## Tension pneumocephalus following mask CPAP

Dear Sir,

Continuous positive airway pressure (CPAP) is frequently used during spontaneous respiration for the treatment of post-operative or post traumatic respiratory insufficiency. We would like to describe a case of tension pneumocephalus following the use of mask CPAP in a patient with a head injury.

An 88-year-old, previously fit and independent man was admitted to casualty following a road traffic accident. On examination, he was noted to have a graze over his right temporal region, a right subconjunctival haemorrhage and rhinorrhea. His X-rays showed a fractured right zygoma and three fractured ribs. His Glasgow Coma Scale (GCS) on admission was 14.

His arterial blood gases deteriorated over the next 12 h to a $PO_2$ of 53 mmHg and a $PCO_2$ of 33 mmHg breathing air. A bilateral intercostal block (T4–T6) was performed using 25 ml of 0.25% bupivacaine and a 21 g needle. By 1 h later the patient had deteriorated further and a subsequent chest X-ray on the ICU showed a large right sided pneumothorax with mediastinal shift. After insertion of a right sided chest drain his oxygen saturation had increased from 80–90%. A decision was taken to administer continuous positive airway pressure (CPAP) of 5 $cmH_2O$ via a face mask. This was well tolerated and his oxygen saturation increased to 96%.

Over the next 30–60 min the patient became increasingly drowsy with an eventual GCS of 7 and an oxygen saturation of 96%. A CT brain scan after intubation and ventilation, showed a massive amount of air in the skull vault extending into the posterior fossa and around the spinal cord. Fractures through the right frontal and maxillary sinuses were also noticed.

The patient was transferred to the regional neurosurgical centre where a subdural drain was inserted via a right frontal burr hole. He was ventilated on ICU.

A repeat CT scan 4 days after admission showed considerable reduction in intracranial air. The patient was successfully weaned from the ventilator, and transferred back to the orthopaedic ward. The GCS before discharge on the fifth day after trauma was 13.

Tension pneumocephalus has been reported previously on only one occasion in association with the use of mask CPAP [1]. It has also been noted as a complication of nasal CPAP used for sleep apnoea [2], manual ventilation via a face mask, and following a general anaesthetic with nitrous oxide using a laryngeal mask [3].

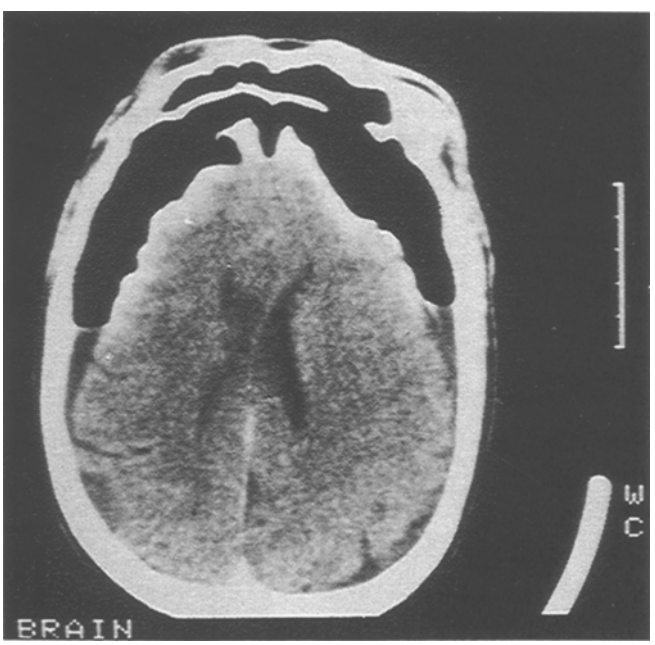


**Fig. 1.** CT brain scan showing a tension pneumocephalus with compression of supra and infratentorial brain

Mask CPAP is a relatively simple and inexpensive form of treatment to improve oxygenation in patients with atelectasis following trauma. It should be used with care in trauma patients, particularly those in whom head injuries cannot be excluded. This case illustrates the potential dangers in using mask CPAP in a patient with a skull fracture. Any evidence of a skull fracture should clearly be taken as a contraindication to mask CPAP. While mask CPAP is continued a CT brain scan should be an early investigation if any neurological deterioration is seen, particularly in the face of good oxygenation.

Yours faithfully

A.E.R. Young and M. Nevin

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## Septic shock and adult respiratory distress syndrome due to *Listeria monocytogenes*

Dear Sir,

A 54-year-old female had been treated for a plasmocytic leukemia and had therefore been neutropenic for 3 months when she was admitted for meningitis and fever of 40 °C. CSF was clear, without bacteria at direct examination. Ceftazidime was empirically started. Four days later, the patient was admitted in ICU because of a shock and respiratory distress (Day 0). Chest X-ray showed widespread alveolar infiltrates. Mean arterial pressure was 40 mmHg; the pulmonary capillary wedge pressure was 7 mmHg, cardiac index 3.9 l/min/$m^2$, arterial systemic resistances 20 IU (with dopamine: 10 µg/kg/min). Lactate was 3.3 mmol/l. The patient was immediately ventilated with $FiO_2 = 1$ and PEEP = +10 $cmH_2O$ ($PaO_2/FiO_2 = 70$). A bronchoalveolar lavage showed an alveolar haemorrhage (72% siderophages) and an aspect compatible with alveolar damage. Blood WBC was 700/$mm^3$, and platelet count was 20000/$mm^3$ on admission.

Cultures of blood and CSF sampled at hospital admission grew *Listeria monocytogenes (Lm)*: amoxicilline and gentamycin were immediately started. The shock required profuse vascular filling, and vasopressors during 6 days. Extubation was possible after 11 days of mechanical ventilation during neutropenia. Chest X-ray showed a disappearance of infiltrates. All bacteriological investigations in the ICU remained negative. The patient left the ICU on D18, the hospital on D52, and was still alive 6 months later.

Blood was drawn from D0 to D10 for determination of 3 cytokines by ELISA: tumor necrosis factor (TNFα), interleukine (IL) 6 (R & D System, Minneapolis USA) and IL1 (Immunotech S.A., Marseille, France) (Table 1).

Bacteremias are frequent in listeriosis, but there are few documented reports describing septic shock (SS) and ARDS due to *Lm* [1]. In shock caused by Gram negative bacilli (GNB), the lipopolysaccharide (LPS) fraction of the bacteria initiates the release of numerous cytokines from host macrophages. Yet the surface components of *Lm* which induce

**Table 1.** Serum cytokine concentration

| | TNF α (pg/ml) | IL1 β (pg/ml) | IL6 (pg/ml) |
|---|---|---|---|
| D.0 | 38 | 2 | 260 |
| D.1 | 31 | 10 | 560 |
| D.2 | 31 | 14 | 150 |
| D.3 | 34 | 40 | 153 |
| D.5 | 36 | 25 | 78 |
| D.10 | 25 | 31 | 10 |
| Normal value | 11 | 19 | 13 |

such a reaction are not clearly individualized; numerous wall components exist, however, which interfere with iron or reactive oxygen molecule, such as the Monocytosis Producing Agent [2], the role of which in virulence of *Lm* has been proved. Macrophages are essential to the hosts defense, which mainly depends on cellular immunity. Various cytokines control the interaction between macrophages and CD4+ cells; the role of interferon (IFN) γ, TNF α and IL6 has been studied during experimental listeriosis. In mice, IFN γ is required for resolution of *Lm* infection [3] although a protective role of TNF α against *Lm* is probable, TNF activity was detectable in neither serum nor spleen during sub-lethal murine infection, and was high in lethal listeriosis [4]. IL1 has the same type of effects as TNF α. IL6 acts as an essential factor in anti-*Lm* resistence; the amount of IL6 seems to be correlated with severity of *Lm* infection: in lethal listeriosis, the level of IL6 in blood and spleen increases until death [5]. So, *Lm* is able to induce the secretion of cytokines by host immune cells, but is hardly ever responsible for SS. SS and ARDS have only been reported in a new-born, who died [1]: the infant had very high levels of circulating TNF α, whereas IL1β, IFN α and γ amounts were moderate. In our report, we note a moderate increase of TNF α and IL1β, and a more important but very transient increase of IL6.

We suggest that the usual inability of *Lm* to generate a cytokine cascade explosion of a magnitude and a duration long enough to produce shock might explain the rarity of SS in Listeriosis. However, in rare cases it apparently occurs.

Yours faithfully,

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# SI Units: conservatism is regression!

Dear Sir,

I read with interest the paper by Redl-Wenzl et al. on the effect of norepinephrine on renal function in septic shock (Intensive Care Med 1993; 19:151–154). Unfortunately the authors used conventional units (serum creatinine in mg %) to express their results. This is a step backwards in the effort to implement the molecular SI units (i.e. the mole and derived units) as widely as possible. This system proposed by R. Dykbaer and K. Jorgensen in 1966 has many advantages over conventional units: standardization, avoidance of confusion, metabolic relevance. The Netherlands, the Scandinavian countries and the UK introduced the molecular SI units in the early seventies, followed by the rest of Europe (except Germany and Austria), Australia, Canada and South Africa. No problems were encountered in any of these countries. Regrettably, in the USA, medical conservatism forced a journal as prestigious as the New England Journal of Medicine to revert to the old system to the dismay of many readers (see N Engl J Med 1993; 328:1040–1041). Intensive Care Medicine is a *European* journal and as such should promote the molecular SI units and not accept any paper not using this system. The young generation of physicians doe not even know what the blood glucose or serum creatinine concentrations mean in mg %. Does the old generation remember what a serum sodium expressed in mg/l means?

Yours faithfully,

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