TABLE 1. Catecholamine Concentrations

	Low Blood Pressure* (60/30 mmHg)	High Blood Pressure* (182/126 mmHg)	Postoperative		
Test			I Month	3 Months	Normai
Norepinephrine					
(pg/ml)	2,524	40,120	178	13,760	110-410
Epinephrine	376	188	86		< 50
(pg/ml) Dopamine	3/6	100	00		\ \50
(pg/ml)	108	5,200			<30

^{* 7} min apart.

showed a large, hypervascular mass occupying and extending from the medial segment of the left lobe of the liver, consistent with a hepato-blastoma.

Anesthesia for partial hepatic resection consisted of fentanyl (62 $\mu g \cdot k g^{-1}$), pancuronium, and isoflurane. Induction of anesthesia was tolerated well. Vital signs were stable for 10 min after incision. Manipulation of the hepatic tumor resulted in increased arterial blood pressure to 220/130 mmHg, lasting for 2–3 min, followed by a decrease to a systolic pressure of 60 mmHg. Hemoglobin oxygen saturation, end-tidal carbon dioxide, arterial blood gases, serum glucose, and ionized calcium were unchanged during these transient increases in blood pressure.

Cycles of alternating hyper/hypotension persisted until shortly before the completion of interruption in the tumor's blood supply. Catecholamine concentrations were measured intraoperatively during one of the periods of blood pressure instability and were analyzed by Smith Kline Bio-Science Laboratories using high-pressure liquid chromatography with a cation exchange column and an electrochemical detector (table 1).

Microscopic appearance of the tissue was interpreted as anaplastic hepatoblastoma. The patient's liver tumor recurred, and 3 months later she developed a lesion in the right humerus. Biopsy of this lesion and review of the previous slides led the pathologist to change the diagnosis to neuroblastoma. The patient's condition continued to deteriorate despite therapy, and she died 5 months after surgery. Permission for autopsy was not obtained. It is unknown whether the liver tumor was a primary tumor or a metastasis from an occult primary tumor.

Anesthesiologists should be aware that neuroblastomas may masquerade as other tumors¹⁻³ and that, neuroblastomas may secrete catecholamines that can cause hemodynamic instability during resection.⁴⁻⁸ As many as 19% of patients with documented neurogenic tumors are found to be hypertensive.⁴ The case reported here is unusual

in that typically there is a paucity of storage granules within neuroblastomas as compared to pheochromocytomas, and, therefore, catecholamines are not stored and released in large quantities.^{4,8,9}

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REFERENCES

- Reynolds CP, Smith RG, Frenkel EP: The diagnostic dilemma of the "small round cell neoplasm": Catecholamine fluorescence and tissue culture morphology as markers for neuroblastoma. Cancer 48:2088-2094, 1981
- Lopez-Ibor B, Schwartz AD: Neuroblastoma. Pediatr Clin North Am 32:755-778, 1985
- Bond JV: Neuroblastoma metastic to the liver in infants. Arch Child 51:879-882, 1976
- Weinblatt ME, Heisel MA, Siegel SE: Hypertension in children with neurogenic tumors. Pediatrics 71:947-951, 1983
- Von Studnitz W, Kaser H, Sjoerdsma A: Spectrum of catechol amine biochemistry in patients with neuroblastoma. N Engl J Med 269:232-235, 1963
- Voorhess ML, Gardner LI: Urinary excretion of norepinephrine, epinephrine and 3-methoxy-4-hydroxymandelic acid by children with neuroblastoma. J Clin Endocrinol 21:321–335, 1961
- Kedar A, Glassman M, Voorhess ML, Fisher J, Allen J, Jenis E, Freeman AI: Severe hypertension in a child with ganglioneuroblastoma. Cancer 47:2077-2080, 1981
- Kogut MD, Kaplan SA: Systemic manifestations of neurogenic tumors. J Pediatr 60:694-704, 1962
- Voorhess ML: Neuroblastoma-pheochromocytoma: Products and pathogenesis. Ann N Y Acad Sci 230:187-194, 1974

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Burns Associated with Pulse Oximetry during Magnetic Resonance Imaging

To the Editor:—We recently have become aware of two patients who suffered burns associated with the use of a pulse oximeter during magnetic resonance imaging (MRI) under general anesthesia. One patient, a man who had undergone imaging of the cervical spine, sustained a full-thickness burn requiring skin grafting of the tip of the little finger where the pulse oximeter sensor had been placed. The second patient, an infant, underwent scanning of the head and had the pulse oximeter probe placed on the great toe with a loop of the connecting cable taped over the leg in order to provide mechanical strain relief as the imaging

platform was moved in and out of the bore of the magnet. Afterwards, a superficial linear burn was found where the cable had been taped to the leg. In neither of these cases did there appear to be any failure of the pulse oximeter or its sensor.

The risk of burns due to pulse oximeter sensors and other metallic objects in proximity to patients during MRI has recently been reported to the radiology community.¹⁻⁵ However, of the (mostly older) published recommendations that we found regarding anesthesia for MRI, none has mentioned this particular hazard.⁴⁻⁸

The problem is believed to be due to electrical currents induced, according to Faraday's Law, in all conductive materials exposed to the radio-frequency magnetic field used during imaging. While the body tissues undergo low-level distributed heating due to these induced currents, wires that form a part of a conductive pathway having a large enclosed area (and hence cutting many magnetic flux lines) have particularly strong currents induced in them. In addition to heating the wires themselves, the currents also produce localized heating in any tissue that completes an electrical circuit with the wire. As with electrocautery and other radio-frequency sources, capacitive and inductive coupling may allow large currents to flow through pathways that would be considered adequately insulated for prevention of shock from the 60-Hz power line.

Although detailed studies concerning the burn hazard in MRI have not been reported, first principles suggest the following as prudent precautions: 1) Keep all unnecessary conductors out of the bore of the magnet. 2) Place the pulse oximeter sensor as far from the imaging site as possible, e.g., on a toe when the head is being imaged. 3) Do not allow any loops to form in wires or cables running into bore of the magnet, e.g., in those of the electrocardiograph or pulse oximeter. 4) Make a braid of the slack portion of the wires connecting the individual electrocardiograph electrodes to the cable. 5) Use adult-type electrocardiograph electrodes when performing imaging in infants. 9-11 6) Keep a thick layer of thermal insulation between any essential wires or cables and the patient's skin.

The burns reported above may have been avoided if the pulse oximeter sensor had been placed on the toe of the adult patient and if the cable leading to the infant patient's sensor had not been looped or taped directly to the skin. These cases illustrate that in order to give anesthesia safely in the MRI environment, it is essential that the anesthetist become familiar with *all* of the special hazards encountered there.^{3,7}

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REFERENCES

- Shellock FG, Slimp GL: Severe burn of the finger caused by using a pulse oximeter during MR imaging. Am J Roentgenol 153: 1105, 1989
- 2. Kanal E, Shellock FG: Burns associated with clinical MR examinations. Radiology 175:585, 1990
- Kanal E, Shellock FG, Talagala L: Safety considerations in MR imaging. Radiology 176:593-606, 1990
- Roth JL, Nugent M, Gray JE, Julsrud PR, Berquist TH, Sill JC, Kispert DB: Patient monitoring during magnetic resonance imaging. ANESTHESIOLOGY 62:80-83, 1985
- Weston G, Strunin L, Amundson GM: Imaging for anaesthetists: a review of the methods and anesthetic implications of diagnostic imaging techniques. Can Anaesth Soc J 32:552-561, 1985
- Nixon C, Hirsch NP, Ormeod IEC, Johnson G: Nuclear magnetic resonance: its implication for the anaesthetist. Anaesthesia 41: 131–137, 1986
- Messick JM Jr, MacKenzie RA, Nugent M: Anesthesia at remote locations, Anesthesia (3rd edition). Edited by Miller RD. New York, Churchill Livingstone, 1990, pp 2061–2088
- Harper JV: Monitoring in unusual environments, Monitoring in Anesthesia and Critical Care Medicine. 2nd Edition. Edited by Blitt CD. New York, Churchill Livingstone, 1990, pp 685–689
- Finlay B, Couchie D, Boyer L: Electrosurgery burns resulting from use of miniature EKG electrodes. ANESTHESIOLOGY 41:263– 269, 1974
- Hall SV, Malhotra IV, Hedley-Whyte J: Electrosurgery burns (correspondence). ANESTHESIOLOGY 42:641, 1975
- 11. Finlay B, Couchie D, Boyce L, Spencer E, Avey T: Electrosurgery burns (reply). ANESTHESIOLOGY 42:641-642, 1975

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Percutaneous Puncture of the Internal Jugular Vein Using Continuously Transduced Pressure

To the Editor:—Successful percutaneous internal jugular vein cannulation requires considerable skill and experience. Inadvertent arterial puncture is the most frequent complication (0-11%), and placement of a large needle or sheath introducer into the carotid artery may cause serious bleeding or postponement of elective operations, may require surgical intervention, and can be lethal. The rate of complication is inversely related to operator experience. The Raulerson syringe (Arrow International, Reading, PA), permits a one-step modification of the Seldinger technique, in which the guide wire is threaded directly through the syringe and needle. The technique promises to facilitate cannulation with less risk of contamination, trauma, guide wire misplacement, and air embolism, but even use of the Raulerson syringe may still allow insertion of a sheath introducer into an artery.

Many techniques of internal jugular vein catheterization, including real-time ultrasonic guidance, have been described.⁵⁻⁷ In addition to being expensive, this method is not always accessible in most hospitals. In many instances, arterial puncture is recognized by the pressure and color of the blood, both of which may be unreliable signs. Arterial puncture may cause spasm of the vessel, which inhibits pulsatile flow.

When this happens, slow return of blood seems to render color an even more unreliable sign.

We have devised a technique of continuous pressure measurement for attempting to cannulate the internal jugular vein. A T-port extension set (Burron Medical Inc., Bethlehem, PA), with the rubber portion removed is placed in between the needle and syringe. The vein first is identified with the 22-G, 1½-inch finder needle. Then an 18-G, 2¾inch thin-walled needle is connected to a T-port in-line with a 5-ml syringe (fig. 1), and internal jugular vein cannulation is reattempted. As soon as the vein is entered, a transduced central venous pressure is demonstrated on the screen. If present, inadvertent arterial cannulation is immediately evident. Even if arterial spasm is present, the pressure tracing is characteristically much higher than is the venous. The Raulerson syringe can be used with this technique and inadvertent cannulation of the artery can be avoided. However, the straight end of the guide wire should be used because the J end will flex before it passes the T-port. The negative pressure exerted by aspiration of the 5-ml syringe should not affect the calibration of the disposable transducers (Transpac II, Abbott Critical Care System, North Chicago, IL).*