Limitations of pulse oximetry: respiratory insufficiency—a failure of detection

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Pulse oximetry measures the adequacy of oxygenation, not the adequacy of ventilation

Acute respiratory insufficiency may occur through failure of any component of the respiratory apparatus from the brain to the lungs. Causes include acute exacerbation of an underlying disease, such as asthma or chronic obstructive airways disease; interference with the mechanical action of the lungs and chest—for example, trauma or pneumothorax; interference with the neuromuscular function—for example, stroke or poliomyelitis; or even injudicious use of sedative or narcotic drugs.

The resultant hypoxaemia or hypercapnia (or both) may be life threatening, and, although these are often readily apparent from clinical signs, in some instances they may be difficult to detect. The introduction of pulse oximetry, a non-invasive method of measuring arterial oxygen saturation, has certainly enhanced the detection of borderline hypoxaemia. Indeed, pulse oximetry is now widely used, its worth having been proved in many clinical settings. Although pulse oximetry measures adequacy of oxygenation, it does not measure the adequacy of ventilation. We present a case in which over-reliance on the pulse oximeter resulted in failure to detect severe ventilatory insufficiency.

Case report

An apparently fit 70 year old woman receiving replacement therapy for hypothyroid disease was admitted for a L4-5 laminectomy to relieve nerve root compression. Preoperative assessment showed nothing remarkable; she had undergone previous general anaesthetics without event, and she had had a hip replacement three years previously.

After benzodiazepine premedication anaesthesia was induced in a standard fashion with thiopentone and fentanyl (a short acting opiate) and maintained with inhalational agents (nitrous oxide, oxygen, and isoflurane). Muscle relaxation was achieved with alcuronium (a non-depolarising muscle relaxant). Ventilation was adjusted to maintain normocapnia. Apart from a persistent borderline bradycardia the three hour operation was uneventful. On completion of the operation neostigmine and glycopyrrolate were given to reverse residual neuromuscular blockade. Incomplete reversal was apparent and a further dose was given, to good effect. Respiration was judged to be adequate, and the patient was extubated and transferred to the recovery room breathing oxygen enriched air (oxygen 4 litres/min via a Hudson mask).

About one hour after arrival in the recovery ward medical staff were asked urgently to review the patient. Since her return from the operating theatre her heart rate, arterial blood pressure, respiratory rate, and blood oxygen saturation had remained stable. Throughout this period, however, she had remained very drowsy and had now become completely unrousable. Initially the recovery staff had attributed the slow recovery to the effects of the anaesthetic drugs and of the narcotic analgesics, but they were now concerned.

On examination she was cold and clammy, mottled, and not responding to painful stimuli. Heart rate was 48 beats/min, blood pressure 120/80 mm Hg and arterial blood oxygen saturation (measured by pulse oximetry) 95%. Respiration was 16 breaths per minute, and excursion was shallow, but there were no signs of respiratory obstruction. Blood gases taken at this point showed a profound respiratory acidosis: hydrogen ion concentration 270 nmol/l, P_{CO_2} 37·4 kPa, P_{O_2} 14·2 kPa, bicarbonate concentration 9·3 mmol/l, and base excess −21·2 mmol/l. Naloxone (an opiate antagonist) and doxapram (a respiratory stimulant) were given with no effect.

After laryngoscopy and intubation without sedation, tidal volume, measured with a Wright's respirometer, was found to be less than 100 ml. Peripheral nerve stimulation showed residual neuromuscular blockade, and on the presumptive diagnosis of increased sensitivity to neuromuscular blocking drugs a further dose of neostigmine and glycopyrrolate was given. Little improvement was seen.

Assisted ventilation was started and the patient transferred to the intensive care unit, where ventilation was adjusted to reduce the P_{CO_2} slowly to avoid the deleterious effects of a rapid reduction of P_{CO_2} that may follow a period of hypercapnia.[1]

Muscle power gradually improved, and the patient was extubated 24 hours after admission to the intensive care unit. Although subsequent neurological examination failed to show any signs of muscle weakness or fatiguability, the findings on electromyographic tests were strongly suggestive of subclinical myasthenia gravis. This diagnosis was confirmed by a high level of antibodies to the acetylcholine receptor found at the neuromuscular junction. We later discovered that her previous operation had been performed under a local anaesthetic with supplementary inhalation anaesthesia and no neuromuscular blocking agents had been used. The most likely explanation is that while this patient was normally asymptomatic, the functional reserve at the neuromuscular junction was severely decreased, resulting in extreme sensitivity to agents interferring with neuromuscular transmission.

Comment

The introduction of the pulse oximeter into clinical practice has been of undoubted benefit. Severinghaus and Astrup described pulse oximetry as "arguably the most significant technological advance ever made in the monitoring of the wellbeing and safety of patients during anaesthesia, recovery, and critical care."[2] Pulse oximetry does, however, have its limitations. Although oximetry will measure the oxygenation of arterial blood, it does not indicate the adequacy of carbon dioxide elimination and thus does not signal the adequacy of ventilation. Thus although it will detect hypoxaemia—that is, failure of oxygenation—it will not indicate hypercapnia—that is, ventilatory failure.

The limitation of the pulse oximeter as a respiratory monitor is highlighted by this case. In the absence of abnormal vital signs and with apparently good tidal volumes, misting on the oxygen mask on exhalation, and normal arterial oxygen saturation, the presence of

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respiratory depression and ventilatory failure was not diagnosed, resulting in a profound, life threatening respiratory acidosis. The diagnostic problem may have been compounded by the supplemental oxygen, which may have maintained the oxygen saturation in spite of respiratory depression.

Ideally, the only satisfactory method of monitoring ventilation is to measure both the adequacy of oxygenation and the adequacy of carbon dioxide elimination. Pulse oximetry is non-invasive, easy to use, and widely available. End tidal carbon dioxide concentration, however, is more difficult to measure (capnography), and, although routinely measured in the operating theatre, is not widely measured elsewhere. The alternative of measuring arterial blood gases will indicate P_{O_2} and P_{CO_2}, but the procedure is more invasive and at best is performed only intermittently.

As this case shows, respiratory depression may develop insidiously, may not be revealed by routine monitoring of vital signs, and may be present despite a normal oxygen saturation. Clinicians should be aware of the limitations of oximetry and not rely on this technique as the sole means of assessing the adequacy of ventilation, be it in a critically ill patient in the intensive care unit or a patient receiving sedation for minor surgery. Moreover, if respiratory depression or failure is suspected the adequacy of carbon dioxide elimination should be assessed.

1 Brown EB, Miller FA. Ventricular fibrillation following rapid fall in alveolar carbon dioxide concentration. *Am J Physiol* 1952;**169**:56-60.
2 Severinghaus JW, Astrup PB. History of blood-gas analysis VI: oximetry. *Journal of Clinical Monitoring* 1986;**2**:270-88.

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Rationing in Action

Prioritising health services in an era of limits: the Oregon experience

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This paper is based on a speech given at the conference on Priority Setting in the Health Service, London, March 1993. A collection of papers from the conference will be published in book form later this year.

How do we decide who should receive the benefits that medical science has to offer? One approach to this decision process, that used by the state of Oregon, is described: who and what are covered, and how health care is financed and delivered, are considered. Oregon's priorities were set on the basis of broad consensus. The objective of health care reform, it was agreed, is to improve, maintain, or restore health—not universal coverage, access to health care, or cost containment. A Health Services Commission was created to consider clinical effectiveness and, through public involvement, to attempt to integrate social values into the priority list. Oregon's legislature can use the list to develop an overall health policy which recognises that health can be maintained only if investments in several related areas are balanced.

As we approach the end of the twentieth century, health care systems around the world are struggling with the dual problems of cost and access. Although there are vast differences between the British system and the American system—and between these systems and those in Canada, Germany, or New Zealand—there is a central issue shared by all nations: what are we buying with our health care dollars and what is the relationship between these expenditures and health?

As populations age and technology expands the cost of health care rises. At the same time we find ourselves facing the need for increased investments in education, in infrastructure, in transportation systems, and in addressing a host of other pressing social problems such as environmental pollution, crime, and substance abuse. The competition for limited public resources between these diverse needs means that we can no longer afford to do everything that medical science has to offer for everyone who might benefit from it. In short, we must set priorities. The question is, how do we decide?

In this paper I will examine how this question was answered in the state of Oregon. My purpose is not to convince you of the merits of the Oregon process, nor to draw any conclusions about its possible relevance to the United Kingdom. Rather, my purpose is to describe our experience as objectively as I can and to share with you what insights I have gained through the experience from my dual perspective as both an American politician and a primary care physician.

Framework for health care reform

Health care reform can be viewed as a debate over how to answer three questions—Who is covered? What is covered? How is it financed and delivered?—asked in the context of an ultimate objective. (This framework is drawn from Aristotle's "teleologic" view of change, according to which change (or reform) must be driven by a clear objective, or final cause, and by three subsidiary factors: the material cause, the formal cause, and the efficient cause.) Successful reform, then, must start with consensus on a clearly articulated objective and must explicitly answer these three questions in a way that is consistent with that objective.

The need for consensus on an objective may sound obvious, but consider the current national health care reform debate in the United States, where the objective seems to be to reduce cost, to improve access, or both. But is reducing cost really the end or is it the means to an end? Why do we want to reduce costs? Because cost is a major barrier to access. Why do we want people to have access to health care? Because we want people to be healthy, which is important to individuals and to our society. Thus, both reducing costs and improving access are actually means to an end—the end, or objective, being to improve, maintain, or restore health. I will elaborate further on this point later.

Who is covered?

Now let us turn to the three questions. The first question—"who is covered?" is not really at issue—or at least is not particularly controvesial. Currently in the United Kingdom, for example, or in Canada, or New Zealand, the answer to this question is "everyone." These countries have developed systems in which virtually all citizens have coverage for some level of health care: universal coverage—with eligibility based generally on citizenship.

The United States, however, has never had a national policy of universal coverage. In fact, eligibility for coverage under the two major government financed programmes, Medicaid and Medicare, is based not on citizenship but rather on category. These two

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