TABLE 1. Changes in BUN, Creatinine, and Potassium*

| | BUN (mg/100 ml) | Creatinine (mg/100 ml) | Potassium (mEq/l) | Change from Initial Potassium (mEq/l) | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | 3 Min | 5 Min | 7 Min | 10 Min | 15 Min |
| Control group | 24.4 ± 0.35 | 1.16 ± 0.07 | 3.32 ± 0.17 | 0.46 ± 0.12 | 0.51 ± 0.11 | 0.66 ± 0.19 | 0.60 ± 0.14 | 0.72 ± 0.12 |
| Uremic group | 182.4† ± 12.40 | 12.12† ± 0.63 | 6.28† ± 0.32 | 0.30‡ ±0.09 | 0.68‡ ± 0.17 | 0.46‡ ± 0.13 | 0.72‡ ± 0.12 | 0.48‡ ± 0.07 |

* Mean ± SE.
† Significant at $P < 0.01$.
‡ Nonsignificant at $P < 0.05$.

to above control levels in both "normal" and uremic monkeys. At no time was there a significant difference between changes of potassium levels in the two groups.

## DISCUSSION

The mean changes in potassium in the two groups are comparable to those seen in normal man during nitrous oxide–oxygen–halothane anesthesia.[7] Like Powell and Colby, who studied rats, we saw no significant change in potassium levels after administration of succinylcholine to control and uremic animals.[6] Unlike Powell and Colby, we did not see much variation in the potassium responses in our uremic group. Our data clearly show that the increase of potassium after succinylcholine is no greater in acutely hyperkalemic uremic monkeys than in control monkeys. Whether potassium changes after succinylcholine may be greater in the chronically uremic monkeys following some latent period remains to be determined. Such work is hampered by a high death rate in undialyzed animals.

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# Severe Hypotension during Prosthetic Hip Surgery with Acrylic Bone Cement

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The use of methylmethacrylate, a self-curing cement, is revolutionizing orthopedic joint surgery. The FDA has recently released this product, and its use to anchor the components in major joint replacement is gaining popularity. Transient hypotension coincident with its insertion into the shaft of the femur has been reported,[1] and is mentioned in the package insert accompanying the product. In recent months several reports of cardiovascular

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collapse associated with the use of acrylic cement in hip replacement have been published.[2-4] We recently had such a case, in which the patient developed severe hypotension.

## Report of a Case

A 76-year-old woman was scheduled for prosthetic replacement of the right femoral head, having suffered an intracapsular fracture the previous day. Physical examination disclosed no abnormalities except marked obesity (weight 230 pounds) and mild hypertension, for which the patient periodically took chlorothiazide. A preoperative chest x-ray was normal, with some evidence of atherosclerotic changes in the thoracic aorta. ECG showed sinus rhythm with left axis deviation and nondiagnostic S-T changes. Hematocrit was 42 per cent. Blood pressure was 146/92 torr. The patient was premedicated with hydroxyzine (Vistaril), 50 mg and atropine, 0.6 mg.

Anesthesia was induced with 100 mg methohexital (Brevital). After administration of 6 mg *d*-tubocurarine and 80 mg succinylcholine, the trachea was intubated. Anesthesia was maintained with halothane 0.5–1.5 per cent, nitrous oxide, 2 l/min, oxygen 2 l/min in a semiclosed system with $CO_2$ absorber. Respirations were assisted. The patient was placed in the left lateral decubitus position and the operation started. The anesthetic course was uneventful for 75 minutes, during which time blood pressure had stabilized at 110/60 torr, with a pulse rate of 88/min. The patient had received 1,400 ml of 5 per cent dextrose in Ringer's lactate solution. Blood loss had been less than 200 ml.

As methylmethacrylate cement was being placed into the femoral shaft, pulse and blood pressure suddenly became unobtainable. Two 15-mg doses of mephentermine (Wyamine) failed to restore the pulse or blood pressure. The patient became apneic and cyanotic. She was hyperventilated with 100 per cent oxygen and turned for closed-chest cardiac compression. Sodium bicarbonate, 88 mEq, and 1 mg epinephrine were given iv. A cardioscope then showed ventricular tachycardia; blood pressure was not yet detectable. Sinus rhythm was restored with 100 mg lidocaine, at which time the pulse was 120/min, with a blood pressure of 160/80 torr. The operation was completed 15 minutes later utilizing oxygen and 0.25 per cent halothane, intermittent succinylcholine 0.1 per cent drip, and controlled respiration.

The patient regained reflexes and moved promptly, but remained depressed, necessitating reintubation and respiratory assistance because of low tidal volume, bilateral respiratory rhonchi, and hypotension. ECG showed T-wave changes anteriorly, consistent with ischemia or pulmonary embolus. A chest x-ray showed pulmonary vascular congestion. Electrolytes were normal. Central venous pressure ranged from 13 to 18 cm $H_2O$. As tidal volume improved, the orotracheal tube was removed. With the patient breathing 8 l/min oxygen spontaneously via nasal catheter, $Paco_2$ was 39 torr, $Pao_2$ 54 torr, and pH 7.34. Sinus rhythm persisted except for a few atrial ectopic beats. No myocardial infarct pattern developed.

The patient's condition improved during a seven-day period, and chest x-rays and ECG changes gradually returned toward normal. Increased inspired oxygen tension was necessary because of persistent subnormal $Pao_2$. Both LDH and SGOT were moderately elevated. No fat appeared in the urine. Supportive care was continued until the patient was able to eat and ambulate with assistance.

## Discussion

Self-curing acrylic cement is packaged as a powder and a volatile liquid which, when mixed, form a dough that sets hard in an exothermic reaction in 5 to 10 minutes. The powder is polymerized methylmethacrylate containing an activator, benzoyl peroxide. The liquid is methylmethacrylate monomer with 2 per cent dimethylparatoluidine, an "initiator." When liquid and powder are mixed, the liquid polymerizes.[1] The heat of polymerization reached is between 80 and 96 C.[4]

The cause of sudden hypotension in this patient remains obscure, but her history closely resembles the course of events reported by others immediately following femoral placement of acrylic cement.[2-5] Phillips *et al.*[5] have observed that blood pressure usually decreases 12 to 90 seconds after placement of cement into the proximal femur, but rarely after acetabular implantation. Perhaps the surface area and vascularity of the acetabulum are less than in the femur, where increased absorption of the monomer may lead to hypotension. Powell *et al.*,[3] who reported two similar cases of acute hypotension and cardiac arrest, proposed that older, less healthy patients who have pre-existing cardiovascular disease or hypovolemia are more likely to develop this complication than others.

Several other causes have been proposed: The hypotension could be secondary to toxic cardiovascular effects of the methylmethacrylate monomer or its additives when directly absorbed into the circulation.[6] Toxic effects of the monomer may acutely augment the depressant cardiovascular action of volatile halogenated anesthetics. The increase in intramedullary pressure that follows introduction of hot acrylic cement may force fat and marrow into the circulation, resulting in embolization

to the lungs.[2] Reflex bradycardia with fall in blood pressure is another possibility, but was not observed in this patient or in other reported cases. Still other possibilities include the direct effect of cement temperature, air or gas embolism, or sensitivity to the acrylic monomer. Further study is warranted.

That transient hypotension frequently occurs and cardiovascular collapse is not rare must be considered by the anesthesiologist when planning anesthesia for prosthetic joint surgery with acrylic cement. Appropriate monitoring devices are indicated, as well as the ready availability of resuscitative drugs and equipment. Certainly blood pressure should be measured frequently after the cement is introduced.


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## Maintaining Optimum Audibility of Korotkoff Sounds during Blood-pressure Monitoring

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Every clinical anesthesiologist uses some type of apparatus to monitor Korotkoff sounds for blood-pressure measurement. Usually the stethoscope receiver is placed over the artery in the antecubital space, beneath the sphygmomanometer cuff.

The mechanism of the generation of Korotkoff sounds is unknown.[1] In fact, 95 per cent of the information available from Korotkoff sounds comes from frequencies below the range normally audible.[2] High-frequency components of Korotkoff sounds have minute amplitudes which provide faithful reproduction of the auscultatory sounds at frequencies of approximately 20 to 300 Hz.[3,4] The presence of shock can diminish or even eliminate the Korotkoff sounds, making blood pressure reading unreliable.[5] The human ear, with a range of 16 to 20,000 Hz, is less sensitive to frequency variations at the lower end of the auscultatory frequency band, but detects frequency changes more readily than intensity changes.[6]

One of the authors noticed a decrease in threshold sensitivity during blood-pressure monitoring while using a custom-fitted monaural earpiece. In the presence of tympanic membrane inflammation due to a viral infection, inflation of the blood-pressure cuff caused pain in the ear. The purpose of this study was to: 1) determine the pressure that could be developed in the external auditory meatus with inflation of the blood-pressure cuff; 2) determine the extent of hearing loss in volunteers that resulted from the increase in external auditory meatus pressure; 3) devise a

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