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