or hypercapnic that the haemoglobin-oxygen dissociation curve is shifted well to the left. In either case a saturation of 95% should start alarm bells ringing.

The lesson to be learnt is that for patients with normal lungs breathing high inspired concentrations of oxygen the doctor should not be comforted by an oxygen saturation of less than 98-99%. A lower saturation without obvious cause should alert the doctor to measure the arterial blood pressures. Pulse oximetry remains an invaluable aid if its results are interpreted correctly.

VINCE MAK

Division of Medicine,
Guy's and St Thomas's Medical and Dental School,
St Thomas's Hospital,
London SE1 7EH

1 Davidson JAH, Hosie HE. Limitations of pulse oximetry: respiratory insufficiency—a failure of detection. *BMJ* 1993; 307:372-3. (7 August.)

Misunderstanding leads to dangerous practice

EDITOR,—A recent editorial and lesson of the week have highlighted the potential limitations of pulse oximetry and the dangers of inappropriately relying on it to identify ventilatory failure.[1,2] We endorse the cautions recommended with regard to its use during anaesthesia and recovery. We have recently become concerned about a potentially dangerous lack of understanding when pulse oximetry is used to titrate oxygen treatment when patients are transferred by ambulance.

In the past six months five patients with infective exacerbations of chronic obstructive pulmonary disease have been admitted with hyperoxia and dangerous hypercapnia. On each occasion the patient had high concentrations of oxygen administered in the ambulance while arterial oxygen saturation was maintained above 90%. On arrival each patient was hyperoxic (range 18·0-40·0 kPa) and severely hypercapnic (range 10·0-22·0 kPa). Three patients were stabilised with a reduction in fractional inspired oxygen, one required doxapram, and another needed intermittent positive pressure ventilation.

We believe that these patients' condition was made worse by the inappropriate use of pulse oximetry due to lack of knowledge. Although there are many circumstances in which monitoring of arterial oxygen saturation may save lives, it must be used with caution in patients with chronic obstructive lung disease. The principles of controlled oxygen treatment in ventilatory failure are well known to every medical student and should not now be forgotten.

JOHN LEAR
M D L MORGAN

Glenfield Hospital,
Leicester LE3 9QP

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2 Hutton P, Clutton-Brock T. The benefits and pitfalls of pulse oximetry. *BMJ* 1993;307:457-8. (21 August.)

Use your nerve stimulator

EDITOR,—J A H Davidson and H E Hosie highlight the dangers of relying on arterial oxygen saturation to indicate adequacy of ventilation.[1] We wish to comment on several aspects of their management.

Muscle relaxation was achieved with alcuronium, but the authors do not state whether a peripheral nerve stimulator was used to monitor the degree of neuromuscular blockade intraoperatively. It is common practice to monitor neuromuscular blockade both during surgery, to ensure good relaxation leading to a near bloodless field, and during reversal of the neuromuscular blockade.

The authors administered neostigmine and glycopyrrolate and stated that incomplete reversal was apparent; presumably this was on clinical grounds. A further dose was given and reversal of neuromuscular blockade was judged adequate, as was respiratory function, and the patient was extubated. No comment was made regarding the variables used to assess respiratory effort.

It is notoriously difficult to assess the adequacy of reversal of neuromuscular blockade clinically. A patient with clinical signs indicating full reversal—for example, normal muscle power and an effective cough—may still have 70% of the acetylcholine receptor sites occupied by muscle relaxant.[2] A peripheral nerve stimulator used at this point would have indicated incomplete reversal despite clinically adequate respiratory effort.

After the patient's arrival in the recovery ward her respiratory function seems to have deteriorated steadily until help was summoned, when she was in a near moribund state. Arterial blood gas pressures indicated an extreme respiratory acidosis, which was unlikely to be corrected by a respiratory stimulant such as doxapram. The only appropriate action in this case was immediate intubation and ventilation.

It is well known that oxygenation may be maintained by diffusion despite apnoea—a fact often used during anaesthesia for rigid bronchoscopy. The authors recognise that pulse oximetry will not detect respiratory failure, but their statement that it will detect hypoxaemia is not true in all situations. A pulse oximeter measures functional haemoglobin oxygen saturation, which may be appreciably different from the percentage total haemoglobin oxygen saturation if other haemoglobin types are present. For example, a pulse oximeter will give a misleadingly high haemoglobin oxygen saturation in patients with appreciable amounts of carboxyhaemoglobin, who may be hypoxic, and results may also be misleading in patients with haemoglobinopathies. Thus not only does a pulse oximeter fail to measure adequacy of ventilation but it does not always reflect the true state of oxygenation.

The authors have presented an interesting case that highlights one of the pitfalls of overreliance on pulse oximetry in assessing respiratory function. The patient's deterioration might have been avoided if neuromuscular blockade had been monitored earlier.

K A POTTINGER

Anaesthetic Department,
Walsgrave Hospital NHS Trust,
Coventry CV2 2DX

M T WEISZ

Anaesthetic Department,
St Mary's Hospital,
London W2 1NY

1 Davidson JAH, Hosie HE. Limitations of pulse oximetry: respiratory insufficiency—a failure of detection. *BMJ* 1993;307:372-3. (7 August.)
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Pulse oximetry a poor guide to limb perfusion

EDITOR,—Although principally concerned with the failure of pulse oximetry to detect hyperventilation in patients receiving supplementary oxygen, Peter Hutton and Tom Clutton-Brock's editorial also alludes to the poor performance of pulse oximetry when it is used to assess peripheral perfusion.[1] The sensitivity of pulse oximeters allows pulse signals to be detected when pulse pressure is too low to provide adequate tissue perfusion and in the presence of proximal arterial occlusion.[2] This point deserves further emphasis, particularly as pulse oximetry has been advocated for use in assessing limb perfusion after trauma.[3]

Severinghaus and Spellman showed persistence of the pulse oximeter signal with normal saturations during experimental complete clamp occlusion of the brachial artery. They also observed the absence of digital blood flow (determined by plethysmography) while the pulse oximeter continued to function, although at this point saturations often fell slowly.[2]

PAUL GIBB

William Harvey Hospital,
Ashford,
Kent TN24 0LZ

MARK CHURCHILL

Kent and Canterbury Hospital,
Canterbury,
Kent CT1 3NG

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Ventilate immediately in severe respiratory acidosis

EDITOR,—J A H Davidson and H E Hosie provide a useful account of postoperative respiratory depression occurring in a patient with apparently normal arterial oxygen saturation.[1] Failure of theatre recovery staff to comprehend the limitations of pulse oximetry contributed to a delay in diagnosis and appropriate therapeutic intervention.[2,3]

I was most concerned, however, by the description of the subsequent management of the patient as effective resuscitation was delayed while naloxone and doxapram were administered. Furthermore, after the patient was intubated tidal volumes were measured, resulting in a further delay before intermittent positive pressure ventilation was started.

As a potentially life threatening respiratory acidosis had been proved and the patient was unresponsive it would have been more appropriate to protect the airway by intubation and start assisted ventilation immediately, in accordance with recently published guidelines for advanced life support.[4] Though measuring tidal volume and assessing the response to naloxone are important in this clinical situation, resuscitation should have been started without delay.

STEPHEN COCKROFT

Shackleton Department of Anaesthetics,
Southampton General Hospital,
Southampton SO9 4XY

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Transcutaneous carbon dioxide monitoring useful in children

EDITOR,—As paediatricians dealing with respiratory failure and disorders of respiratory control, we were not surprised by the lesson in J A H Davidson and H E Hosie's paper.[1] We were surprised, however, that the authors did not mention the use of transcutaneous monitoring of partial pressure of carbon dioxide. This is used routinely alongside pulse oximetry in our unit for infants and children with moderate to severe respiratory failure. In addition, it is used for all patients in our paediatric intensive care unit and those receiving high dependency care, so that arterial blood gas sampling is required less frequently.

We use Hewlett Packard or Kontron monitors with the sensor heated to 42°C, allowing the sensor to be resited every 12-18 hours. An adjustment to