CORRESPONDENCE

resulted in an obvious subarachnoid puncture. A second successful
atraumatic attempt was made at T10–11. With the Tuohy needle
bevel oriented cephalad, another Arrow FlexTip Plus catheter was
threaded without resistance to about 15 cm at the skin and secured.
Aspiration of the catheter yielded no fluid. A test dose of 5 ml of 2%
lidocaine with 1:200,000 epinephrine was injected slowly producing
a gradual decline in blood pressure (BP) from 124/82 to 95/47 mmHg
and moderate resolution of her pain. A detailed neurologic examina-
tion was not performed. Our Acute Pain Service (APS) began an
epidural infusion of 0.125% bupivacaine plus hydromorphone 6 mg/
ml at 14 ml/h. Approximately 2.5 h later the patient was transferred
to a “step-down” unit for further observation. At this time the patient
was comfortable with a sensory deficit to cold from T1 to L1, coughing
effectively, and performing deep-breathing maneuvers easily. She did
not report any extraordinary sensations of numbness or demonstrate
any muscle weakness.

Our APS examined the patient the next morning and found her to be
alert and oriented with motor and sensation to light touch intact.
Except for mild pruritus and nausea controlled with intravenous pro-
metrazine 6.25 mg, she was comfortable. When examined that after-
noon there was no change in her condition. Sometime that evening
the patient’s husband recalled that she was more somnolent and
seemed weaker. At about 0200 the next day, the nurse found the
patient barely responsive with a respiratory rate of 6 breath/min. She
was not moving her extremities. Her O2 saturation was 86%, and her
BP was 80/50 mmHg with a heart rate of 78 beats/min. Her pupils
were 2+ and reacted sluggishly. The epidural infusion was stopped
and 100% O2 by mask, 11 of crystalloid and intravenous naloxone
0.4 mg was administered by the nursing staff. The patient quickly
aroused, and her sensorium cleared. Her upper and lower extremities
were paralyzed. Sensation to pinprick was absent from the C6, derma-
tome and below. Aspiration of the epidural catheter did not return
any fluid until it was withdrawn 3–4 cm whereupon a few ml of clear
fluid was obtained. Unfortunately the fluid was discarded without
analyzing for CSF. The catheter was removed before radiographic
determination of its true position could be obtained. Appropriate
concentrations of drug in the remaining epidural infusion were verified
by our pharmacy. Over the next several hours, the patient’s strength
and sensation recovered in a manner consistent with the recession
of a local anesthetic-induced block. She never complained of a head-
ache. The patient was discharged from the hospital without any
sequelae.

The final position of our catheter was never determined with cer-
tainty because neither CSF was aspirated nor radiographic evidence
obtained. However, on clinical grounds, it is difficult to explain the
transition of a purely analgesic state to one marked by quadriplegia
and depressed consciousness without proposing subarachnoid depo-
sition of our 0.125% bupivacaine·6 mg·ml−1 hydromorphone infa-
sate. It is generally appreciated that, theoretically, any epidural cathe-
ter can penetrate the dura. The new catheter by Arrow does not appear
to be an exception in spite of its flexibility.

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Detection of Heparin in Salvaged Blood

To the Editor.—Autotransfusion devices or “Cell-Savers” are fre-
cently used after termination of cardiopulmonary bypass (CPB) to
process blood remaining in the extracorporeal circuit. Although the
packed RBC product produced by these devices contain insignificant
amounts of heparin, many anesthesiologists still administer addi-
tional protamine to reverse heparin that may remain in “cell-saver”
blood, a practice that could potentially lead to excess protamine
administration.

The Hepton®/LIMS (Medtronic, Inc., Blood Management Business,
Parko, CO) performs a variety of functions related to anticoagulation
management during cardiovascular surgery, including ACT and a hepar-
in assay.1 The latter is performed by heparin/protamine titration, in
which blood is automatically dispensed to individual channels
containing thromboplastin reagent and different concentrations of
protamine. After mixing of these components in a reaction chamber,
termed “run time,” the first channel in which clot formation is detec-
ted is then used to calculate the heparin concentration. If the run
time exceeds 249 s, the test is not considered valid because depletion
of coagulation factors (or rarely, deterioration of cartridge reagents)
may have occurred.

Some anesthesiologists have tried to use the Hepton®/LIMS to
determine the presence of heparin in “cell-saver” blood. Unfortu-
nately, assays for heparin that require the presence of either coagula-
tion proteins or antithrombin III should not work on “cell-saver”
blood because washing also removes these components. A recent
experiment confirmed this.

Blood remaining in the extracorporeal circuit after termination of
CPB was processed by an autotransfusion device (BRAT®, Cobe

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Laboratories, Arvada, CO) using a 1:1 wash cycle. Two independent blood samples from different devices were analyzed for heparin using a 4 channel Heparin Assay "Yellow" cartridge (test range: 0.0 - 1.5 mg/kg) with simultaneous measurement of ACT. A test concentration of 0.0 mg/kg was obtained in both samples. However, examination of individual channel times in the Hepcon®/HMS cartridge, three out of four recorded channel run times exceeded 400 s. In addition, blood failed to coagulate in ACT channels, exceeding 600 s.

Although the Hepcon®/HMS reported the absence of heparin in both "cell-saver" blood samples, this result was inaccurate. In addition, no warning system existed on the display screen to advise the user that channel run times exceeding 249 s indicated depletion of coagulation factors.

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References


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In Reply: — Dr. Loubser is correct in stating that HPT results with run times longer than 249 s are considered to be invalid in the Hepcon® HMS. However, the most common cause for a prolonged run time is inappropriate cartridge selection as opposed to depletion of coagulation factors. Our Heparin Protamine Titration (HPT) is considered to be a quantitative test and performs very reliably even in cases where the fibrinogen is diluted or depleted to levels considered to be physiologically low (e.g., 50 mg/dl). The HPT will typically perform within specification when patients are bleeding due to coagulopathy and is not considered to be an indicator of coagulopathy, only heparin concentration.

The HMS will provide a warning if the test has exceeded 249 s. Because the design of the HPT is for use with fresh whole blood (not components or citrated samples), any follow-up action recommended assumes the use of a fresh whole blood sample. The HPT cartridge can measure a limited range of possible heparin concentrations. If a user selects the incorrect cartridge range, a channel will usually detect, but with a long run time. This is an indication to repeat the test with another test range. This is rarely an indication of coagulation factor depletion, therefore we do not see it as appropriate to indicate such a state in a warning.

Studies from our Sequestra® and AT 1000® devices indicate that cell washing generally can remove 90% or more of the residual heparin, plasma, and associated factors from blood, rendering the sample essentially fibrinogen free. This type of sample is not within the scope for which our HPT test was developed.

When asked if the HPT will detect heparin in a washed cell pack, we recommend specific methods that provide adequate fibrinogen to obtain a reliable result. One method suggests measuring residual heparin in the patient's blood after the washed product is returned. A second method utilizes PRP or PPP to provide clotting factors for measuring heparin in a cartridge such as our Theracon HPT. Theracon HPTs can measure very low levels of heparin and can be used with a citrated sample.

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2. In house data, Medtronic Blood Management.

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