular deformity at the left mid-thigh level, consistent with a femoral shaft fracture. Left pedal pulses were palpable and there was no neurological deficit. When fully exposed, the patient was noted to have bruising on the posterior aspect of his right knee. Despite normal colour, the right leg below the knee was cool to palpation. Distal motor and sensory assessment was normal although passive and active right knee movements were resisted. The right femoral pulse was

palpable. The right dorsalis pedis, posterior tibial, and popliteal pulses were absent.

Doppler ultrasound confirmed the presence of weak arterial signals distally. Plain radiographs identified no bony injury, joint dislocation or subluxation in the right lower limb. Percutaneous-transfemoral digital-subtraction angiography identified an intimal flap of the right popliteal artery (fig 1) and emergency exploration was performed. A large haematoma was noted over the posterior aspect of the right knee joint with rupture of the hamstring tendons, the posterior joint capsule, both collateral ligaments, and both cruciate ligaments. The involved arterial segment was isolated and excised, a segment of saphenous vein was interposed, and arterial perfusion was restored. The muscular compartments of the right leg were decompressed with

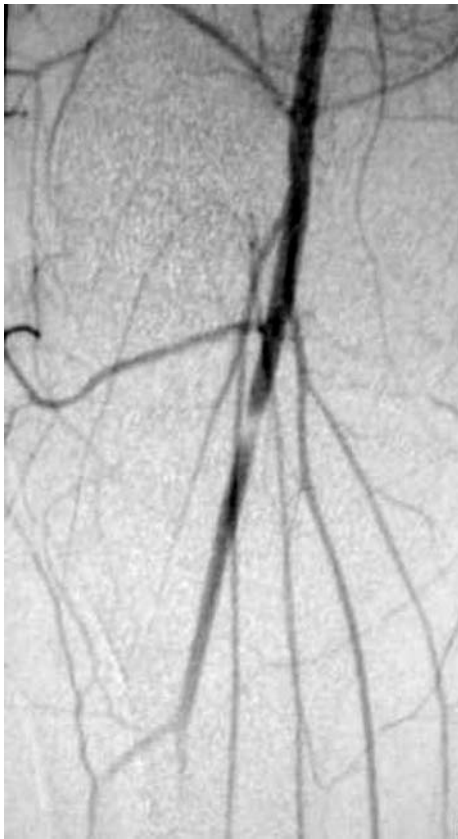


Figure 1 Digital subtraction angiogram of the right femoropopliteal vessels, showing intimal disruption of the right popliteal artery, characterised by segmental loss of the normal smooth luminal contour.

medial and lateral fasciotomies. The left femoral shaft fracture was managed with a statically locked retrograde intramedullary nail.

Delayed reconstruction of the right knee ligamentous injuries was subsequently undertaken.

Traumatic knee dislocation is uncommon, typically anterior and in up to 50% of cases the knee joint will be in a reduced position at presentation. Tethered proximally at the adductor hiatus and distally as it passes deep to soleus, the popliteal artery is injured in 30% of knee dislocations.¹⁻³ The presence of normal arterial pulses or Doppler signals, although reassuring does not exclude an arterial injury. Kaufman *et al* in their series showed a low but definite (13%) frequency of nonocclusive arterial injury (spasm or intimal flap) after traumatic dislocation of the knee.³ Progression to complete occlusion, although rare, has been reported.⁴ Prompt recognition of the presence of an arterial injury and the restoration of blood flow are paramount for limb salvage.

The type of injury described above is an occult dislocation and although it is not uncommon in the high velocity trauma patient, more overt injuries may distract the attending doctor. It is evident from the case described that a high index of suspicion and careful secondary survey are necessary to prevent the possible complications of this injury.

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REFERENCES

- 1 Chapman JA. Popliteal artery damage in closed injuries of the knee. *J Bone Joint Surg Br* 1985;**67**:420-3.
- 2 Walls RM, Rosen P. Traumatic dislocation of the knee. *J Emerg Med* 1984;**1**:527-31.
- 3 Kaufman SL, Martin LG. Arterial injuries associated with complete dislocation of the knee. *Radiology* 1992;**184**:153-5.
- 4 Gorman JF, Hauman RL, Rosenberg JC. Delayed thrombosis following arterial trauma. *J Trauma* 1966;**6**:681-4.

Hypoventilation and hypoxia in reversal of cardiogenic shock in an infant with congenital heart disease

A Sacchetti, G Wernovsky, C Paston, M Fernandes

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The case is presented of a child in cardiogenic shock in whom oxygen administration exacerbated a systemic to pulmonary shunt that caused a critical deterioration in his cardiovascular status requiring hypoventilation and restoration of baseline hypoxia for reversal.

A 5 week old boy presented to the emergency department with rapid breathing. History by the mother noted an atrial septal defect, ventricular septal defect, pulmonary atresia, and hypoplastic right heart. The child underwent a palliative central shunt four weeks earlier. On

physical examination the infant was pale, diaphoretic with heart rate 160 bpm, respiratory rate 76, no measurable blood pressure, and no obtainable pulse oximetry. Lungs were clear with a normal heart auscultation. Extremity examination showed pronounced pallor and mottled limbs with only femoral pulses palpable. Cardiac monitor showed sinus tachycardia.

The child was immediately placed on 18 l/min of oxygen via a facemask. An arterial blood gas measurement obtained on oxygen revealed a pH of 6.79, pCO₂ of 20 mm Hg, pO₂ of 102 mm Hg, and a calculated HCO₃⁻ of 3 meq/l. A right intraosseous tibial access was obtained and the child received 1 meq/kg of sodium bicarbonate. The child remained clinically in shock and was endotracheally intubated and

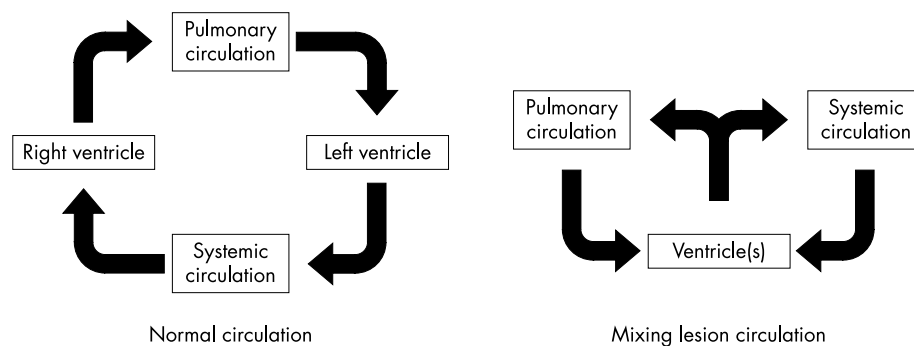


Figure 1 Comparison of a normal circulation and that of a child with an anatomical mixing lesion.

ventilated with 100% oxygen. A second arterial blood gas measurement after intubation showed pH of 6.79, $p\text{CO}_2$ of 26 mm Hg, $p\text{O}_2$ of 109 mm Hg, and a HCO_3^- of 4 meq/l. A 2 meq/kg bolus of bicarbonate was given. Bedside echocardiography showed a hypoplastic right heart and a patent central shunt between the aorta and pulmonary artery.

Consultation with the patient's cardiologist was obtained and withdrawal of supplemental oxygen and controlled hypoventilation with room air (FiO_2 21%) was recommended. About five minutes after starting this change in management, the child's clinical appearance of shock resolved with reversal of pallor and detection of a blood pressure of 91/70.

A second emergency department presentation one month later with profound metabolic acidosis, hyperventilation, and shock was again successfully treated with paralysis and hypoventilation.

DISCUSSION

Supplemental oxygen for the reversal of hypoxia is considered essential in the treatment of shock.¹

The pulmonary vascular bed is extremely reactive physiologically. Alveolar hypoxia, alveolar hypercapnia, acidosis, and hypoventilation all produce pulmonary vasoconstriction while hyperventilation, hyperoxia, alveolar hypocapnia, and alkalosis vasodilate the pulmonary circulation.²⁻⁴ Children with anatomical heart anomalies and communications to permit mixing of blood between the pulmonary and systemic circulations are typically chronically cyanotic with the potential for hypoxic pulmonary hypertension at baseline.

In children with normal cardiopulmonary anatomy the pulmonary and cardiovascular beds are structurally and physiologically separated. In children with "mixing lesions", the two vascular beds freely communicate with shared pumping chambers. Figure 1 shows this difference.

Blood flow between these two vascular beds may not always be equal. If a state occurs in which pulmonary vascular resistance increases or systemic vascular resistance decreases blood will preferentially shift to the peripheral circulation at the expense of blood flow to the lungs. This is the pathophysiology behind the hypercyanotic spell ("Tet spell") seen in children with tetralogy of Fallot and similar lesions. Increases in systemic vascular resistance or decreases in pulmonary vascular resistance will produce the opposite effect shunting blood to the pulmonary circulation at the expense of peripheral perfusion.

This may occur when a child's baseline state of hypoxic pulmonary hypertension is suddenly reversed with additional oxygen or assisted ventilations that decrease alveolar carbon dioxide concentrations.

In the patient presented, a univentricular heart distributed blood to both the pulmonary and systemic circulations. Dehydration and acidosis produced compensatory hyperventilation that coupled with the supplemental oxygen provided

on arrival in the emergency department reversed baseline pulmonary hypertension, lowered alveolar carbon dioxide tensions, and increased pulmonary blood flow at the expense of aortic flow. Institution of iatrogenic hypoxia and hypoventilation restored the pulmonary hypertension and re-established blood flow to the systemic circulation.

Supplemental oxygen remains the initial resuscitative manoeuvre in any child. However, in children with cardiac lesions, especially those with known mixing lesion careful attention must be directed to the child's clinical response to interventions. Pulse oximetry and arterial blood gas measurements may represent the single best indicator of haemodynamic stability in patients with mixing lesions. Low arterial oxygen saturations (75%–85%) with a normal pH indicate an acceptable balance of pulmonary blood flow with adequate peripheral perfusion. Increased oxygen saturations (>90%) with metabolic acidosis represent significantly increased pulmonary flow at the expense of decreased systemic flow. In children with mixing lesions in whom baseline information is not available an oxygenation saturation of 80%–85% is a realistic target, assuming normal oxygen consumption, haemoglobin, and cardiac output.^{5,6}

Intentional hypoxia and hypoventilation are counter intuitive treatments and recognition of the rare child who might require these treatments is extremely difficult. Use of some form of medical identification jewellery linked to an emergency information form for children with special health care needs should be encouraged in these patients and can help direct care until consultation with a specialist is obtained.⁷⁻⁹

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REFERENCES

- 1 **AAP-ACEP**. *Respiratory distress in APLS: the paediatric emergency medicine course*. 3rd ed. Dallas, TX: American College of Emergency Physicians, 1998:3–16.
- 2 **Tabbutt S**, Ramamoorthy C, Montenegro LM, *et al*. Impact of inspired gas mixtures on preoperative infants with hypoplastic left heart syndrome during controlled ventilation. *Circulation* 2001;**104**:1159–64.
- 3 **Ramamoorthy C**, Tabbutt S, Kurth CD, *et al*. Effects of inspired hypoxic and hypercapnic gas mixtures on cerebral oxygen saturation in neonates with univentricular heart defects. *Anesthesiology* 2002;**96**:283–8.
- 4 **Chang AC**, Zucker HA, Hickey PR, *et al*. Pulmonary vascular resistance in infants after cardiac surgery: role of carbon dioxide and hydrogen ion. *Crit Care Med* 1995;**23**:568–74.

- 5 **Wernovsky G**, Bove EL. Single ventricle lesions. In: Chang AC, Hanley FI, Wernovsky G, et al, eds. *Paediatric cardiac intensive care*. Baltimore: Williams and Wilkins, 1998:271–87.
- 6 **Wernovsky G**, McElhinney D, Tabbutt S. Postoperative management after first-stage palliation. In: Rychik J, Wernovsky G, eds. *Hypoplastic left heart syndrome*. Norwell, MA: Kluwer Academic, 2002:105–28.
- 7 **Sacchetti A**, Sacchetti C, Carraccio C, et al. The potential for errors in children with special health care needs. *Acad Emerg Med* 2000;7:1330–3.
- 8 **American College of Emergency Physicians**. Emergency information form for children with special health care needs. *Ann Emerg Med* 1999;34:577–82.
- 9 **American College of Emergency Physicians**. Emergency information form. (<http://www.acep.org/1,374,0.html>).

Variable radio-opacity of a metallic foreign body

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A 10 year old boy was seen in the accident and emergency department giving a history of swallowing the metal disc that makes up part of the ring pull from a steel soft drinks can. He could feel it stuck in his throat, at the level of the cricoid, and it was uncomfortable to swallow. He was otherwise completely fit and well. Examination was unremarkable.

A plain AP radiograph of the neck was obtained (fig 1), with clear views to the level of the third thoracic vertebra, which was normal, and the patient reviewed. In view of the persistent symptoms, and the definite history, a lateral radiograph of the neck was performed (fig 2). This showed a radio-opaque object at the level of the first thoracic vertebra.

A referral to the ENT surgeons was made, oesophagoscopy performed under general anaesthetic, and the foreign body removed. The child made a full, uneventful recovery.

DISCUSSION

There have been discussions in the medical literature previously regarding the most appropriate investigations for swallowed foreign bodies in children in the accident and emergency setting. Important factors to consider are that sharp or potentially caustic foreign bodies (such as watch batteries) should be removed, but that smooth, non-obstructing foreign bodies may be allowed to pass the length of the digestive tract without surgical intervention. It is important to remember that the narrowest parts of the gastrointestinal tract, and therefore the most likely points for obstruction to occur, are at the cricopharyngeal sphincter, the upper third of the oesophagus as it passes over the arch of the aorta and the gastro-oesophageal junction. Objects that have passed into the stomach are unlikely to impact elsewhere in the intestinal tract.

The textbooks in both ENT and accident and emergency medicine suggest that for a history of a potentially radio-opaque swallowed foreign body that has become lodged, radiographs should be obtained of the chest, abdomen, and neck (in that order) until the foreign body is seen.^{1,2} In a child this can be achieved in a single film. The orientation of discoid foreign bodies such as coins varies depending on their impaction in the oesophagus or trachea. In the oesophagus they will appear as a round disc on an AP film, and in the trachea they will appear side on as a bar.

The radio-opacity of various objects has been investigated previously, and it is known that ring pulls from aluminium drinks cans are radiolucent.¹ Most soft drinks cans these days seem to be made from steel. In the reported case, the steel disc was not seen on the AP view, but is seen on the lateral view. This prompted a radiograph to assess whether this was actually the case (fig 3). It is clearly shown that the steel disc is visible radiographically only when viewed from the side.



Figure 1 Plain AP radiograph of the neck.

This is because of the increased thickness of metal when viewed from the side.

CONCLUSIONS

How should a suspected foreign body in the oesophagus be investigated? There seems to be a case for obtaining both AP and lateral radiographs. This would be beneficial not only for the reasons of variable radio-opacity dependent upon orientation as listed above, but also to give a better idea of anatomical location. The disadvantage of this however would be the increased radiation exposure.

There have been unusual cases reported previously where multiple radiographs have been suggested. In particular, a case reported in 1993 in which a single coin was seen in the oesophagus on the AP radiograph, which was removed endoscopically.³ A second look in the oesophagus to check for abrasion revealed a second, smaller coin that had been located either anteriorly or posteriorly to the fist and therefore not seen on AP radiography. They would have been seen as two separate coins on lateral radiography. This case, and the suggestions made by the authors to perform multiple radiographic investigations in all cases, sparked a series of letters on the subject covering the above points. Nine years later there still seems to be no consensus opinion.



Figure 2 Lateral radiograph of the neck.

Another method of investigation to consider is the use of portable metal detectors to localise the position of metallic foreign bodies. A randomised controlled study from Canada published in 2000 reported a 100% accuracy in the diagnosis of metallic foreign bodies that had been accidentally ingested.⁴ The difficulty with this method of investigation is that in the author's experience there are only a minority of departments that have access to a portable metal detector.

Clinically it can be very difficult for a patient to locate precisely where a foreign body lies in the oesophagus. All cases should therefore be treated with caution and investigations tailored to suit the particular case and the department in which the case presents.

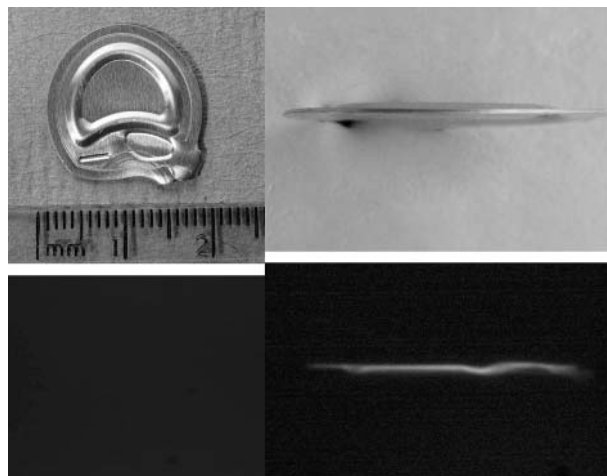


Figure 3 Photographs and radiographs of a steel disc from a drinks can in different positions.

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REFERENCES

- 1 **Birchall MA**, Croft CB. Examination and endoscopy of the upper aerodigestive tract. In: *Scott-Brown's otolaryngology*. 6th ed. Vol 5. Oxford: Butterworth Heinemann, 1997:10–11.
- 2 **Moulton C**, Yates D. Ophthalmic, ENT and facial conditions. In: *Emergency medicine*. 2nd ed. Oxford: Blackwell Science, 1999:329–9.
- 3 **Anonymous**. Minerva pictures. *BMJ* 1993;**307**:273.
- 4 **Gooden EA**, Forte V, Papsin B. Use of a commercially available metal detector for the localization of metallic foreign body ingestion in children. *J Otolaryngol* 2000;**29**:218–20.

Occult congenital anomaly of the atlas presenting in the setting of acute trauma

A W O'Sullivan, F Mc Manus

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Congenital absence of the posterior arch of the atlas is rare. Its detection in the setting of acute trauma may be confusing and mimic a fracture. This case serves to highlight the adjunctive use of computed tomography in equivocal cases. Most patients can be managed conservatively, however operative treatment should be reserved for patients in whom atlanto-axial instability has been shown.

A 26 year old woman was referred to our service complaining of neck pain after being involved in a low velocity road traffic accident in which she was

struck from behind. On examination she had no neurological signs and standard cervical spine radiographs showed features suggestive of a fracture of the posterior arch of C-1 (fig 1). However, computed tomography identified this as a congenital defect in which the posterior arches were absent and the anterior arches had failed to fuse in the midline (fig 2). Magnetic resonance imaging confirmed the above findings, showing fibrous bands in the place of the posterior arch. It also excluded any soft tissue injury, haematoma, or disc prolapse. Lateral flexion/extension plain radiographs of the cervical spine outruled atlanto-axial instability. The patient was treated conservatively and her neck pain resolved spontaneously within 48 hours. She remains well at follow up.



Figure 1 Lateral cervical spine radiograph showing absence of the posterior arches of the atlas.

DISCUSSION

Congenital defects of the ring of the atlas may be asymptomatic or may present with neurological symptoms secondary to atlanto-axial instability and resultant cord compromise.¹ They may also be detected on plain radiographs after trauma as in this case. This case serves to emphasise that not all plain film cervical abnormalities, even in the setting of acute trauma, are traumatic in origin and the important adjunctive role of computed tomography in accurately delineating equivocal cases.² It also emphasises that treatment of these anomalies should be dependent on the presence or absence of atlanto-axial instability, with or without neurological symptoms.³ Most patients can be managed conservatively, however a small number of patients with instability will require operative stabilisation. Instability can be shown on lateral flexion/extension plain radiographs or magnetic resonance



Figure 2 Computed tomography of the atlas showing the absent posterior arches and failure of the anterior arches to fuse in the midline.

imaging of the cervical spine. The second method has the advantage of assessing any signal change of the cord during the manoeuvre and should be the investigation of choice.

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REFERENCES

- 1 Currarine G, Rollin SW, Diehl JT. Congenital defects of the posterior arch of the atlas. *Am J Neuroradiol* 1994;**15**:249-54.
- 2 Bauchulis BL, Long WB, Hynes GD, et al. Clinical indications for cervical spine radiographs in the traumatized patient. *Am J Surg* 1987;**153**:473-8.
- 3 Clarke CR. Congenital malformations. In: *The cervical spine*. 3rd edn. Philadelphia: Lippincott-Raven, 1998:336-7.

Hypovolaemic shock by rat bites. A paradigmatic case of social deprivation

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This report describes an unusual complication of a comparatively common problem of rat bites, causing a near fatal case of hypovolaemic shock. An 8 month old girl was bitten on her head and hands by Norway rats (*Rattus norvegicus*) while sleeping after falling from her parent's bed. She could not be attended to because of her parents' alcoholic condition. She was found in hypovolaemic shock. She required ventilatory and haemodynamic support for five days. At late follow up, the child had no medical problem. In this case, evident social risk factors include extreme poverty, poor hygiene, and rat infested environment, which are frequently associated in potential rat bite victims.

There is scarce information in the medical literature about death or significant complications attributable to house rat bites in children. We present an extraordinary case of hypovolaemic shock in a child, which illustrates a near fatal illness in some conditions of severe social deprivation.

CASE REPORT

An 8 month old girl was left in bed the night before the admission to hospital while sleeping together with her parents who were drunk. She fell down during the night. Her aunt visited them the morning after and she noticed that the baby was lying under the parent's bed severely ill. Her parents did not give any warning of the situation or that the baby was crying. Upon reporting the case to the police she was brought to the emergency room. The parents had no police records and the family had not been evaluated by social services.

On physical examination, the patient had extensive frontal wounds, on the right (1×2.5×0.5 cm) and left sides (7.5×2.5×0.5 cm) of the forehead. Additionally, multiple punctiform wounds were found on the face, nose, mouth, scalp, and both hands (fig 1). The patient was drowsy, cold, pale, dehydrated, with decreased capillary refill time and heart rate of 180/min. Arterial blood pressure was 91/54 mm Hg. She weighed 8 kg. There was no clinical evidence of old injuries on the physical examination. The skeletal survey and brain CT scan showed no abnormality. Significant blood loss



Figure 1 Multiple wounds on right hand caused by rat bites (*Rattus norvegicus*).

from her wounds was evident. Normal saline (20 ml/kg) was given by intraosseous infusion; blood gas measurements showed metabolic acidosis (pH 7.1).

The patient was transferred to the paediatric intensive care unit. Considering the history given by the relatives and the type of the wounds, she was diagnosed in hypovolaemic shock caused by rat bites and she was intubated and mechanically ventilated. Pulse, perfusion, and arterial pressure improved significantly with red cells transfusion, fresh frozen plasma, normal saline, and albumine infusions (total volume 70 ml/kg). Antimicrobial treatment with penicillin, clindamycin, and cefotaxime was started intravenously.

The laboratory tests showed a blood cell count with leucocytosis of $81 \times 10^3/\mu\text{l}$; packed cell volume 20%; haemoglobin 70.0 g/l; platelet count $416 \times 10^3/\mu\text{l}$. Lactacidaemia initially was 0.74 mmol/l (normal value <0.22 mmol/l), blood sugar 17.2 mmol/l and prothrombin time 26 seconds.

During the first day of the clinical course, haemodynamic stability was obtained. Rabies prophylaxis was given (intramuscular Verorab vaccine). Tetanus immunisation was not given. The surgical treatment was performed 36 hours after admission. Lacerations with associated soft tissue devitalisation were debrided. Blood cultures were negative. The remainder of the course was uneventful. She was finally discharged from the paediatric intensive care unit at the seventh day.

The medical history disclosed that the family lived in an urban slum in a single room house with dust on the floor, close to sewer ducts where the Norway rats (*Rattus norvegicus*) freely emerged onto the surface level.

The patient stayed under legal protection, for neglect and mistreatment.

DISCUSSION

This patient represents a rare and dramatic case in which social deprivation, poverty, and alcoholic addiction in a rat infested environment led to a severe and potentially fatal disease of a child.

Domestic dogs and cats cause most of the animal bites in children. It is very unusual that rat bites can originate multiple and extensive wounds that cause hypovolaemic shock.

Previously, a similar case was reported by Yanai *et al.* In that case a 3 month old girl died because of blood loss resulting from multiple rat bites.¹ Usually the wounds caused by rat bites in children tend to occur after death.

Rat bites primarily affect children under 5 years old. Most bites are inflicted on the face and hands and usually occur at night while sleeping.²

Norway rat (*Rattus norvegicus*) is also known as the brown rat, house rat, wharf rat, or sewer rat. Its colour is dark brown to black and the total length is 30–45 centimetres. The tail is shorter than the length of head and body. Adult Norway rats weigh 400–500 g. These rats mature in 2–5 months, and live as an adult for 6–12 months. The pregnancy lasts three weeks. The average female rat has four to six litters a year, with 6 to 12 young. Rats have poor eyesight, relying more on their hearing and their excellent senses of smell, taste, and touch.

Norway rats are primarily nocturnal and live in close association with people as they depend chiefly on humans for food, then once established in a house they tend to remain, preferentially in warehouse, chicken houses, garbage dumps, and sewer. These rodents eat nearly any type of food and prefer cereal grains, meat and fish, nuts, and pet food.

Rats are an example of urban pest that transmit disease by several mechanisms, directly by contaminating food with their urine or faeces. Sometimes they transmit disease indirectly, as when fleas bite a disease infected rat, then a person or by biting people.

Frequently, rat bites cause local bacterial infection, which have good prognosis. Antibiotics such as cephalosporins or penicillinase resistant penicillins are usually adequate for treatment of infectious complications of rat bites.

Rats also have been associated with rat bite fever (by *Streptobacillus moniliformes* or *Spirillum minus*),³ Haverhill fever,⁴ tetanus,⁴ multiple skin abscesses,⁵ and rabies.⁶

In summary, this case shows a situation in which extreme poverty, poor hygiene, and rat infested environment are conditions frequently associated with the occurrence of potentially serious illness in children secondary to rat bites.

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REFERENCES

- 1 Yanai O, Goldin L, Hiss J. Fatal rat bites. *Harefuah* 1999;136:611–13.
- 2 Hirschhorn RB, Hodge RR. Identification of risk factors in rat bite incidents involving humans. *Pediatrics* 1999;104:e35.
- 3 Hudsmith L, Weston V, Szram J, *et al.* Clinical picture, Rat bite fever. *Lancet Infect Dis* 2001;1:91.
- 4 Mathiasen T, Rix M. Rat-bite-an infant bitten by a rat. *Ugeskr Laeger* 1993;155:1475–6.
- 5 Brooks JE. A review of commensal rodents and their control. *Rev Environ Cont* 1973;3:405–53.
- 6 Marshall J, Sharp E, Barrison IG. Once bitten, twice shy. *BMJ* 1994;309:1694–5.

Clinical use of the Empey index in the emergency department

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Percutaneous tracheostomies (PCT) performed in the intensive care unit (ICU) are becoming a routine procedure in the care of the patient who is likely to require ventilatory support for seven days or more. As the frequency of this procedure increases so will the numbers of complications, such as tracheal stenosis. We discuss the diagnosis of tracheal stenosis in the emergency setting and the use of the Empey index.

CASE HISTORY

A 26 year old woman presented to the emergency department (ED) complaining of shortness of breath and difficulty clearing secretions. She had been discharged from hospital two weeks previously after a four week stay, including 20 days in the ICU. On that occasion she presented with increasing shortness of breath and stridor attributable to bacterial tracheitis. While in the ICU she developed pneumonia and severe sepsis. On day four of her ICU stay she underwent PCT in the ICU with a Blue Rhino tracheostomy set, which remained in situ for 16 days. Before her ICU stay she had been fit and well.

On presentation to the ED she was anxious but looked well with Sao_2 100% on oxygen, respiratory rate 20, blood pressure 110/70, pulse 98. Examination of her chest showed a prolonged inspiratory phase and transmitted upper airways noises, presumed to be from secretions but no stridor. Besides a well healed tracheostomy scar and appearing to be undernourished the rest of the examination was normal.

A chest radiograph and lateral soft tissue neck were normal, as were the arterial blood gas pressures. In view of the recent ICU admission bedside spirometry (Micro Medical) was performed and showed a forced expiratory volume in one second (FEV_1) of 0.87 litres and a peak expiratory flow rate (PEFR) of 70 litres per minute; giving an Empey index of 12.4. The patient underwent endoscopic examination of the airway (above the cords) while in the ED and this was normal. On day 2 of the patient's admission she underwent computed tomography, which showed significant tracheal stenosis (see fig 1). She subsequently underwent endoscopic stenting and made a good postoperative recovery.

DISCUSSION

The Empey index^{1,2} is a way of predicting if a patient has upper airways obstruction and can be performed simply at the bedside. It is the calculation of the ratio of FEV_1 (ml):PEFR (l/min); a normal person will have a ratio of less than 10 while a person with upper airways obstruction will have a ratio greater than 10, the higher the index the more severe the obstruction. The index has been well validated on a variety of patients with upper airways obstruction but also in patients with other forms of lung disease including asthma and emphysema.

At near total lung volumes flow (expiration) is dependent on effort and the pressure within the alveoli; the addition of an upper airways obstruction increases the resistance in the

Learning points

The diagnosis of tracheal stenosis should be suspected in any patient presenting with respiratory symptoms after an intensive care unit (ICU) stay. The Empey index is a reliable bedside test that can aid the diagnosis of tracheal stenosis in the ED.



Figure 1 CT scan showing tracheal stenosis (arrow) with a minimum diameter of 0.5 cm by 0.8 cm at the level of the 3rd/4th tracheal ring.

upper respiratory tract and reduces the flow at high lung volumes. At the lower lung volumes the collapse of bronchioles during expiration is much more of a determining factor in flow generation. From figure 2 it can be seen that the FEV_1 is very similar in both subjects—that is, FEV_1 is little affected by the presence of upper airways obstruction. FEV_1 is measured over one second—that is, over a range of diminishing lung volumes, it is less dependent on effort and upper airways resistance. PEFR is measured in the first two milliseconds of a maximal expiration—that is, at high lung volumes. Hence in upper airways obstruction PEFR is much more affected than the FEV_1 . In asthma and chronic obstructive pulmonary disease both FEV_1 and PEFR are affected, and in emphysema the peak flow is relatively less reduced compared with the FEV_1 .

Diagnosis of tracheal stenosis may also be aided by chest radiography and lateral soft tissue neck, which may show alteration of the air column. Magnetic resonance imaging and computed tomography are also helpful.^{3,4} Flow volume

Abbreviations: PCT, percutaneous tracheostomy; ICU, intensive care unit; ED, emergency department

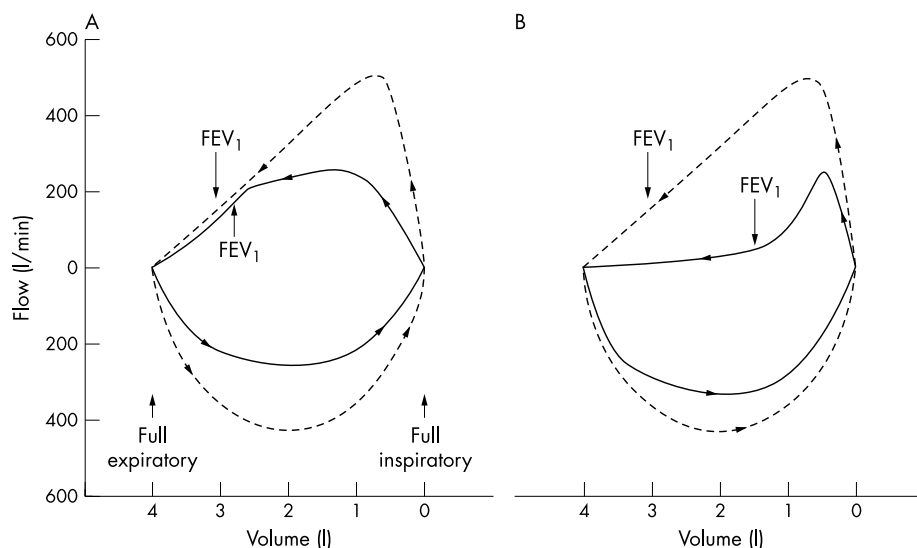


Figure 2 Maximal expiratory and inspiratory flow volume curves (continuous lines) for (A) a patient with upper airways obstruction, and (B) a patient with asthma. The interrupted lines show the flow volume curve for a normal subject. (Reproduced with permission BMJ Publishing Group (see reference 1).

loops may also be helpful in long term follow up and monitoring for re-stenosis.

It is important to consider tracheal stenosis in any patient who has undergone a prolonged stay in ICU. Tracheal trauma is the most common cause of stenosis in children and adults. About 90% of all acquired chronic subglottic stenosis results from endotracheal intubation, reported rates vary from 0.9% to 8.3%.⁵ Intubation causes injury at multiple levels; pressure on the arytenoid cartilage, pressure or motion of the tip of the tube against the subglottic cartilage causing ischaemia and necrosis. Factors important in the development of stenosis include length of time of intubation⁶ and size of tube. Clearly in a patient who already has an inflamed trachea and is septic then these additional risk factors are likely to predispose to subglottic stenosis.

Tracheostomy allows for easier weaning of ventilated patients, requiring less sedation and reducing both dead space and airway resistance. In addition it has many advantages for the patient including facilitating oral communication and speech, permitting more mobility and comfort. Nursing care is also facilitated with improved oral, nasal, and facial hygiene, easing airway care and suctioning.

PCT is thought to reduce the incidence of laryngeal and subglottic stenosis, the rate of which has been quoted as 1.9%⁷ but the incidence varies greatly between series and is dependent on the technique used. One long term follow up of 340 critical care survivors of PCT found that 31% of patients had a tracheal stenosis of more than 10% and symptomatic stenosis present in 6% of patients.⁴ Most stenoses are asymptomatic and can involve between 10%–40% of the diameter, above 75% symptoms are usual. Most cases are diagnosed within one year and most present within 2–12 weeks.⁸ Presenting features include dyspnoea, stridor, hoarseness, cough, and recurrent pneumonitis.

Factors that minimise the risk of stenosis include using properly sized tracheostomy tubes with high volume low pressure cuffs, flexible ventilator connectors, and placing the tube between 1st and 2nd or 2nd and 3rd tracheal rings (to

lower risk of cricoid injury). Operative correction is only rarely required and may include procedures such as tracheopexy with muscle-fascia repair and resection of stenotic segment with end to end repair.

CONCLUSION

The diagnosis of tracheal stenosis should be suspected in any patient presenting with respiratory symptoms after an ICU stay. The absence of stridor, as in our case, does not exclude the presence of significant tracheal stenosis.

The Empey index is a reliable bedside test that can aid the diagnosis of tracheal stenosis in the ED.

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REFERENCES

- 1 Empey DW. Assessment of upper airways obstruction. *BMJ* 1972;3:503–5.
- 2 Topham JH, Empey DW. Practical assessment of obstruction in the larynx and trachea. *J Laryngol Otol* 1974;88:1185–93.
- 3 Callanan V, Gillmore K, Field S, et al. The use of magnetic resonance imaging to assess tracheal stenosis following percutaneous dilatational tracheostomy. *J Laryngol Otol* 1997;111:953–7.
- 4 Norwood S, Vanilla VL, Short K, et al. Incidence of tracheal stenosis and other complications after percutaneous tracheostomy. *Ann Surg* 2000;232:233–41.
- 5 Garnett JD. Subglottic stenosis in adults. <http://emedicine.com> (accessed Jun 2002).
- 6 Whited RE. A prospective study of laryngotracheal sequelae in long-term intubation. *Laryngoscope* 1984;94:367–77.
- 7 Bliznikas D, Buredes S. Percutaneous tracheostomy. <http://emedicine.com> (accessed Feb 2003).
- 8 Weber AL, Grillo HL. Tracheal stenosis: an analysis of 151 cases. *Radiol Clin North Am* 1978;16:291–308.

Gastropericardial fistula: a case report and review of literature

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Pneumopericardium is a rare occurrence. A variety of oesophageal and gastric lesions have been reported to perforate into the pericardium causing pneumopericardium. Although peptic ulceration in hiatus hernia has also been reported to perforate into pericardium, other types of diaphragmatic hernia have not been described as causing perforation into the pericardium. We report a case of a young man who presented with chest pain and was found to have pneumopericardium secondary to a gastropericardial fistula in a recurrent diaphragmatic hernia.

CASE REPORT

A 29 year old man presented in March 2002 to the accident and emergency department with two hour history of severe chest and abdominal pain and dyspnoea. He was in severe pain with a pulse rate of 112/min, blood pressure 169/74 mm Hg, and respiratory rate of 26/min. Resuscitation was begun with oxygen, intravenous fluids, and analgesics. On examination he had decreased air entry at left chest base, tenderness, and guarding in upper abdomen and an upper midline scar. Chest radiography showed pneumopericardium (fig 1). Electrocardiogram showed sinus tachycardia and 1 mm ST segment elevation in V2 and V3. Blood investigations showed a white cell count of $29.1 \times 10^9/l$. Blood gas analysis showed respiratory acidosis with pH of 7.26 and $Paco_2$ of 7.11 kPa. He was in excruciating pain with persistent tachycardia and tachypnoea and had to receive a total of 55 mg of intravenous morphine in titrated doses. Repeat examination showed distended neck veins. Repeat chest radiography showed increase in the size of pneumopericardium suggesting cardiac tamponade. Pericardiocentesis was done by subxiphoid approach under local anaesthesia with 1% lignocaine (lidocaine) and 60 ml of air was aspirated. This resulted in immediate clinical improvement and the pulse and blood pressure gradually returned to normal. A contrast meal using Gastromiro showed the presence of diaphragmatic hernia with perforation of stomach into pericardial sac (fig 2). Thus a diagnosis of gastropericardial fistula secondary to perforation of the stomach in diaphragmatic hernia was made.

In December 1997 he sustained a stab injury and developed a left pneumothorax, which was treated with chest drain uneventfully. In January 2000 laparotomy for peritonitis revealed a diaphragmatic hernia containing stomach, which was repaired. Over the next year he presented 10 times to the A&E department with episodes of a recurrent pain in his left shoulder. Examination and investigations for local and referred pain at the left shoulder included radiography of left shoulder, CT arthrogram of left shoulder joint, and ultrasound scan of the abdomen all of which were normal.

On this admission through a combination of laparotomy and left anterolateral thoracotomy the fistula was disconnected, and the defects in the stomach and diaphragm were repaired. Splenectomy was needed to gain access to the diaphragmatic defect. At follow up at one year he had no



Figure 1 Chest radiograph showing pneumopericardium.

recurrence of his symptoms, in particular his left shoulder pain resolved completely.

DISCUSSION

Pneumopericardium is a rare but well recognised entity. Britcheteau first described it in 1844.¹ The causes are of two groups: traumatic and non-traumatic, most being traumatic. Injuries account for most cases in the traumatic group. Less common causes in this group include thoracic procedures, endoscopy, and positive pressure ventilation. Predominant causes in the non-traumatic group include acute asthma and the oesophageal lesions namely peptic ulceration, carcinoma, and spontaneous rupture. Less common causes in this group include intrapericardial perforation of lung abscess or tuberculosis cavity and pericarditis attributable to gas forming organisms. Pneumopericardium has been reported to occur from sub-diaphragmatic lesions namely anaerobic and pyogenic liver abscesses that have penetrated through the diaphragm. Gastropericardial fistula resulting from peptic ulcer and carcinoma is the cause of pneumopericardium in less than 15 cases. The stomach in these cases was usually intrathoracic, through the hiatus as a hernia or after oesophagogastrectomy.²⁻⁵ Pneumopericardium can also occur after trans-diaphragmatic perforation of gastric carcinoma or



Figure 2 Contrast meal showing gastropericardial fistula.

ulcer into the pericardium,^{6–8} particularly in the presence of Zollinger-Ellison syndrome.^{2,8}

Pain in the left shoulder was thought to be a symptom of pericardial irritation in few previous case reports.⁴ Our patient presented to A&E department on numerous occasions with the same symptom. This was attributed to some local cause in the left shoulder, but probably represented diaphragmatic irritation. The diagnostic criteria of hydropneumopericardium as described by Shackelford in 1931^{1,2} are (a) a high pitched tympanitic percussion note, (b) a loud metallic splashing sound synchronously with the heart sounds, and (c) a characteristic chest radiograph with an air fluid level in the pericardial cavity.

Most cases of pneumopericardium have been diagnosed by chest radiography. Contrast radiography showed if a hiatal or other type of diaphragmatic hernia was present, and whether an oesophageal or gastric ulcer was present. The precise cause can be found in 70% of cases.⁹ Computed tomography may be helpful if the cause is not evident on contrast radiography. Oesophago-gastro-duodenoscopy (OGD), although used in a few cases,^{6,7,9,10} should be used with caution, as insufflation of air exacerbates cardiac tamponade.³ ECG may show changes of atrial fibrillation or pericarditis,^{9,10} but will be normal in most patients.

Only two cases have been reported to survive after conservative treatment with pericardiocentesis and antibiotics.^{6,9} The emergency management of pneumopericardium is twofold—firstly, early recognition of its presence by plain chest radiography and secondly, detection of signs of cardiac tamponade. Pericardial decompression is a lifesaving

emergency measure in the presence of cardiac tamponade. Delay in intervention in these cases can be life threatening. Surgery is the definitive treatment of pneumopericardium irrespective of the cause and location of the fistula.^{4,5,7} Successful outcome of gastropericardial fistula depends on both the emergency and definitive managements. Emergency management includes early recognition of pneumopericardium and pericardial decompression. Definitive management is resection of fistula and repair of diaphragmatic hernia. Adequate exposure and effective repair of the diaphragmatic defect may require splenectomy.⁸

In our case the initial injury to the diaphragm probably occurred at the time of stab injury. This defect in the diaphragm was asymptomatic until it declared itself as diaphragmatic hernia two years later. We have been unable to find a similar case in literature. Our patient underwent pericardial decompression within an hour and surgery within three hours of presentation to the hospital. Our case emphasises the importance of lifesaving measures in the emergency management of pneumopericardium namely, early recognition of pneumopericardium and emergency pericardial decompression in suspected cases of tamponade.

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REFERENCES

- 1 Shackelford RT. Hydropneumopericardium. *JAMA* 1931;**96**:187–91.
- 2 Edwards JRM, Humeniuk V. Gastropericardial fistula. *Aust N Z J Surg* 1996;**66**:257–9.
- 3 Monro JL, Nicholls RJ, Hatley W, et al. Gastropericardial fistula—a complication of hiatus hernia. *Br J Surg* 1974;**61**:445–7.
- 4 Salling N, Falensteen AM, Larsen LG. Non-traumatic perforation of gastric ulcer in a hiatal hernia to the pericardium. *Acta Med Scand* 1983;**213**:225–6.
- 5 Simice P, Zwirewich CV. Gastropericardial fistula complicating benign gastric ulcer: case report. *Can Assoc Radiol J* 2000;**51**:244–7.
- 6 Chapman PR, Boals JR. Pneumopericardium caused by giant gastric ulcer. *AJR Am J Roentgenol* 1998;**171**:1669–70.
- 7 Ghahremani GG, Yaghami I, Brooks JW, et al. Pneumopericardium due to trans-diaphragmatic perforation of a gastric ulcer. *Dig Dis* 1976;**21**:586–91.
- 8 Liu DH, Crastopol P, Phillips W. Perforation of a gastrojejunal ulcer into the pericardium. *Arch Surg* 1967;**94**:294–8.
- 9 Hall MN, Little JM Jr. Penetration of the pericardium by a gastric ulcer—survival after pericardiocentesis. *J Am Board Fam Pract* 1990;**3**:289–91.
- 10 Cozart JC, Sundaresan S, Chokshi HR, et al. Gastropericardial fistula with pneumopericardium: an unusual complication of benign peptic ulceration. *Gastrointest Endosc* 1999;**49**:387–90.