
Sir,—We wish to comment on the letter from McDonald [1] in which the management of a difficult airway is described using alfentanil and propofol. We are unclear why inhalation induction preceded fiberoptic intubation. It has been shown that awake fiberoptic intubation maintains the natural airway and there is easier identification of upper airway structures than when the patient is asleep [2]. Although propofol–alfentanil is useful for intubation without neuromuscular block in uncomplicated patients, in our experience the dose of alfentanil used produces a prolonged period of apnoea. As it was difficult to ventilate the lungs of this patient, the use of a technique likely to cause apnoea appears contraindicated. The argument that alfentanil can be antagonized safely with naloxone is unfounded as there are numerous reports of complications after opioid antagonism by naloxone [3].

We feel that in this case the technique of choice is awake fiberoptic intubation as, in the words of Benumof, “no bridges are burned” [4]. If this was unsuccessful we would then proceed to a retrograde intubation technique with the patient awake at all times.

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**Prediction of arterial from end-tidal $P_{CO_2}$**

Sir,—The correspondence [1] between Dr Farmery and Dr Fletcher on prediction of arterial $P_{CO_2}$ from end-tidal $P_{CO_2}$ is interesting, both theoretically and practically. The observations of Dr Farmery in a young healthy subject of the difference between which the management of a difficult airway is described using alfentanil and propofol. We are unclear why inhalation induction preceded fiberoptic intubation. It has been shown that awake fiberoptic intubation maintains the natural airway and there is easier identification of upper airway structures than when the patient is asleep [2]. Although propofol–alfentanil is useful for intubation without neuromuscular block in uncomplicated patients, in our experience the dose of alfentanil used produces a prolonged period of apnoea. As it was difficult to ventilate the lungs of this patient, the use of a technique likely to cause apnoea appears contraindicated. The argument that alfentanil can be antagonized safely with naloxone is unfounded as there are numerous reports of complications after opioid antagonism by naloxone [3].

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The Hewlett-Packard capnometer (HP47210A) analyses carbon dioxide in the mainstream gas, by means of a window in an airway adapter inserted in the inspired gas pathway. In contrast with sidestream sampling devices, when gas has to pass from the sample port by way of a catheter to the analyser, one might assume that with the Hewlett-Packard instrument there is no delay in the analysis of this gas, and hence the signal output represents the gas composition in the adapter. Unfortunately, the Hewlett-Packard device does not provide a real-time measure of carbon dioxide in the inspired gas pathway. There is a degree of delay, presumably because the signal is manipulated digitally within the measuring device. I have attempted to measure the delay time and the response time of the capnometer and found that there do not seem to be unique values for these variables. In particular, there appears to be a considerable degree of smoothing of the analogue output signal and the 1:8 ratio is altered.

The plot shown by Dr Farmery depends upon end-tidal carbon dioxide measurements and these may well be correct. However, he goes on to make measurements of $dP/dV$, which is the rate of change of $P_{CO_2}$ with exhaled volume, within a single breath. To do this accurately requires the exhaled volume at a particular instant to be related to the $P_{CO_2}$ in the airway at the same instant. Unless he has estimated the degree of delay introduced in the signal by this particular capnometer, such measurements may not be correct. This is because the rate of exhalation is not constant and the constant delay within the measurement device results in a varying volume discrepancy because of the varying expiratory flow. This may explain his observations that the pattern of expiration within the individual accounted for changes in $dP/dV$.

I do not wish to cast excessive doubt upon Dr Farmery’s conclusions, which appear at least in a young healthy subject to be consistent and dependent mainly on end-tidal estimates which will be unaffected by instrument delay. His hypothesis that the influence of tidal volume may be through variations induced in alveolar gas composition can be simply tested experimentally, for example with end-inspiratory pauses of different duration. The main purpose of my letter is to emphasise that the Hewlett-Packard capnometer, although apparently a “real-time” device, is far from being so in practice and within-breath measurements have to take instrument delay time into consideration.

G. B. Drummond
Royal Infirmary, Edinburgh

The correspondence [1] between Dr Farmery and Dr Fletcher on prediction of arterial $P_{CO_2}$ from end-tidal $P_{CO_2}$ is interesting, both theoretically and practically. The observations of Dr Farmery in a young healthy subject of the difference between which the management of a difficult airway is described using alfentanil and propofol. We are unclear why inhalation induction preceded fiberoptic intubation. It has been shown that awake fiberoptic intubation maintains the natural airway and there is easier identification of upper airway structures than when the patient is asleep [2]. Although propofol–alfentanil is useful for intubation without neuromuscular block in uncomplicated patients, in our experience the dose of alfentanil used produces a prolonged period of apnoea. As it was difficult to ventilate the lungs of this patient, the use of a technique likely to cause apnoea appears contraindicated. The argument that alfentanil can be antagonized safely with naloxone is unfounded as there are numerous reports of complications after opioid antagonism by naloxone [3].

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The Hewlett-Packard capnometer (HP47210A) analyses carbon dioxide in the mainstream gas, by means of a window in an airway adapter inserted in the inspired gas pathway. In contrast with sidestream sampling devices, when gas has to pass from the sample port by way of a catheter to the analyser, one might assume that with the Hewlett-Packard instrument there is no delay in the analysis of this gas, and hence the signal output represents the gas composition in the adapter. Unfortunately, the Hewlett-Packard device does not provide a real-time measure of carbon dioxide in the inspired gas pathway. There is a degree of delay, presumably because the signal is manipulated digitally within the measuring device. I have attempted to measure the delay time and the response time of the capnometer and found that there do not seem to be unique values for these variables. In particular, there appears to be a considerable degree of smoothing of the analogue output signal and the 1:8 ratio is altered.

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FIG. 1. Relationship between volume and airway $P_{CO_2}$ vs time.
of a carbon dioxide sample and registration of a signal. This is not an easy task because one cannot be sure exactly when the carbon dioxide "front" reaches the sample window. A simple way to overcome this problem is to measure the time at which the "carbon dioxide signal decay" occurs in relation to the time at which flow reversal occurs, when a subject suddenly and forcibly inspires at some point during expiration (i.e. zero expiratory pause). Figure 1 is an exaggerated illustration of this. Delaying the volume signal by six samples reduced the time lag to zero.

It is interesting to consider what would be the effects of failure to compensate for this time lag. First, one would expect the value obtained for the anatomical deadspace to be erroneously high. This error is dependent on the magnitude of the time lag and on flow rate. As the flow rate is not constant, neither is the error.

Deadspace error = \frac{\text{measured } dP}{\text{true } dV} - 1

(1)

where \( r d \) = deadspace transit time, \( d t \) = time lag and \( f \) = flow rate.

Second, one might expect, prima facie, that the error in the value of \( dP/dV \) is similarly affected. In fact, however, the value of \( dP/dV \) is altered very little by an uncorrected time lag of this magnitude. In addition, the error in \( dP/dV \) appears to be solely a function of \( dP/dV \). It is independent of flow (unlike the deadspace error) and hence remains constant. Any changes in \( dP/dV \) within or between individuals cannot therefore be a result of this measurement error as Drummond suggests.

These assertions are based on empirical evidence (by observation of the effects of differing time lag values in the analysis of prerecorded breaths) and on a computerized mathematical lung model based on equations of alveolar carbon dioxide kinetics [1, 2].

The constancy of the error (E) may be explained more simply as follows:

\[ E = 1 - \frac{\text{measured } dP}{\text{true } dV} = 1 - \frac{dP(t + d\Delta t)/dV}{dP(t)/dV} = 1 - \frac{dP(t + d\Delta t)/dt}{dP(t)/dt} \]

(2)

The function \( dP(t + d\Delta t)/dt \) can be expanded by the expansion of Taylor [3] to give:

\[ P(t + d\Delta t) = P(t) + d\Delta t \cdot \frac{dP}{dt} + \frac{d^2P}{dt^2} \cdot \frac{d\Delta t}{2!} + \frac{d^3P}{dt^3} \cdot \frac{d\Delta t}{3!} + \text{etc} \]

(3)

From equation (3) it can be seen that the measured value of \( dP/dt \) is expressed in terms of derivatives (of successively increasing order) of the real-time values. For exponential functions, all such derivatives are equal and therefore cancel out when substituted in equation (2), the only remaining terms being \( d\Delta t / dt, d^2P/dt^2, \text{ etc} \), which converge. Hence, the measurement error, E, is seen to be constant for a given time lag. The plot of P against V should therefore remain linear as it is virtually independent of flow.

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Magnetic resonance imaging of extradural blood patches

Sir,—It was interesting to see magnetic resonance imaging of extradural blood patches from Beards and colleagues [1]. However, I could not help thinking some opportunities were lost. Only five cases were imaged, all with different histories and physically different. A time course of the natural history of a blood patch cannot be concluded from this study. The rapid reduction of post lumbar puncture headache still appears to be a conflicting argument. Initially it is suggested that displacement of CSF by the blood patch (18-20 ml) relieves the symptoms, but they also noted other occasions where much smaller volumes have been used successfully. There must be other mechanisms involved not described here.

Haematoma formation compressing the spinal was not correlated against reported symptoms from the patient, but this study has not examined this question.

S. J. Wilson
Gloucestershire Royal Hospital


Sir,—In our study [1], we reported imaging of five patients who received extradural blood patching. All patients were imaged on one occasion only, at periods ranging from 30 min to 18 h. Dr Wilson suggests that it is not appropriate to conclude a time course for the natural history of the blood patch from this study. Whilst it is true that we have not performed consecutive imaging in individual patients, it is perhaps difficult for those not involved in magnetic resonance imaging (MRI) to appreciate the logistical difficulties such a study would present. The comment that all the patients were “different” is true in that it was not the same patient imaged over consecutive time periods, but all had received dural puncture leading to post lumbar puncture headache, all had been treated with a trial of conservative therapy which failed, all were adults in their second to fourth decade and in all cases the blood patch was placed at, or one level above, the original puncture site.

We would disagree with the comment that mechanisms other than volume displacement of cerebrospinal fluid by the blood patch must be involved in the rapid reduction of post lumbar puncture headache. This is discussed in some detail on page 186 of our article [1]. It is true, however, that early studies used small injection volumes of only 2-3 ml of blood. After the initial work of Gormley [2] most workers required larger volumes of blood. As discussed, the original study used end-hole spinal needles rather than the Tuohy needles favoured today. We believe that one possible reason for the large injection volume now favoured is that the Tuohy needle causes leakage of blood into the interfascial spaces so that the volume of blood in the extradural space is less than believed.

Whatever the volume of blood remaining within the extradural space after patching, there can be no doubt from our images that there was marked compression of the thecal sac. Over a period of 3-4 vertebral segments around the injection site of the extradural blood patch, there was no residual patent CSF space and the mass effect was surprisingly extensive. Indeed the neuroradiologist involved in the study commented that he was surprised that these patients were not paraplegic in view of the degree of neural compression present.

The use of MRI in patients who do suffer radicular pain is particularly important and should be performed. It was not the intention of this study to investigate this unusual occurrence but rather to examine the natural history of the extradural blood patch. The presence of subcutaneous haematoma was again not correlated with back pain.

The technique of MRI will be important in studying the progression of the extradural blood patch. It must be appreciated that studies of this type are subject to considerable restrictions in terms of patient recruitment, related to both the rarity of the disorder and the extreme rarity of the complications.

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