

tive pressure phase of the ventilatory cycle, or by a characteristic noise heard through the esophageal stethoscope when suction is applied. Because a subatmospheric pressure as great as 450 torr may be generated by wall suction, quick detection of this problem is imperative. The subatmospheric pressure alarm appears to be a useful adjunct in detecting the problem when other means have not been decisive.

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### Evidence for *In vivo* Biotransformation of Nitrous Oxide

*To the Editor:*—Dr. Linde and Dr. Avram<sup>1</sup> pose the question: "Can mammalian systems biotransform N<sub>2</sub>O?" I think the answer must be yes. It is known that nitrous oxide can oxidize the cobalt moiety of vitamin B<sub>12</sub> to its bivalent form *in vitro*.<sup>2</sup> It has been clearly established that methionine synthesis is inhibited when rats or humans breathe 50 per cent nitrous oxide for several hours,<sup>3,4</sup> and there can be little doubt that this must be due to oxidation of B<sub>12</sub> occurring *in vivo* as it also happens *in vitro*. Demonstration of the biotransformation of nitrous oxide by this mechanism is difficult. Even if all the B<sub>12</sub> in the body was oxidized (and this does not appear to be the case), the quantity of nitrous oxide required would still be much less than 1 ml, and the detection of this small amount of <sup>15</sup>N<sub>2</sub> formed from <sup>15</sup>N<sub>2</sub>O would be virtually impossible in view of the substantial background level of <sup>15</sup>N<sub>2</sub> in the atmosphere.

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### Asystole after Intravenous Dantrolene Sodium in Pigs

*To the Editor:*—The recommended method of administering dantrolene sodium for malignant hyperthermia (MH) crisis in patients is 1 mg/kg, iv, rapidly, then continuing to 10 mg/kg if needed. Animal studies report no appreciable effect on the heart at these doses in one study<sup>1</sup> and cardiac depression in other studies.<sup>2,3</sup>

We have had two 35-kg MH positive pigs die from intravenous dantrolene administered during an MH crisis. One pig received a 10-mg/kg dose as a bolus;

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the other pig received a total dose of 30 mg (0.43 mg/kg) of dantrolene prior to arrest. Both pigs were in sinus rhythm (rate 130-140) prior to the dantrolene, but developed severe sinus bradycardia followed by cardiac arrest from asystole during the dantrolene administration. The first pig may have been given dantrolene in too high a concentration and too rapidly, but the second pig had an abrupt asystole while the dantrolene was being titrated and arrested after 30 mg was given. Neither pig was collapsing

nor considered terminal when the dantrolene was started. We have had extensive experience in the use of dantrolene in MH pigs in doses up to 10 mg/kg, iv, without cardiac arrest, even with deteriorating animals.

Despite species differences, the use of dantrolene sodium in humans has been recommended because of success in treating porcine MH. The pig is thought to be less sensitive to dantrolene than humans, the recommended dose in the pig being 2 to 2.5 times that for humans. We suggest that the cardiovascular effects of dantrolene may be more pronounced than has been thought, and recommend that intravenous dantrolene be administered slowly and not as a 1 mg/kg bolus.

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## Permanent Paralysis of C-5 after Cannulation of the Internal Jugular Vein

*To the Editor:*—In order to administer parenteral nutrition to a 40-year-old patient after bilateral truncular vagotomy and pyloroplasty, the left internal jugular vein was cannulated according to the technique described by Boulanger.<sup>1</sup> The left side was used due to several failed attempts at right venipuncture. Sorenson® equipment was used without any problems; correct catheter tip position was demonstrated by roentgenograms.

In the postoperative period, approximately 24 hours after puncture, a flaccid paralysis of the left arm appeared. The catheter was removed without subsequent improvement. Twenty days later, an electromyographic (EMG) analysis revealed a complete denervation of C-5. Because the brachial plexus apparently was not stretched during surgery, we believe this neurologic damage was caused by the puncture of the internal jugular vein. An additional EMG analysis was not performed because the patient died 10 days later due to diffuse peritonitis and septic shock. An autopsy was not performed.

Transient or permanent neurologic damage related to cannulization of the superior vena cava is unusual. This complication has been attributed to direct needle trauma,<sup>2</sup> diffusion of local anesthetic,<sup>3</sup> hematoma compressing the nerve,<sup>4</sup> and chemical action of drugs and fluids escaping from the vein when the catheter is only partially inserted.<sup>5</sup> In our case, we believe the

neurologic damage of C-5 was probably caused by the needle used for puncture. This problem probably can be minimized by performing punctures in awake patients whenever possible, and advising them to report any paresthesia which might be felt during the insertion.

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