muscle movements; a literature search has failed to record any tonic-clonic seizure due to this agent. Wilder demonstrated activation of temporal lobe epileptic foci by the use of intracarotid and intravenous administration of methohexitol in small doses. Sleep is known to enhance epileptiform activity in psychomotor epilepsy. Therefore, an EEG was performed to exclude this diagnosis.

In this patient the cause of the seizure was hypoglycemia. Over the years, anesthesiologists have commented on the occurrence of hypoglycemia with clinical signs such as lethargy, sweating, pallor, and tremulousness, which accompany the adrenergic response to a rapid decrease in blood glucose concentration. Thomas studied blood glucose levels after induction of anesthesia in two groups of children: one group was starved for as long as eight hours and another group was allowed to drink milk until four hours before anesthesia. The study, using 40 mg/dl as the level for hypoglycemia, showed that 28 per cent of children less than 47 months of age and weighing less than 15.5 kg, who had been starved, were hypoglycemic. There was no patient with hypoglycemia in the group that had been fed until four hours prior to anesthesia. In neither group was there any sign of regurgitation or vomiting. Also found in the study was the fact that none of the children with confirmed hypoglycemia had clinical signs or symptoms of the condition. Of interest is the case report of a 5-year-old girl who underwent adenososillectomy and who convulsed postoperatively. At that time “no glucose was found in the blood.”

Once the diagnosis of a hypoglycemic seizure has been made, or even contemplated, speed is of the essence, as repeated seizures can lead to brain injury. Studies of paralyzed animals subjected to repeated seizures have demonstrated that a point is reached when the compensatory factors that increase substrate supply to the convulsing brain cannot compensate, leading to a decrease in ATP. When a pediatric patient has a seizure during or after anesthesia a sample of blood for glucose determination should be obtained and an intravenous infusion of glucose started. The use of a Dextrostix is invaluable for an immediate and relatively accurate estimation. When the patient is suspected to be hypoglycemic, dextrose, 25 per cent, 2–4 ml/kg (0.5–1.0 mg/kg), is given intravenously. Thereafter, one should maintain dextrose infusion at a rate of 0.5 g/kg/hr until the child can maintain an adequate blood glucose value. If, after treatment, one is still unsure of the diagnosis, a full evaluation, including measurements of blood levels of calcium, magnesium, and ketone, lumbar puncture, and EEG, should be performed.

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V-Lead Adapter
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Tektronix® Models 408, 412, and 414 patient monitoring oscilloscopes were designed to record from conventional limb leads of the electrocardiogram (ECG). One can record a precordial ECG with these monitors by one of several methods that include: 1) a Tektronix 408 or 412 with modification 735D, or type 414 with option 4; 2) modified limb-lead placement; 3) a V-lead adapter (013-0180-01) recently introduced by Tektronix. The modified oscilloscopes have full-lead selectors and will display precordial and limb-lead ECGs. However, one loses the option to record from limb leads when the modified limb-lead and V-lead adapter methods are used.

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Fig. 1. V/limb-lead adapter. Four-pole, double-throw (4PDT) toggle switch is used to select either a V or limb leads for display. Lead-selector switch on the monitor must be in the II position and toggle switch in the V position to display the precordial lead. Resistors are 3/4 watt carbon, ±10 per cent tolerance. R1 = 5.6 kΩ, R2 = 1.8 kΩ. The ECG cable and patient leads connect to the chassis (shaded arm) via pin plugs and jacks, respectively.

Fig. 2. V/limb-lead adapter. The V lead is recorded when 4PDT switch is in the V position illustrated and lead I is selected on the monitor.

This paper describes two adapters for use with Tektronix 408, 412, 414, and similar monitors of other manufacture to display either a precordial or any one of three limb-lead ECGs. Either adapter is readily interposed between patient leads and ECG cable without modification of leads, cable, or oscilloscope. The adapters do not contain batteries or any other active devices, and electrical isolation between oscilloscope and patient is not adversely affected when either adapter is used.

Figure 1 is the circuit schematic of the simpler arrangement. Four electrodes are placed on the patient’s right arm (RA), left arm (LA), left flank (LL), and precordium (V). When the toggle switch is placed in the limb position one can display lead I, lead II, or lead III via front-panel selector switch on the monitor. One can record from the V lead by selecting lead II on the monitor and the V position of the toggle switch.

The junction of the R1 resistors is not a true central or isoelectric terminal because the circuit common from the input difference amplifier in the monitor is returned to one of the three electrodes (LA) that are used to form the central terminal. The circuit common is normally floating and is isolated from chassis ground in Tektronix monitors. Thus, differences between true V-lead recordings and recordings obtained with this adapter in use with a high-input-impedance monitor, such as the Tektronix 400 series, usually will not be significant to the anesthesiologist.

The adapter shown schematically in figure 2 overcomes this potential limitation when monitors with other input configurations are used by adding a fifth patient electrode to either right or left flank (RL), and by returning the circuit common to electrode (II) when the V lead is recorded.

Resistance values are not critical. Satisfactory recordings are obtained when values for R1 range between 5 kΩ and 100 kΩ; however, all values for R1 should be equal in any one adapter. Resistor R2 is included to minimize 60-Hz and other common-mode inter-
Pseudoaneurysm, a Late Complication of Radial-Artery Catheterization

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The most common complications of radial arterial catheterization are thrombus, embolus and hematoma formation, usually detected prior to or shortly after decannulation. We report the uncommon occurrence of pseudoaneurysm formation 18 days after decannulation in a patient who had an otherwise uncomplicated hospital course.

REPORT OF A CASE

A 68-year-old man was admitted to the hospital with a six-week history of nausea, vomiting, and weight loss. Gastrointestinal roentgenograms revealed a mass in the right upper quadrant. His medical history included sixty pack-years of cigarette smoking, with chronic bronchitis, and claudication, for which a right lumbar sympathectomy had been performed two years prior to admission. Physical examination disclosed no abnormality except an increased anteroposterior chest diameter and mild inspiratory wheezes. Allen’s test for adequacy of ulnar collateral vessel filling was normal at 3 sec. Results of routine laboratory tests and esophagogastroduodenoscopy were within normal limits. Roentgenograms of the chest and pulmonary function tests were consistent with mild chronic obstructive pulmonary disease. An exploratory laparotomy with general anesthesia was scheduled.

Prior to induction of anesthesia a #20 Long-Dwel†‡ catheter was placed percutaneously in the left radial artery on the first attempt, without difficulty. The vessel was not transfixed during this procedure. Physiologic saline solution with 2 units of heparin/ml was infused continuously through the catheter at a rate of 3 ml/hour by an Intraflow§ system. The left arm was extended at the shoulder and flexed at the elbow without tension on the brachial plexus. It remained under direct observation throughout the operation. There was no evidence of compromised perfusion. Anesthesia and operation were without incident. A Whipple procedure was performed for adenocarcinoma of the pancreatic duct. Postoperatively, the patient was observed in the intensive care unit, where his condition remained hemodynamically stable, with good peripheral perfusion.

Twenty-four hours after operation the arterial catheter was removed, and manual pressure applied for 5 min. Perfusion remained normal and unchanged, with full pulses, normal Allen’s tests, and no hematoma formation. There were no arterial punctures made after decannulation. There was no evidence of complication until the eighteenth postoperative day, when the patient was noticed to have a 1.5 x 1.5-cm painless pulsatile mass over the site of cannulation. No thrill was detected. Peripheral perfusion remained good. Over the following week the mass was unchanged, and the patient was discharged on the twenty-third postoperative day. However, on the thirtieth postoperative day the patient was readmitted because the mass had enlarged to 3 x 3 cm. A thrill and bruit were noticed; peripheral perfusion was unimpaired.

Surgical exploration revealed a pseudoaneurysm 3 x 3.5 cm in dimensions at the site of arterial puncture in the left radial artery (fig. 1). The left radial artery was ligated proximally without compromising perfusion of the hand.

DISCUSSION

The most common complications associated with radial arterial catheterization are transient thrombosis (incidences as high as 88 per cent), occlusion after cannulation (to 60 per cent), and hematoma formation (to 40 per cent). These complications generally occur early in the cannulation-decannulation course. Late complications, such as pseudoaneurysm, have been suggested as possibilities, but have not been documented. Data are available, however, for axillary and femoral arterial pseudoaneurysms associated with...