Short answer question case series: ventilatory considerations in a patient with morbid headache

A middle-aged man with a history of smoking and hypertension complains of acute onset severe headache and neck pain last night following the use of intranasal cocaine. On examination, HR 99 blood pressure 132/90 respiratory rate 22 SpO2 95% room air. He is alert but mildly agitated. His cranial nerve, motor-sensory, and funduscopic exams are normal. The remainder of his physical exam is unremarkable, except for mild meningismus. An emergent CT demonstrates an acute subarachnoid haemorrhage (SAH, figure 1). The patient develops increased work of breathing with a pulse oximetry reading of 92% on ambient air. A stat chest X-ray (figure 2) is obtained:

1. What does the X-ray show (figure 2)?
2. Given the rapid deterioration and X-ray, the patient is intubated, how can his ventilation be optimised?
3. Describe the Hunt–Hess Classification for SAH.
4. How does the World Federation of Neurological Surgeons (WFNS) scale differ?

The X-ray demonstrates pulmonary oedema, which is most often due to cardiogenic causes but may also be due to infections, salicylate toxicity, opiate use and neurogenic causes. In this case, salicylate and opiate testing were negative and a bedside ultrasound did not demonstrate any cardiac dysfunction, making neurogenic pulmonary oedema (NPE) most likely.

NPE is a clinical diagnosis made by the presence of radiographic evidence of alveolar fluid accumulation without haemodynamic evidence to suggest a cardiogenic aetiology (if measured, pulmonary artery wedge pressure ≤18 mm Hg). NPE has been associated with status epilepticus, head injury or cerebral haemorrhage, but its exact pathophysiology is poorly understood. It may be that a sympathetic surge from the medulla oblongata causes a neuromediated pulmonary venoconstriction, which increases the pulmonary capillary hydrostatic pressure and results in pulmonary oedema.

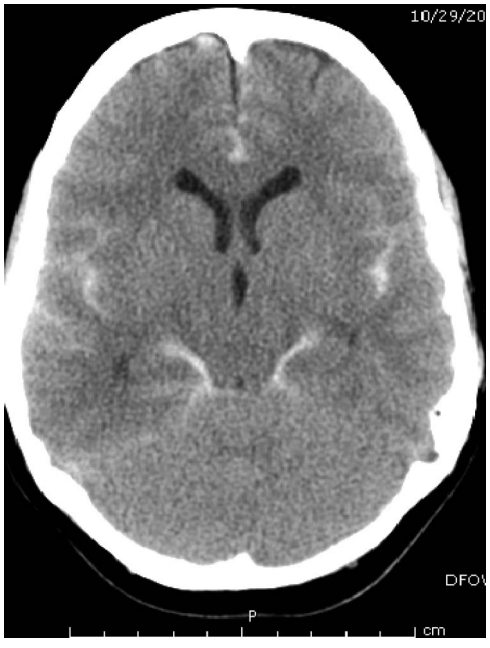

Figure 1 Head CT image.

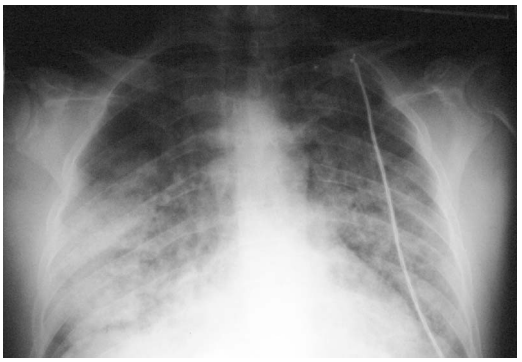

Figure 2 Chest radiograph.

It is important to distinguish non-cardiogenic from cardiogenic causes of pulmonary oedema. Diuresis would be indicated for cardiogenic volume overload, but may have untoward haemodynamic consequences in the setting of NPE. Although the presentation of acute respiratory distress syndrome (ARDS) may mimic NPE, ARDS protocols call for low tidal volumes and increasing positive end-expiratory pressure rather than simply increasing the FiO_2. However, increasing positive end-expiratory pressure in NPE should be used with extreme caution, as it has been linked to decreased cerebral perfusion pressure and worse outcomes (due to increased intrathoracic pressure, decreased venous return and systemic hypoperfusion).

Peak pressures may be elevated in lung parenchymal disease, especially if the plateau pressure is also elevated (as opposed to elevated peak pressures, but normal plateau pressure in cases of airway obstruction). While slowing the respiratory rate may help in some of those cases, it may inadvertently allow for hypercapnia, which would cause cerebral vasodilation and worsen intracranial pressure, possibly aggravating the vicious cycle of NPE.

Prone positioning, however, improves oxygenation of dependent areas. Also called the *lung recruitment manoeuvre*, prone positioning in mechanically ventilated patients optimises functional residual capacity, redistributes blood flow (minimizes V-Q mismatch), aids in diaphragm excursion and improves secretion removal. It is a natural way to improve oxygenation and ventilation without untoward physiologic consequences (as mentioned above). In a recent multicentre, prospective, randomised, controlled trial[3] investigators found that in patients with severe ARDS, early application of prone positioning was associated with significantly decreased mortality. It is simple to do and could help in cases of NPE.

SAHs may or may not be associated with an aneurysm. Cocaine use can cause sudden, large increases in blood pressure causing a rent in the arterial vasculature.

Various scales are used to grade and prognosticate SAH, the most common of which are the Hunt–Hess scale (table 1) and the WFNS scale (table 2). The Hunt–Hess scale is popular among emergency physicians for its ease of use and widespread acceptance:

Table 1 Hunt-Hess Subarachnoid Hemorrhage Scale

Grade	Neurologic status
1	Asymptomatic or mild headache and slight nuchal rigidity
2	Severe headache, stiff neck, no neurologic deficit except cranial nerve palsy
3	Drowsy or confused, mild focal neurologic deficit
4	Stupor, moderate or severe hemiparesis
5	Coma, decerebrate posturing

Hunt–Hess grades 1 and 2 have good survival rates, but grade 3 and above have >50% mortality.

The WFNS scale offers the added advantage of incorporating Glasgow coma scale; however, it is more complicated and may be more difficult to remember:

Table 2 World Federation of Neurological Surgeons Subarachnoid Hemorrhage Scale

Grade	Glasgow coma scale score	Motor deficit
1	15	Absent
2	13–14	Absent
3	13–14	Present
4	7–12	Present or absent
5	3–6	Present or absent

Some studies demonstrate that WFNS mortality steadily increases from grade 1 to grade 5, while others challenge its prognostic ability.

Timothy Horeczko, Timothy Jang

Department of Emergency Medicine, Harbor-UCLA Medical Center, Torrance, California, USA

Correspondence to Dr Timothy Horeczko, Harbor-UCLA Medical Center, Department of Emergency Medicine, 1000 W. Carson St., Torrance, CA 90509, USA; tbj@ucla.edu

Contributors All authors contributed to the write-up of this case.

Competing interests None.

Provenance and peer review Commissioned; internally peer reviewed.

To cite Horeczko T, Jang T. *Emerg Med J* 2014;**31**:423–424.

Published Online First 24 February 2014

Emerg Med J 2014;**31**:423–424. doi:10.1136/emermed-2013-203564

FURTHER READING

1 Coplin WM. *Lecture: Critical care of the patient with acute subarachnoid hemorrhage*. Neurotrauma & Critical Care, Wayne State University. https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=4&cad=rja&ved=0CEEQFjAD&url=http%3A%2F%2Fwww.philadelphia.edu.jo%2Facademics%2Fabatiha%2Fuploads%2FCritical%2520care%2520of%2520the%2520patient%2520with%2520acute%2520subarachnoid%2520hemorrhage.ppt&ei=AO7eUqD6GMnjoATL3oGgBA&usg=AFQjCNFrdkQU3wKZQTQXPqii22gE6wyONg&sig2=1KKceCUnV76LaSrJp2vfpg&bvm=bv.59568121,d.cGU (accessed 1 Jan 14).

2 Elmer J, Hou P, Wilcox S, *et al*. Acute respiratory distress syndrome after spontaneous intracerebral hemorrhage. *Crit Care Med* 2013;41:1992–2001.

3 Guérin C, Reignier JM, Richard JC, *et al*. Prone positioning in severe acute respiratory distress syndrome. *N Engl J Med* 2013;368:2159–68.

4 Rosen DS, Macdonald RL. Subarachnoid hemorrhage grading scales: a systematic review. *Neurocrit Care* 2005;2:110–8.

Short answer question case series: ventilatory considerations in a patient with morbid headache

Timothy Horeczko and Timothy Jang

Emerg Med J 2014 31: 423-424 originally published online February 24, 2014
doi: 10.1136/emermed-2013-203564