Hg systolic in serious cases, and the pulse may become very weak. Death, if it occurs early, is usually the result of paralysis of the respiratory center. If death is delayed, vasomotor collapse, pulmonary edema or hypostatic pneumonia may follow acute poisoning from overmedication and frequently results in the fatal issue. The latter result, however, is more apt to occur from the long acting barbiturates. Bronchopneumonia may occur in a patient who has regained consciousness. The prognosis depends upon the amount and the type of barbiturate ingested. The longer the patient survives the better his chance for living. . . . When a patient suffering from barbituric acid poisoning is found at home in a coma, it is best to have him removed to a hospital for treatment. At the hospital evacuate the patient's stomach with a stomach pump, washing it with potassium permanganate of a strength of about 1:3000. . . . After the stomach is thoroughly washed, if the patient is cyanotic, oxygen should be administered by nasal catheter. While the stomach is being evacuated, the nurse may prepare and have in readiness, a hypodermic containing a 0.3 per cent solution of picrotoxin. One cc. containing 3 mg. can be injected intravenously; fifteen to twenty minutes later another 3 mg. may be given. If the respiration improves, or if the patient attempts to make slight movements, 6 mg. may be sufficient. In cases of severe poisoning, however, it may be necessary to give up to 12 mg. Slight twitching of the face indicates that the optimum effects of the drug are being reached. Picrotoxin itself is a poison; with this in mind, the physician should have a rapidly acting soluble barbiturate such as sodium pentobarbital or sodium pentothal on hand and ready for intravenous injection if the patient has a convulsion. . . .

"In the absence of picrotoxin, metrazol may be given intravenously at intervals until the desired effect is obtained. I have used strychnine sulfate hypodermically every hour until there was an increase reflex excitability with good results. Five per cent glucose is also excellent for elimination and to prevent pulmonary edema; 500 to 600 cc. of the 5 per cent glucose may be given. As the barbiturates are eliminated in the urine, the patient should be catheterized twice in twenty-four hours. If the respiration is weak, give 7 to 10 per cent of carbon dioxide with oxygen. On the second or third day, the patient may have a high temperature even though the stethoscope or x-ray may fail to reveal pulmonary complications. If this temperature persists beyond the third day, a poor prognosis may be expected. If pulmonary edema occurs, give the patient intravenously an ampoule of salyrgan and 50 cc. of 50 per cent glucose and administer oxygen. . . . The therapeutic dose does not cause death, but when fifteen times the ordinary hypnotic dose has been absorbed, the patient's life is in danger. Death has followed less than 1 Gm. (15 gr.), but recovery occurred after the ingestion of 18 Gm. 22 references.

J. C. M. C.

WEISMAN, S. A.: *A Study of the Analeptic Value of Certain Drugs in the Treatment of Quinidine Depression.* Am. Heart J. **24**: 240–271 (Aug.) 1942.

"The present study records data on the relative values of ten drugs which are used experimentally as analeptics in varying states of cardiovascular and respiratory depression caused by quinidine. . . . Dogs were anesthetized with pentobarbital sodium, which was given intraperitoneally in doses of 35 mg. per kilogram of body

weight. Quinidine was then administered intravenously in doses of varying size; 25 mg. per kilogram and less were considered small doses, and more than 25 mg. per kilogram was considered large. We found that 45 mg. of quinidine per kilogram was a sublethal dose. . . .

"Coramine, picrotoxin, metrazol, and caffeine sodium benzoate were of some value as respiratory stimulants when depression of the cardiovascular and respiratory systems had been induced by small doses of quinidine. The order of effectiveness of these drugs as respiratory stimulants in light quinidine depression was as follows: metrazol, caffeine sodium benzoate, coramine, picrotoxin. After large doses of quinidine, depression was often aggravated by metrazol, coramine, and picrotoxin. Paredrinol, ephedrine, benzedrine, paredrine, epinephrine, and neosynephrin showed definite value as circulatory stimulants, after both small and sublethal doses of quinidine. Ephedrine, epinephrine, and benzedrine also showed definite respiratory effects. There was some evidence that paredrinol produced respiratory stimulation, although very little. Ephedrine was the least effective in counteracting the circulatory depression caused by small or large doses of quinidine. The effect of benzedrine and paredrinol on the blood pressure rise was very much the same; however, the time of appearance of the maximum rise in blood pressure after the administration of these drugs was definitely much shorter after giving benzedrine. The effect of neosynephrin lasted twice as long as that of epinephrine. Paredrine was one of the most valuable pressor substances in this group. It was about twice as effective as ephedrine, benzedrine, or paredrinol. Although the blood pressure response was much less after its administration, compared to that of epinephrine and neosynephrin, the effect of paredrine lasted much longer. The most effective pressor substance in this group was neosynephrin. The blood pressure response was about twice as high, and the effect lasted twice as long after giving neosynephrin as after administering epinephrine, after both small and large doses of quinidine." 31 references.

J. C. M. C.

ROCHBERG, SAMUEL, AND APGAR, VIRGINIA: *Metabolic Effects of the Anesthetic Agents.* Am. J. Surg. **57**: 336–345 (Aug.) 1942.

"Before the anesthetist puts his patient to sleep, it is important that he know what effect the drug or drugs he is going to use will have upon that particular individual's metabolism both during and after the operation. It is the duty of the anesthetist to maintain the physiological 'status quo' of his patient. . . . It is obvious that rarely is there a need for chloroform in the anesthetist's armamentarium, because of its dangerous metabolic effects. Ether, which is supposed to be our safest anesthetic agent, produces marked metabolic disturbances, which, though only temporary in nature, may be most harmful to the very ill patient. Of great importance is the fact that cyclopropane is almost innocuous to the body metabolism. When vinethene or chloroform is administered by the open drop method, the patients should be given additional oxygen by either the oral or nasal route. Vinethene should not be used for long procedures, and should not be used too often on the same individual. Avertin should not be employed in cases of hepatic or renal disease, in patients with a subnormal metabolism, or in elderly patients who frequently have potential kidney dysfunction and whose physiological functions will not withstand further depression. The