Failed intubation

To the Editor:

The editorial responses to Davies *et al.*[1] re-emphasize the importance of assessment, and of familiarity with any technique and equipment which the anaesthetist may use when intubation fails. The importance of learning drills for failed intubation early in postgraduate training, and for the availability of anaesthetists' assistants,[2] is mentioned by all of the authors. Fear,[3] in his concluding paragraph, raises the medico-legal use of published management recommendations and professional standards. He correctly indicates that, if litigation occurs, expert testimony and cross-examination will result in these discussions carrying significant weight in the evaluation of defendants' actions. It is not clear whether Fear believes this is appropriate.

The alternative to the use of published discussions of professional standards and recommendations is a defense from within a void of professional discussion about appropriate protocols. This would promote one of two extremes. First, this lack of fine-grained discussion would lead to very low standards of care being the rule for judging professional practice. Very low standards for defense of professional behaviour has the disadvantage of making challengeable the disciplinary actions of professional associations. Monitoring of professional behaviour according to vague requirements is often viewed by the courts as an illegal restriction of practice. Vague or poorly articulated standards also inhibit the improvement and development of the profession in support of the well-being of patients. The second extreme is that inconsistency between "medical communities" could lead to enforcement of legal rather than medical-informed definitions of what a patient should be able to expect. It is preferable for legal judgments to be based on professional discussions which discuss controversies and limitations so that individual anaesthetists are not held hostage to arbitrary standards in whose formation they have not participated.

Of greater importance, however, is the professional and ethical commitment to evaluate procedures for their effectiveness and safety. As reflected in the article and the editorials, there is controversy over the effectiveness and safety of protocols for failed intubation, and this may vary more among anaesthetists than among protocols. At the very least, such controversy and inconsistency demands careful data gathering for retrospective review, although a prospective randomized study of the protocols by trained anaesthetists would probably yield a more dependable data-base for evaluation (although this might be difficult to implement).

The possibility of litigation, and the development of carefully evaluated protocols, combine to support detailed discussions and research. Physicians' knowledge, skill, and focus on patient interest is the ethical foundation for patient trust. This is particularly true for patients under general anaesthesia who must trust anaesthetists to act proficiently and in their best interests. Discussions of appropriate protocols in professional journals and forums, evaluations of safety and efficacy and dissemination of information and skills through educational protocols are essential to justify and fulfill this trust.

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REFERENCES

1 *Davies JM, Weeks MB, Crone LA, Pavlin E.* Failed intubation in the parturient. Can J Anaesth 1989; 36: 668–74.
2 *Tunstall ME.* Editorial: failed intubation in the parturient. Can J Anaesth 1989; 36: 611–3.
3 *Fear DW.* Editorial: failed intubation in the parturient. Can J Anaesth 1989; 36: 614–6.

Pulmonary artery catheter failure

To the Editor:

During the course of administering an anaesthetic to a 66-year-old man for resection of an abdominal aortic aneurysm, a pulmonary artery catheter was inserted (American Edwards Laboratory, 93A-131-7F). The insertion of the catheter was without incident and the catheter functioned normally during the course of surgery and immediately after transfer into the recovery room.

Two hours after the patient was admitted to the recovery room, we were informed by his nurse that the catheter has become "wedged." In fact, when reading from the distal port, a normal pulmonary artery waveform was evident. On switching to the proximal port (right atrium) only a flat line, without distinguishing features, was evident on the screen. The catheter was flushed, first with the in-line flush device, then with a heparinized-saline loaded syringe, without change in the waveform. However, when flushing with the syringe, a stream of clear fluid was seen to exit the catheter at the 109 cm mark, close to the horse-tail end of the catheter. When the catheter was more closely examined a 4 mm linear fracture was evident at this point. The fracture was

covered with waterproof tape and a dampened right atrial waveform was seen on the monitor. The catheter was then removed. With the fracture occluded, the proximal port was flushed and 0.5 ml of clotted blood was recovered. We were unable to determine from examining the catheter how it came to be damaged. The patient appeared to have suffered no ill effects.

On reviewing the incident, we felt that there was a potential for two pathophysiologic events. The first was that of repeated clot formation and release into the central circulation with repeated flushing of the catheter. The second was the potential for entrainment of air through the fractured catheter and into the right atrium.

We report this as an uncommon cause of pulmonary artery catheter failure. This occurrence re-emphasized the teaching that assessment of "a problem with a waveform" should begin with a careful inspection of the patient end of the system first.

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Rate-pressure product

To the Editor:

What an interesting paper by Miller and Martineau[1] on the therapeutic effect of esmolol in patients with intraoperative myocardial ischaemia. However, it was such a pity that such an innovative paper was marred by repeated references to rate–pressure product as a derived index of myocardial oxygenation. As they correctly state, coronary filling time is dependent on heart rate. Rapid heart rate may thus shorten this period and interfere with myocardial oxygen delivery. This is a distinct discreet physiological phenomenon. Systolic blood pressure is one of many factors determining myocardial oxygen demand. It is also a discreet physiological phenomenon. Nobody, as yet, has provided a mathematical relationship between these two phenomena. It is therefore unscientific to multiply them together, divide them into each other or offer other arithmetical manipulation until an arithmetical relationship has first been proven. I do concede that when both these variables reach their upper limits the temptation to multiply them together and produce a figure of many thousands seems irresistible. Physiology is difficult enough to understand when each phenomenon is considered on its own. It is time that nonsense like "rate-pressure product" was dropped from anaesthetic parlance.

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REFERENCE

1 *Miller RD, Martineau RJ.* Bolus administration of esmolol for the treatment of intraoperative myocardial ischaemia. Can J Anaesth 1989; 36: 593–7.

REPLY

We are grateful for the thoughtful comments expressed by Dr. O'Sullivan regarding our use of rate–pressure product (RPP) as an index of myocardial oxygen consumption ($M\dot{V}O_2$).[1] We would concur that RPP is only an indirect estimate of MVO_2, which may be misleading when either component of the product of heart rate (HR) and systolic blood pressure (SBP) diverges from the other. For example, a similar RPP will result with a HR of 200 bpm and SBP of 50 mmHg, as would occur with a HR of 50 bpm at a SBP of 200 mmHg. Despite its limitation, we disagree with several points raised by Dr. O'Sullivan.

First, $M\dot{V}O_2$ is not determined by as many factors as was suggested, but rather is a function of three primary determinants: heart rate, contractility, and ventricular wall tension. The RPP incorporates two of these factors: one directly (HR), and the other indirectly (SBP as an estimate of afterload). Secondly, we do not feel it is entirely unscientific to multiply these two indices. The RPP has been used by cardiologists during exercise tolerance testing, where it has been shown that RPP correlates well ($r = 0.83$) with $M\dot{V}O_2$ during exercise in patients with coronary artery disease (CAD).[2] A RPP greater than 20,000 has also been shown to correlate with ischaemic ST-segment changes,[3] as it did in the patient described in our study.[1] Treatment of intraoperative myocardial ischaemia was associated with a significant decrease in RPP, as a result of parallel decreases in HR and SBP.

Unquestionably, a simple and accurate technique for estimating myocardial oxygen consumption with easily measured, non-invasive variables would be desirable for perioperative monitoring of patients with coronary artery disease or those with CAD risk factors. In the past several years, the RPP has tended to fall into disfavour as an estimate of $M\dot{V}O_2$ because of its sensitivity to inverse relationships of HR and SBP. In our patient, the changes in these two variables occurred in parallel, and therefore probably reflected the trend towards decreasing $M\dot{V}O_2$ following treatment. Others have recently proposed the pressure–rate quotient (PRQ) as an alternative index, and have shown that the PRQ effectively predicts myocardial ischaemia in an animal model of coronary stenosis.[4] Any indirect index of $M\dot{V}O_2$ will always have limitations, but if interpreted in the