tesla GE Signa° system. The ventilator is completely powered by wall oxygen and functioned well within the magnetic environment. Expired gas was exhausted through a Puritan-Bennett bellows spirometer (with alarm removed the unit proved to be nonmagnetic) so that an estimate of tidal volume could be made. Of the three patients anesthetized to date, the exhaled gas volumes agreed with the settings on the ventilator, and arterial blood gas samples obtained during the imaging session had $P_{O_2}$ and $P_{CO_2}$ levels appropriate for the ventilator settings. Examination of the MR images obtained while this ventilator was in use showed that the images were of diagnostically acceptable quality and that there was no perceptible degradation. This would be expected, considering that this ventilator is completely fluidic and contains no electrical components. Our experience supports the experience of Dunn et al., who used an earlier and a much less modified version of this ventilator on awake, passively ventilated volunteers in a 0.5 tesla magnet.

The basic anesthetic approach we use in our patients is a totally intravenous technique consisting of sodium thiopental, a narcotic, and a muscle relaxant as required to maintain the appropriate anesthetic state. Patients are anesthetized and intubated outside the magnet room then transferred to the magnet in the anesthetized state. An esophageal stethoscope is used to monitor breath and heart sounds. Blood pressure is typically monitored using an Accutorr° 2A (Datascopc Corp., Paramus, NJ) oscil- loto nometeric device. In the GE Signa° system this unit has no attraction to the magnet at the 150 gauss line, and the couplings and cuff are essentially nonmagnetic.

As emphasized by several authors, it is crucial to recognize the dangers of bringing even small ferric-containing materials to the vicinity of the magnet. Thus patients, particularly those who have been in an intensive care unit, must be scrupulously searched for small ferric components such as pins and restraint buckles because these may prove a danger to the patient or create problems with signal acquisition. Also commonly carried devices such as keys, stethoscopes, and pens can be extremely dangerous in the MRI environment. Even with these constraints, our experience to date suggests that even quite ill patients, e.g., those with acute head injury, can be cared for during MRI and that this group of patients can receive the benefit of this diagnostic modality when indicated.

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Atropine Resistance in Brain-dead Organ Donors

To the Editor:—The study by Bruce1 on brain-dead organ donors was interesting, and prompts me to describe a case that illustrates the cardiac response to atropine in such patients.

REPORT OF A CASE

A 65-yr-old woman weighing 53 kg was admitted to the Intensive Care Unit with a 1-day history of a deteriorating level of consciousness. Physical examination revealed a dense right-sided hemiplegia. Spon-
taneous respiration and verbal responses were absent, the eyes did not open to pain, and the pupils were mid-dilated with a sluggish reaction to light. CT scanning of the head demonstrated massive subarachnoid hemorrhage with midline shift and compression of the cerebral sulci. Cerebral angiography revealed a large basilar artery aneurysm. After neurosurgical consultation, a joint decision was made to treat the patient medically with full respiratory and cardiovascular support. The patient continued to deteriorate, and, within 5 days, had become totally unresponsive with fixed dilated pupils and a flat EEG. She satisfied all criteria for the diagnosis of brain-death as proposed by the Royal Colleges and Faculties of the United Kingdom.²

Prior to withdrawal of life support systems, the efficacy of atropine was assessed in this patient. Incremental doses of atropine 0.6 mg iv were administered up to a total of 3.6 mg over 5 min. However, arterial blood pressure and heart rate remained unchanged, and were 125/60 mmHg and 75 beats/min, respectively.

Atropine resistance has subsequently been confirmed in four other patients with brain-death.

The phenomenon of atropine resistance in brain-dead patients is consistent with the underlying pathophysiology. Brain-death is associated with pannecrosis of nervous tissue with extensive foci throughout the cerebrum and brain-stem.⁵ Presumably, extirpation of the vagal motor nucleus (nucleus ambiguous) abolishes tonic vagal activity and, hence, the anti-cholinergic effect of atropine on the heart is annulled. In this context, it is interesting to note that a low systolic blood pressure without a slow pulse rate were cited as the cardiovascular signs most likely to be associated with brain-death by the Collaborative United States Study on Brain-Death.⁴

Atropine resistance in brain-dead organ donors may be of interest to anesthesiologists involved in the care of such patients for at least two reasons. First, it may be used as an additional confirmatory sign of brain-stem death. Second, bradycardia occurring in such patients will presumably be refractory to atropine. After ruling out other causes for bradycardia, it may be necessary to consider isoproterenol infusion or temporary transvenous pacing to maintain an adequate heart rate.

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