

2. Ottosson JO: Effect of lidocaine on the seizure discharge in electroconvulsive therapy. *Acta Psychiatr Scand* 35:7, 1960.

3. Essman WB: Anticonvulsive properties of xylocaine in mice susceptible to audiogenic seizures. *Arch Int Pharmacodyn Ther* 164:376-386, 1966.

***Aeromonas hydrophilia* Septicemia in a Previously Healthy Man**

To the Editor.—Ramsay et al (239:128, 1978) recently reported a case of *Aeromonas hydrophilia* sepsis in a patient undergoing hemodialysis therapy. We report a case of *A hydrophilia* septicemia seen at Walter Reed Army Medical Center.

Report of a Case.—A 79-year-old man with a history of heart disease, but otherwise in good health, was seen in the emergency room for two episodes of dull epigastric pain associated with nausea during a 24-hour period. The patient was admitted for possible myocardial infarction. Cardiac enzyme levels and ECGs showed no evidence of myocardial infarction, but on the second hospital day the patient had a temperature of 39 °C. Four blood cultures grew *A hydrophilia*. The strain was sensitive to gentamicin, amikacin, and tobramycin and resistant to ampicillin, cephalothin, and carbenicillin. Stool culture subsequently grew *A hydrophilia*. He received a ten-day course of gentamicin sulfate, with resolution of epigastric pain and fever.

Comment.—*Aeromonas hydrophilia* is a Gram-negative anaerobic rod found in natural water sources and as an occasional inhabitant of the normal gastrointestinal tract. It is a rare cause of septicemia and usually is seen only in immunorepressed or debilitated patients.¹ Our patient represents a case of *A hydrophilia* sepsis in an otherwise immunologically intact host.

FREDERIC D'ALAURO
RICHARD ANSINELLI, MD
Walter Reed Army Medical Center
Georgetown University
School of Medicine
Washington, DC

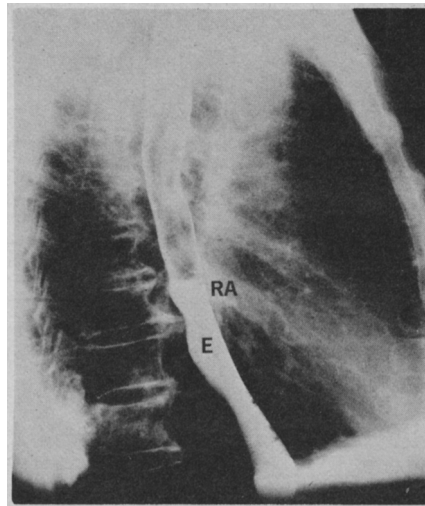
1. Bulger RJ, Sherris JC: The clinical significance of *Aeromonas hydrophilia*. *Arch Intern Med* 118:562-564, 1966.

See also *JAMA* 239:1742, 1978.—ED.

Dysphagia From Thoracic Ankylotic Spur

To the Editor.—I have seen a patient with dysphagia from an ankylotic thoracic spur. The esophagus was impinged between the spur and the heart.

Report of a Case.—An 80-year-old man has been careful about his health all his life and had been known to have ankylosing spondylitis since he was much younger. He exercises daily and takes vita-



Barium swallow done April 3, 1978. Huge ankylotic spur at D8-9 causing impingement of esophagus (E) between spur and right atrium (RA) of patient's heart.

mins. About three months previously he noted that one of his large vitamin capsules became lodged in his esophagus; it took a long time for the capsule to pass into his stomach. The vitamin became progressively harder to swallow. A barium swallow showed impingement of the esophagus between a huge ankylotic spur at D8-9 and the right atrium of the heart (Figure). The patient also has ankylosis in the cervical spine and matches the HLA-B27 histocompatibility antigen. The patient does not wish osteophytectomy.

MICHAEL R. RASK, MD
Sahara Ranch Medical Center
Las Vegas

The Nutritionally Unsound 'Nutritional and Metabolic Antineoplastic Diet' of Laetrile Proponents

To the Editor.—In *Laetrile Case Histories*, Richardson and Griffin¹ delineate the "nutritional and metabolic antineoplastic diet" of laetrile proponents. The diet calls for the following:

1. No meat, fish, or fowl. These are the major sources of absorbable iron in the American diet. Their lack results in a much higher frequency of iron deficiency and iron deficiency anemia, thereby harming cancer patients.²

2. No dairy products. These are the main sources of calcium in the American diet. Lack of adequate calcium damages bone maintenance, thereby harming cancer patients.²

3. No animal protein. Animal protein is the exclusive source of vitamin B₁₂ in the American diet, with the exception of vitamin B₁₂-fortified foods and microorganisms such as

seaweed. Lack of this vitamin interferes with basic biochemical processes in normal tissue, thereby harming cancer patients.²

4. Increased ingestion of fruits and vegetables. Such a diet is high in bulk and low in calories, just opposite to the needs of cancer patients.² In addition, it is low in needed animal protein, thereby harming cancer patients.² Furthermore, fruits and vegetables contain varying quantities of the enzyme β -glucosidase, which releases cyanide from laetrile,³ thereby making cyanide poisoning almost certain.

5. Megadoses of vitamin C (ascorbic acid). In studies being prepared for publication with Dr R. C. Backer, we found that megadoses of vitamin C release cyanide from laetrile. This increases the probability that the patient will suffer not only from chronic cyanide poisoning, with progressive fatigue, weakness, nerve damage, blindness, and deafness, but also acute bouts of cyanide hypotension, nausea, vomiting, abdominal cramps, rash, fever, diarrhea, headaches, vertigo, dyspnea, paralysis, unconsciousness, convulsions, and death.³ Other possible undesirable effects of megadoses of vitamin C are delineated elsewhere.⁴

6. Megadoses of vitamin E. Possible undesirable effects of megadoses of vitamin E are delineated elsewhere.⁴

7. Oral pancreatic enzymes. These have no value except in pancreatic disease.⁵ Since they are proteins, they are destroyed in the intestine, and thereby not only waste money, but also are a possible source of undesirable side effects for cancer patients.²

8. Pangamic acid ("vitamin B₁₅"). Proponents have *trade named* this substance as a vitamin, but the Food and Drug Administration is not aware of any accepted scientific evidence that this substance has nutritional properties, states it "is not generally recognized by scientific experts as safe for human consumption," and considers it illegal to be sold as a dietary supplement (*FDA Talk Paper*, March 31, 1978).

9. Laetrile. This chemical was isolated in 1835 as amygdalin. It is a cyanogenetic glycoside, which the FDA states has no known value in human nutrition.³ It has been proposed as a cancer remedy since 1845, but was never found to be of any value against cancer.³ In the time of the pharaohs, it was used as an extract for official executions. Proponents *trade named* amygdalin as

"laetrile" and then as "vitamin B₁₇." It is two parts sugar, one part benzaldehyde, one part cyanide, and no parts vitamin.³

Laetrile may actually *cause* cancer, since cyanogenetic glycosides ("laetriles") have been shown to be goitrogens in Africa, mutagenic in rats and pigs, and may give a positive Ames test result; while taking laetrile, a second cancer developed in a patient with cancer.³

Thus, the "nutritional and metabolic" program of laetrile proponents is perhaps as unhealthy for cancer patients from the nutrition and metabolism standpoint as it is possible for the mind of man to conceive.

VICTOR HERBERT, MD, JD
Veterans Administration Hospital
Bronx, NY
State University of New York
Downstate Medical Center
Brooklyn

Dr Herbert is supported by a Veterans Administration Medical Investigatorship and Public Health Service grant AM 20526.

1. Richardson J, Griffin P: *Laetrile Case Histories*. New York, American Media and Bantam Books, 1977.
2. Goodhart RS, Shils ME (eds): *Modern Nutrition in Health and Disease*. Philadelphia, Lea & Febiger, 1975.
3. Schmidt ES, Newton GW, Sanders SM, et al: Laetrile toxicity studies in dogs. *JAMA* 239:943-947, 1978.
4. Herbert V: Megavitamin therapy. *J Am Pharm Assoc* 17:764-766, 1977.
5. Herbert V: Laetrile: The cult of cyanide. *Am J Clin Nutr*, to be published.

Inadvertently Induced Hypoglycemia

To the Editor.—Factitious hypoglycemia, induced by oral antidiabetic agents,^{1,2} should be suspected in diabetic or psychiatric patients and in subjects who have diabetic relatives or work in medical institutions.³

None of this was true in our patient, a 62-year-old housewife, who was admitted to our department because of unconsciousness. Her blood glucose level was 33 mg/dl. After intravenous (IV) administration of glucose, she promptly regained consciousness, and the blood glucose level rose to 96 mg/dl.

The patient's past history was irrelevant, except for glaucoma, presently treated with acetazolamide (Diamox) and pilocarpine. Two days before admission the patient complained of an unusual weakness and vertigo; she became slightly confused and had a voracious appetite. A copious intake of sweet drinks and chocolate abated these symptoms. Six hours after admission, while receiving a 5% glucose infusion, the patient became noticeably confused. Her blood glucose level again dropped to 43 mg/dl. After IV administration of 20 ml of a 50% glucose solution, an

immediate improvement of her condition was noted. A similar episode occurred 12 hours later.

The presumption that the recurrent hypoglycemic attacks were related to some iatrogenic factor led us to investigate the possibility that an absent-minded pharmacist erred and instead of acetazolamide (Diamox) tablets, supplied chlorpropamide (Diabinese) to our patient. The similarity of names of both drugs (ie, "dia" prefix) is the reason these tablets lie side by side in the alphabetically arranged medicine chest. The enthusiasm provoked by this suspicion proved justified: nine tablets of Diabinese were found in our patient's home. This batch was given to her three days before admission, and her troubles began after she started taking them.

Since hypoglycemia due to inadvertence is rare,² one has to consider a similar circumstance in patients with unexplained hypoglycemia.

DAN ADERKA, MD
JACK PINKHAS, MD
Bellinson Medical Center
Petah Tikva, Israel

1. Scarlett JA, Mako ME, Rubenstein AH, et al: Factitious hypoglycemia. *N Engl J Med* 297:1029-1032, 1977.
2. Miller DR, Orson J, Watson D: Upjohn, down glucose. *N Engl J Med* 297:339, 1977.
3. Jordan RM, Kammer H, Riddle MR: Sulfonylurea-induced factitious hypoglycemia: A growing problem. *Arch Intern Med* 137:390-393, 1977.

Fiduciary Responsibility

To the Editor.—"We Physicians Are Fiduciary Failures" (239:1629, 1978) points up a most valuable concern, but fails to take into account many other equally important aspects of the problem. Indeed, if we are to discharge our fiduciary relationship to the patient honorably, we should consider only that individual's physical, mental, and financial well-being rather than that of some third party. Obviously the latter is inappropriate, but if we are going to play a perfectly open game with the patient in terms of cost, should we not also explain to the patient the cost-effectiveness ratio of each given test or procedure, the role of the sequence in which these are done, including delays that may be introduced as a result of sequence (eg, a small-bowel contrast study can be done almost immediately after intravenous pyelography but not vice versa)? Furthermore, the patients would have to assume some liability for consequences of their decisions, since it would seem unreasonable to hold the physician alone responsible for the consequences of decisions other than those that he recommended.

While physicians may not always have at their fingertips the exact information as to the cost of each item referred to in the COMMENTARY, they are sharply aware that most patients want to have done for them whatever is most likely to be helpful. Patients and their families do not want almost the best, but they want and expect the best and are prepared to go to court if they have reason to believe that they have not received it. They want no stone left unturned, the cost be damned, especially since someone else is paying the charges. Somehow the public manages to feel very cost conscious when insurance premiums are being discussed, but not when the discussion shifts to benefits.

Third-party payers are not the innocent bystanders they would have us believe. They have a long tradition of opposing cost-saving ideas, eg, preadmission testing, outpatient rather than in-hospital diagnostic evaluation, outpatient surgery, and office care in general. That behavior is perhaps not too surprising, since carrier company profits are usually considered as a percentage of total business volume. In that circumstance, why should the carriers seek to reduce cost and hence their own profit?

M. J. WIZENBERG, MD
The University Hospital and Clinics
Oklahoma City

Diagnosis of Carbon Monoxide Poisoning

To the Editor.—In a recent article (239:1515, 1978) Kelley and Sophocleus point out the difficulty in making the clinical diagnosis of carbon monoxide poisoning. They emphasize that ophthalmoscopy should always be performed in patients with nausea, headache, and dizziness, and that retinal hemorrhages should suggest the possibility of CO poisoning. Following their recommendations will certainly add to the clinical examination of such patients, but I believe several statements in their article are confusing regarding the laboratory diagnosis of CO poisoning.

In several places the authors refer to *serum* CO measurements. Clearly, anticoagulated whole blood, rather than a clotted serum tube, is the proper specimen to submit to the laboratory, since virtually all the CO in the blood is tightly bound to the RBC's hemoglobin.