provide pre-induction data for the individual treatment groups.

Charles L. MacAdams MD FRCPC
Ian R. Thomson MD FRCPC
Department of Anesthesia
St. Boniface General Hospital
University of Manitoba
Winnipeg, Manitoba R2H 2A6

REFERENCE

- 1 Miller DR, Wellwood M, Teasdale SJ *et al.* Effects of anaesthetic induction on myocardial function and metabolism: a comparison of fentanyl, sufentanil and alfentanil. *Can J Anaes* 1988; 35: 219–33.

REPLY

We are grateful for the interest in our study expressed by Drs. MacAdams and Thomson. They carefully reviewed our manuscript and correctly identified limitations which were encountered in our assessment of myocardial contractility in patients undergoing narcotic anaesthesia. We concur with some of their observations. Volume loading before and after induction produced only a small increase in systolic blood pressure and thus limited our ability to assess systolic function. We also agree that the imprecision inherent in the method of determination of the left ventricular end-systolic volume index may have decreased our ability to assess systolic function. However, despite these considerations, we believe that there is compelling evidence for a decrease in myocardial contractility associated with narcotic anaesthesia.

First, the large variability associated with our measurements decreased the likelihood of our finding a statistically significant depression in systolic function. The fact that we were able to demonstrate such a difference suggests that the depression was profound. We were able to demonstrate a significant difference because we employed a powerful statistical tool. Analysis of covariance permitted the simultaneous evaluation of the slope and the position of the systolic pressure-volume relation. The figure in the letter from Drs. MacAdams and Thomson demonstrated that the systolic pressure-volume points post-induction were below the pre-induction values. This difference in position may represent a decrease in end-systolic elastance. Our sensitive method of analysis which incorporates both slope and position permitted us to conclude that this represented depression in systolic function.

Secondly, we believe that there is additional evidence for depressed myocardial contractility with narcotic anaesthesia induction. Preload recruitable stroke-work index also decreased with anaesthetic induction. Glower¹ and colleagues have demonstrated that this relation provides a better estimate of myocardial contractility than does end-systolic elastance. There are in vivo measurements, and we do not find it surprising that loss of consciousness with narcotic induction, and the accompanying loss of sensory input, is associated with a reduction in myocardial contractility. Such a demonstration is dependent upon the sensitivity of the measurements. The importance is in the comparisons. In an effort to increase the accuracy of these comparisons we have recently developed

on-line pressure-volume loop capability to pin-point more precisely ventricular end systole for future studies.

Finally, although the negative effects on cardiac function were observed, to varying degrees, with all three narcotics, the adverse effects on myocardial metabolism (and the greatest decrease in diastolic compliance) were observed only after alfentanil. The fact that all three groups were ventilated with equal positive pressure yet showed different diastolic compliance slopes suggests ischaemic-induced changes. This was supported by adverse metabolic changes in the alfentanil group.

In conclusion, we believe that anaesthetic induction with the narcotic agents studied produced small but significant alterations in myocardial function and metabolism (the latter in the alfentanil group) in patients undergoing elective coronary bypass surgery. Patients with impaired ventricular function and those who have acute ischaemia may have a more pronounced response to narcotic induction. Caution should be exercised under these circumstances.

Sallie Teasdale MD FRCPC
Donald Miller MD FRCPC
Richard Weisel MD FRCSC
Departments of Anaesthesia and Cardiovascular Surgery
Toronto General Hospital
University of Toronto
Toronto, Ontario

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Fentanyl, sufentanil, alfentanil and myocardial function

To the Editor:

Although it is stated in the article "Effects of anaesthetic induction on myocardial function and metabolism: a comparison of fentanyl, sufentanil and alfentanil" (*Can J Anaesth* 1988; 35: 219–33) that the patients in the alfentanil group had a progressive decrease in blood pressure for nearly five minutes, this is not shown in Figure 2. The mean arterial pressures in the alfentanil and sufentanil groups were equally depressed (compared with fentanyl) one minute after induction, but it is the sufentanil group which subsequently shows the persistently lowest blood pressures.

It is also stated that induction caused a reduction in cardiac index with all drugs but unfortunately this was not represented diagrammatically. Ejection fraction is shown, and this demonstrates the greatest increase in the alfentanil group. This cannot have been due to a reduction in afterload (systemic vascular resistance was not measured but mean arterial pressure according to Figure 2

is higher in the alfentanil than the sufentanil group) and therefore must have been due to an increase in inotropy. As four patients (12 per cent) in the alfentanil group had phenylephrine and calcium, is this increase in ejection fraction representative of the use of these agents? Also, are these the same four patients who were observed to develop anaerobic myocardial metabolism and reduced diastolic compliance?

At least ten per cent more of the patients in the alfentanil group were beta-blocked and at least five per cent more were calcium channel blocked. We wonder if this could have any bearing on the 12 per cent of patients in the alfentanil group requiring vasoconstrictors and inotropes.

Michael Finn MD

Jonathan Richardson MRCP FFARCS
Department of Anaesthesiology
Oregon Health Sciences University
Portland, Oregon, U.S.A.

Early ether anaesthesia

To the Editor:

Dr. Titrer's article¹ gives interesting insights into the controversies which followed the introduction of ether anaesthesia by Morton in Boston in October 1846. However, New York City on December 12 was not the only North American city outside Boston in which ether was administered before it was used in London and Paris. The Boston Daily Evening Transcript of November 24, 1846 (Figure) published a report of two amputations, one in Salem and one in Danvers, Massachusetts during which the patients had inhaled "the preparation of Dr. Morton."

It is also possible that ether was used in Halifax, Nova Scotia, by mid-December 1846. News of Morton's use of ether in Boston appeared in a Halifax newspaper as early as November 11, 1846.² Dr. Parker, a leading Halifax surgeon, recalled in his autobiography that "Lawrence VanBuskirk, a dentist, practising in Halifax at the time, as soon as he learned that ether was being used by inhalation there in practical dentistry, with commendable enterprise visited Boston and familiarized himself with its use."³ Early in 1848, the *Royal Gazette* of Halifax reported: "As the attention of the public has in several instances of late been called to the use of Ether and Chloroform, by the medical faculty of this city, it is due to Dr. L.E. VanBuskirk to state that more than a year ago, and before any operations had been performed in England under its influence, or had even found their way into the medical Journals of the United States, he had constructed from a

Evening Transcript.

BOSTON, TUESDAY EVENING, NOVEMBER 24, 1846.

NO 3831

The Salem Register relates two cases of amputation, which took place in that city and Danvers last week, under the operation of the preparation of Dr Morton, of this city. The first was that of the young Irish girl, whose arm was so severely mangled on the railroad, on Thursday. The operation was performed by Dr Pierson, of Salem, accompanied by Dr Fisk, who administered the preparation. There was apparently no consciousness of the operation, until near its close when the patient inquired what they were doing; but with inhalation, she was insensible again, and supposed, when she came to, she had been dreaming. The other case was that of Wm A. Osborne, who was injured on the Essex Railroad in the crushing of his foot, by the locomotive running over it. The lower part of the leg was taken off successfully under the operation of the same preparation, administered by Dr Fiske. [Courier.

FIGURE The Boston Daily Evening Transcript, November 24, 1846, report of amputations under "the preparation of Dr. Morton."

diagram sent in a private letter, an inhaler, and was in the constant practice of using the sulph. Ether in dental operations."⁴ However, no reports in November and December, 1846 Halifax newspapers have been found to confirm this claim, so credit for the first British North American (Canadian) anaesthetic must still go to the dentists Adams and George VanBuskirk (Lawrence's brother) in St. John, New Brunswick.⁵

J. Roger Maltby MB BCHIR FFARCS FRCP

Department of Anaesthesia
Foothills Hospital at the University of Calgary
1403 29th St. N.W.
Calgary, Alberta T2N 2T9

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- 2 Anon. Experiment in surgery. *The Royal Gazette* 1846. November 11: 1 (col 2).
- 3 Parker WF. Daniel McNeill Parker MD. His Ancestry and a Memoir of His Life. Toronto: Briggs, 1910: 394.
- 4 Anon. The new discovery. *The Royal Gazette* 1848. February 16: 5 (col 4).
- 5 MacDougall JA. The earliest ether anaesthetic in British North America - a first for St. John, New Brunswick? *Can J Anaesth* 1987; 34: 496-504.