instituted. The comments regarding hypothermia are appreciated and thought to be addressed on page 2931 in the section that deals with hypothermia.

Dr Koufman is appropriately concerned with complications of long-term endotracheal intubation. The tube sizes recommended have been reduced when compared with the previous standards but are larger than he would like in order to permit suctioning and appropriate ventilation during the acute phases of cardiac arrest and resuscitation. It is hoped that the victim will be immediately resuscitated, extubated, and therefore not subjected to the complications of prolonged intubation to which Dr Koufman eludes. These sizes of tubes are routinely used during anesthesia and, as long as they are Z 79 implant-tested tubes, and not reused, there should be no problem with stenosis.

> William H. Montgomery, MD Straub Clinic and Hospital Honolulu

1. CORRECTION. JAMA 1986;256:1727.

 Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 1980:244:453-509.

To the Editor.—In reading the "Standards and Guidelines for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care (ECC)," I have found two points that were particularly disturbing and would like to bring them to your attention.

In the guidelines it discusses cough CPR and states that "self-induced CPR is possible; however, its applications are limited to clinical situations in which the patient has a cardiac monitor." I feel this statement seriously limits the lifesaving potential of this technique. As the quality assurance director of the Northeast Georgia Medical Services Council and instructor in our training programs for advanced emergency medical technicians (EMTs), I am aware of several medical instances wherein cough CPR was effective in maintaining consciousness in arrested patients in their homes while EMTs were responding to the call. None had cardiac monitoring being performed at the time. In these instances the EMT answering the call for help (where an oncoming heart attack was suspected) instructed the caller to have the victim begin coughing forcefully and persistently if the victim began sensing a loss of consciousness. Upon arrival of the EMTs and the beginning of electrocardiographic monitoring the victims were in ventricular fibrillation yet were conscious.

The second point I found disturbing

is concerned with the section on the management of obstruction of the airways by foreign bodies. The guidelines failed to mention the use of gravity as a helpful aid in the dislodging of airway foreign bodies, particularly in infants and small children. From personal experience and that of EMTs in our region, I have become convinced that taking the advantage of the use of the force of gravity can be an important adjunct in the dislodging of foreign objects. Tipping a small child into a 45° Trendelenburg position or even further and continuing the Heimlich maneuver or chest blows has been effective in dislodging foreign bodies when these maneuvers were not effective with the victim upright or supine. Inverting dogs and cats with airway foreign bodies has also been effective in expelling the objects when success was not observed in an upright position or horizontal recumbency.

> Dennis T. Crowe, DVM, DACVS The University of Georgia College of Veterinary Medicine Athens

 Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 1998-955-9005.

#### 'Hello? Hello? . . . He Doesn't Seem to Answer'

To the Editor.—In reference to the cover of the June 6 issue, you describe "[a paramedic] who is holding two defibrillation paddles." If this is in fact the case, the paramedic is about to defibrillate his right ear—not a safe practice. However, if you look more closely, you will see that the paramedic is in fact holding a telephone.

Stephen R. Gorfine, MD New York

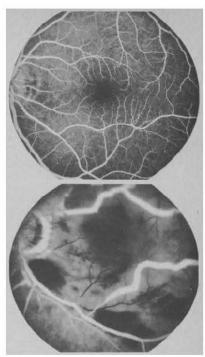
1. JAMA 1986;255:2843.

# Acute Ischemic Retinopathy due to Gentamicin Injection

To the Editor.—We recently reviewed a number of cases in which gentamicin was inadvertently injected into an eye and caused blindness. The clinical picture of acute ischemic gentamicin toxicity of the retina and optic nerve is a remarkable one (Figure). The entire retina becomes ischemic and necrotic. [15]

In the cases seen, gentamicin was either mistaken for balanced salt solution, or some other medication, and injected into the eye or was injected in such a way that a high concentration of it came in contact with the retina.

Near the end of intraocular surgery, eye surgeons may inject balanced salt solution or some other physiological solution into the eye in order to regain intraocular pressure. In addition, at



Top, Normal retina: fluorescein angiogram, left macula. Note normal, patent, branching retinal vessels. Vision, 20/20. Bottom, Acute occlusive gentamicin toxicity of the retina: fluorescein angiogram, left macula. Note massive nonperfusion (vascular occlusion) of retina. Remaining few vessels leak fluorescein dye. Hemorrhages are also present. Vision, perception of light only.

the end of certain eye operations, gentamicin is often injected under the conjunctiva, prophylactically. If the gentamicin is drawn up early in the case and left in an unlabeled syringe, it can be mistaken for balanced salt or for some other medication and inadvertently injected.

To avoid the problem of inadvertent gentamicin injection, the physician should never inject into a patient anything that is not labeled. The only time a physician should accept a syringe full of medication is when he himself draws the medication out of the bottle or sees it drawn up, then uses it immediately. Nurses should never draw up medications and leave them unlabeled. If a medication must be drawn up in advance, it should be drawn up into a labeled syringe.

Howard Schatz, MD H. Richard McDonald, MD San Francisco

 Conway BP, Campochiaro PA: Macular infarction after endophthalmitis treated with vitrectomy and intravitreal gentamicin. Arch Ophthalmol 1986;104:367.
 McDonald HR, Schatz H, Allen AW, Chenoweth JD:

 McDonald HR, Schatz H, Allen AW, Chenoweth JD: Retinal toxicity secondary to intraocular gentamicin injection. Ophthalmology, in press.

 Snider JD, Cohen HB, Chenoweth RG: Acute ischemic retinopathy secondary to intraocular injection of gentamicin, in Ryan SJ, Dawson AK, Little HL: Retinal Diseases

JAMA, Oct 3, 1986-Vol 256, No. 13

Letters 1725



w York, Grune & Stratton, 1985, pp 227-232. 4. Conway BP, Campochiaro PA: Macular infarction after endophthalmitis treated with vitrectomy and intravitreal gentamicin. Arch Ophthalmol 1986;104:367.

### Hyperkalemia due to Salt **Substitutes**

To the Editor.—Near-fatal hyperkalemia from potassium supplements and salt substitutes continues to be an important clinical problem.1

Although its incidence is unknown, it seems more likely to occur in azotemic patients, diabetics, or patients using drugs that affect potassium homeostasis (β-blockers, nonsteroidal anti-inflammatory drugs, captopril, spironolactone, triamterene, and amiloride). Salt substitutes may be prescribed to reduce sodium or increase potassium intake but are also selfprescribed by an unknown number of patients. We describe a patient who experienced near-fatal hyperkalemia caused by repeated ingestion of soup heavily "seasoned" with a salt substi-

Report of a Case.—A 70-year-old woman was admitted to a local hospital for transient severe muscle weakness following dinner. She became so weak she could not stand and noted paresthesias of her hands and mild dyspnea. She denied chest pain or diaphoresis. On arrival to the emergency room she was asymptomatic. She had known angina pectoris, which was well controlled with diltiazem hydrochloride, 60 mg four times per day, metoprolol, 50 mg four times per day, and isosorbide dinitrate, 40 mg four times per day. Her blood pressure was 110/50 mm Hg supine, 98/60 mm Hg standing. Her pulse rate was 90 beats per minute, and her temperature was 37°C. The remainder of her physical examination results were unremarkable. Laboratory values were normal with the exception of the electrolytes: sodium, 145 mEq/L (145 mmol/L); chloride, 113 mEq/L (113 mmol/L); potassium, 9.1 mEq/L (9.1 mmol/L); bicarbonate, 29 mEq/L (29 mmol/L); serum urea nitrogen, 23 mg/dL (8.2 mmol per liter of urea); and creatinine, 1.2 mg/dL (106 mmol/L). The electrocardiogram demonstrated very peaked T waves in V2, V3, and V4, a QRS interval of 0.08 s, and a PR interval of

Potassium Content of Salt Substitutes

	mEq/Teaspoon
Morton's Lite Salt	38
Morton's Salt Substitute	72
No-Salt	64
Nu-Sait	67
Adolph's Salt Substitute (unseasoned)	65

0.21 s. A repeated potassium level drawn with a large-bore needle and syringe without a tourniquet was 7.4 mEq/L (7.4 mmol/L). The hyperkalemia was treated successfully with intravenous calcium chloride, bicarbonate, and sodium polystyrene sulfonate. Her peaked T waves reverted to normal and her potassium level at discharge had returned to 4.7 mEq/L (4.7 mmol/L). The patient denied use of potassium supplements but was unaware of the potassium content in her salt substitute. She remembered consuming large quantities of a homemade soup twice on the day of admission. Analysis of her soup revealed its potassium concentration to be 94 mEq/L (94 mmol/ L). Subsequent serial serum potassium levels measured since she has been an outpatient have been normal.

Comment.—A recent trip to a local grocery store confirmed that the labels of the leading salt substitutes now contain warnings about use without the advice of a physician. They do not, however, warn against use in cooking or state a safe maximum daily dose, and, in fact, they have the sort of wide-bore openings useful for cooking. The potassium content per teaspoon for several brands is noted in the Table.

Several points should be made concerning this case. (1) Potassium in all forms may be overly prescribed by physicians and overly consumed by the public.4 (2) Severe muscle weakness can result from both hypokalemia and hyperkalemia.<sup>5</sup> (3) A patient could present with a cardiac arrest due to hyperkalemia associated with an agonal-appearing electrocardiogram and the cause could be easily missed. Hyperkalemia in our patient was most likely due to excessive intake of potassium in the form of a salt substitute. (4) Although  $\beta$ -blockers can clearly alter potassium distribution between intracellular and extracellular compartments, the effect is specific for  $\beta_2$ -blockers.<sup>6</sup> Our patient was using metroprolol, a selective  $\beta_1$ -blocker. (5) It seems likely that our patient's hyperkalemia was adversely affected by diltiazem, as suggested by recent studies.7-9 Thus, calcium blockers should probably be added to the list of drugs that affect potassium homeostasis.

The public needs to be more aware of the potential danger of salt substitutes. Physicians should routinely inquire as to their use as they would other drugs in the counseling of their patients.

> Robert E. Hovt. MD Kilmarnock, Va

1. Snyder EL, Dixon T: Abuse of a salt substitute. N Engl

McCaughan D: Hazards of nonprescription potassium supplements. Lancet 1984;1:513-514.

3. Alvo M, Warnock DG: Hyperkalemia: Medical staff conference, University of California, San Francisco. West J Med 1984:141:666-671.

4. Kassirer JP, Harrington JT: Fending off the potassium

pushers. N Engl J Med 1985;312:785-787.
5. Adams RD, Victor M: Principles of Neurology, ed 3.

New York, McGraw-Hill International Book Co, 19 6. Ponce PS, Jennings AE, Madias NE, et al: Drug-induced

hyperkalemia. *Medicine* 1985;64:357-370.

7. Johnson IME, McDougall JG, Coghlan TP, et al: Potassium stimulation of aldosterone secretion in vivo is reversed by nisoldipine, a calcium transport antagonist. Endocrinology 1984;114:1466-1467.

8. Kelleher S, Gillum D: Increased serum K due to combined calcium channel and beta-adrenergic blockade.

Proc Am Soc Nephrol 1984;17:44A.

9. Nugent M, Tinker JH: Cardiovascular effects of verapamil during acute hyperkalemia and after calcium therapy. Anesthesiology 1982;57:A3.

## Keeping in Trim: **Nailed Doc Docks Nails**

To the Editor.—Recently, I had an elderly patient in the hospital whose toenails had been neglected and uncut for a long period of time. Because I noticed this and because the patient asked me to, I requested that the nurses take care of this. I was informed that nurses no longer cut toenails! There is no podiatrist on the hospital staff. As an orthopedic consult was pending I hoped one of the residents would undertake the nail trimming when examining her deformed foot. Alas! Since the patient chose not to have surgery on her foot, the nails remained uncut.

At this stage I accepted the concept that in a hospitalized patient, trimming of the toenails is part of primary patient care and I borrowed a nail trimmer from the nursing staff (who were delighted to loan it to me) and promptly attended to it, to everyone's delight.

> Bala V. Manyam, MD Southern Illinois University School of Medicine Springfield

## **Ethylene Oxide and Cancer**

To the Editor.—In their March 28, 1986, article, "Epidemiologic Support for Ethylene Oxide as a Cancer-Causing Agent," Hogstedt et al1 assert that there is a strong indication that ethylene oxide is a carcinogen even at low-level exposures. The evidence presented in the article to substantiate this claim is very weak and certainly lends no credence to the authors' theory.

The major piece of supporting evidence for this claim appears to be the results of the study at plant 3, where there was one leukemia death vs 0.16 expected. The single case of leukemia for plant 3 occurred in group C, where workers had multiple chemical exposures and the lowest ethylene oxide exposures. What does stand out as

JAMA, Oct 3, 1986-Vol 256, No. 13

Letters

