which occurred during treatment of Menière's disease.[2] A "large volume" of procaine had been used. The author attributed this complication to medial angulation of the needle and to the large volume of local anaesthetic injected. In our case, the volume of local anaethetic used was only 7–8 cc with each block. The needle was inserted perpendicular to the frontal plane, using the anterior tubercle of C6 as a landmark, making it unlikely that the needle was angled medially.

Perhaps there was medial spread of the anaesthetic solution to the contralateral side, whether deep or superficial to the prevertebral fascia. Murphy states that injection deep to the prevertebral fascia may spread the local anaesthetic posteriorly and laterally to the fascia, involving some or all of the roots of the brachial plexus.[1] Our patient did not develop any neurologic changes in the distribution of the brachial plexus.

The possibility of some variation in the anatomy of the stellate ganglion, with communication between the two sides must also be considered. This would allow local anaesthetic to track across to the contralateral side. If this was true, however, one would expect a bilateral stellate ganglion block, rather than only on the contralateral side and the repeated blocks would produce the same results.

The mechanism of production of a contralateral Horner's syndrome after a stellate block remains poorly explained and its occurrence is rare. Nevertheless, this case report illustrates the importance of good monitoring, follow-up and vigilance in the performance of seemingly routine repeated simple procedures. Unexpected results can occur and must be diagnosed and dealt with appropriately.

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Capnography and air embolism

To the Editor:
A recent report[1] by Symons and Leaver describes a case of air embolism resulting in cardiovascular collapse. The authors state that the capnogram quantitates both the severity of the pathophysiological event and the effectiveness of the treatment. The first sign of embolism was the fall in end–tidal carbon dioxide (ET-CO_2) concentration from approximately four to one per cent, preceding by about 45 seconds a decrease in arterial pressure and a change in Doppler sound. We would like to make the following remarks:

1 The tracing in the figure does not show the pattern which is characteristic for pulmonary embolism.[2–4] The fall in ET-CO_2 concentration occurring in the first six expirations is probably caused by air embolism. Thereafter the further fall in ET-CO_2 concentration is likely to be caused by the change in cardiac output illustrated by the decrease in systemic arterial blood pressure.

Changes in cardiac output are well known to result in changes in expiratory carbon dioxide concentration.[5] We therefore believe that the tracing shows a combined effect: pulmonary embolism and change in cardiac output. The capnogram in this case does not provide a quantitative measurement of the primary pathophysiological event.

Illustration of both effects occurring separately are shown in the two figures. Figure 1 is a recording of a typical washout curve: a rapid fall in ET-CO_2 concentration with a gradual return to the preexisting level. The pulmonary artery pressure (PAP) increases at the same time, as is expected in the case of pulmonary embolism.[4,6] There are no changes in blood pressure, central venous pressure and heart rate. This is a typical case of air embolism without systemic haemodynamic consequences. Figure 2 shows a recording with a fall and rise in ET-CO_2 concentration caused by a change in cardiac output.

2 The change in Doppler ultrasound occurred 45 sec after the decrease in ET-CO_2 concentration, at the same time as the fall in blood pressure. Most likely this change in Doppler sound was not caused by air in the heart but by the
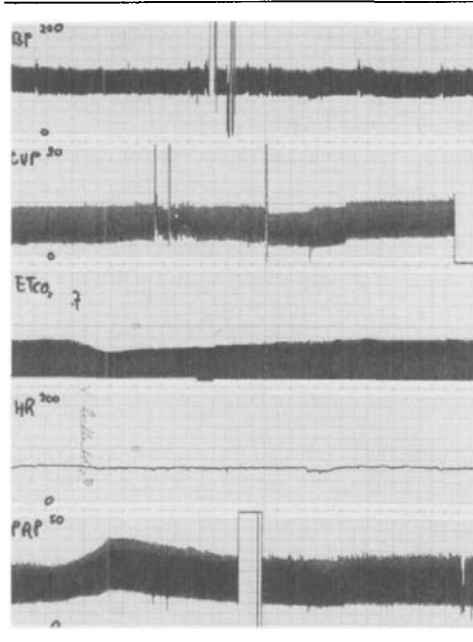
FIGURE 1   Recording made during an episode of pulmonary embolism without changes in cardiac output. Abbreviations: BP – systolic arterial blood pressure; CVP – central venous pressure (mmHg); ET-CO_2 – end-tidal carbon dioxide concentration (vol. per cent); HR – heart rate (beats per min); PAP – pulmonary artery pressure (mmHg).

fall in cardiac output. Probe placement might not have been optimal. From this case it can not be concluded that Doppler sound is an unreliable detection method.

3 In the discussion the authors review specific monitoring methods for the detection of air embolism. Unfortunately they only mention Doppler ultrasound and capnography and exclude pulmonary artery pressure monitoring. This last parameter detects the primary pathophysiological event in venous air embolism: the intense pulmonary vasoconstriction.[6]

4 Symons and Leaver state that the central venous pressure rises as does the pulmonary artery pressure. In a recent study of 16 patients with proven pulmonary embolism a rise in central venous pressure occurred in only 11 patients.[4] We consider central venous pressure monitoring highly unreliable as a detection method for air embolism.
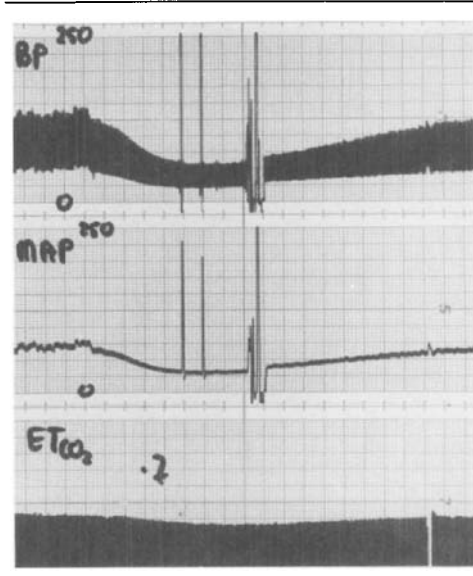

FIGURE 2   Recording made during an episode of pulmonary embolism associated with a reduction in cardiac output.

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