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ACUTE BARBITURAL INTOXICATION

HOWARD R. MARVEL, M.D.*

Barbituric acid derivations are the most popular and widely used drugs of the sedative and hypnotic group. They are easily available and do not come under the stringent regulations of the Harrison Narcotic Act. In fact only twenty-seven states have laws preventing their sale without prescription by a physician. As a result accidental and deliberate intoxications are common. In the last ten years there has been a remarkable increase in the number of cases of barbiturate intoxication arriving at large city hospital emergency rooms. A review of the subject of acute barbiturate intoxication is appropriate to all of us who must treat these emergencies as they arrive.

Although there are numerous barbiturate preparations on the market, only a few are of major importance. In terms of duration of action these are:

- **Long duration**: Phenobarbital
- **Moderate duration**: Amytal
- **Short duration**: Seconal
- **Barbital**: Pentobarbital

An ultra-short actor, sodium pentothal, actually a thiobarbiturate, while commonly used in anesthesia, is not generally used with suicidal intent.

Barbital and phenobarbital are just as capable of producing coma as the shorter acting drugs. Their absorption from the gastro intestinal tract is somewhat slower but their elimination from the body is also much slower. Traces of barbital may be found in the urine nine days after a hypnotic dose. Eight per cent of the drug will be excreted in twelve hours, twenty per cent in twenty-four hours and thirty-five to sixty-five per cent in forty-eight hours. The slow rate of excretion is the basis for the cumulative effects of the long actors. In the presence of kidney damage the excretion rate may be even slower.

The moderate and short acting barbiturates are detoxified by the liver. Their action may be prolonged and more profound in cases where severe liver impairment is present.

The lethal dose of the various barbiturate preparations for human beings can not, of course, be definitely established. The presence or absence of kidney or liver disease as well as the general health must be considered in evaluating the prognosis of a given case. The reader is referred to the recent publications of Koppanyi and Fazekas for further information regarding lethal dosage.

The diagnosis of acute barbiturate intoxication is usually simple. The great majority of patients who attempt suicide will tell relatives or friends what they

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have done or will admit taking the drug on direct questioning before they lapse into coma. Frequently, an empty medicine bottle will be noted near the patient. In some instances, however, a patient in a stuporous state may take repeated doses without realizing what he is doing. Rarely the ingestion may be accidental or may occur in a concealed form with homicidal intent.

The signs and symptoms vary. In the beginning there is dullness in the head, muscular incoordination and impairment of smell and taste. Later there is dysphagia, occasionally nausea and vomiting, stupor, sleep and coma. Occasionally excitement and hallucinations occur before stupor and sleep overcome the patient. The patient in coma progresses on to shallow respirations and circulatory collapse. Death may be due to circulatory collapse, bronchial pneumonia or respiratory failure.

The diagnosis should always be suspected in the comatose patient who is breathing quietly and regularly. Laboratory tests are slow and tedious and, therefore, do not have much practical value. They are, however, of medico-legal importance.

Considerable controversy exists in respect to the various methods of treatment available for the patient. Treatment possibilities vary from the symptomatic therapy proposed by Nilsson on to the various stimulants and analeptics and finally to electrostimulation.

Koppanyi believes that prognosis based on tables of degrees of coma is not reliable. He believes that all cases in coma should receive an orientation dose of metrazol, (5cc of a ten per cent solution intravenously). If a favorable response occurs, only symptomatic care is indicated. A favorable response consists of coordinated voluntary movement, a return of corneal reflexes, an increase in muscle tone and improvement in circulatory status. If the response is favorable then metrazol, 5cc every ten to fifteen minutes until complete awakening, is used. If the response is unfavorable picrotoxin is used. The initial dose is twenty-five milligrams intravenously, then fifteen milligrams every fifteen minutes until there is evidence of awakening, or at least a return of reflexes. The dose is then reduced but not discontinued until the patient can perform voluntary purposeful movements.

Reed, Driggs, and Foote, after experiences with 300 cases, have proposed the following system of classification of coma.

Group 0—A patient who is asleep but can be roused and will sit up in bed, drink fluids, etc.

Group 1—A patient who is comatose but will withdraw from painful stimuli such as venipuncture, slapping, pinching, etc. There is no circulatory embarrassment and all reflexes are intact.

Group 2—A patient who does not withdraw from painful stimuli but has no respiratory or circulatory depression and, most important of all, whose reflexes are intact.
Group 3—A patient whose reflexes are mostly or entirely gone but who shows no evidence of respiratory or circulatory depression.

Group 4—A patient with absent reflexes, respiratory depression with cyanosis, or circulatory failure and shock, or both.

The authors emphasize that grouping should not be done until an airway is established. They also consider the tendon reflexes most reliable and have found pupil size and corneal reflexes unreliable criteria in many instances.

Analeptic drugs are then used according to the grouping as follows:

Group 0 to 1—observation, no analeptics.

Group 2—Caffeine and sodium benzoate 0.5 gram every two hours. Do not exceed six grams in twenty-four hours; or amphetamine twenty-five milligrams every two hours but not over three hundred milligrams in twenty-four hours.

Group 3—Caffeine or amphetamine as above, if reflexes not restored in fifteen to twenty minutes give picrotoxin every fifteen to twenty minutes intravenously. Use 1cc (three milligrams); if no twitching give a second dose of six milligrams and watch, continue as necessary until twitching occurs.

Group 4—Caffeine or amphetamine as before, then if necessary picrotoxin six to nine milligrams every twenty minutes. A Drinker respirator should be used if necessary. Oxygen should be used but with great care because the anoxic stimulus on the carotid body is the only mechanism left for maintaining respiration.

Most authors agree as to the symptomatic measures necessary for satisfactory care of the patient. They are:

1. Careful observation—blood pressure, pulse, respirations, and reflexes every half hour. Observation is of the greatest importance because the degree of coma may change and the plan of therapy may need to be altered.

2. Immediately on arrival an adequate airway should be established. If laryngospasm is present an endotracheal tube should be inserted. Bronchoscopy should be done if aspiration of vomitus occurs.

3. Place bed in the shock position and turn the patient frequently.

4. Prophylactic antibiotics; penicillin, usually, but in the presence of shock and hypostatic pneumonia broad spectrum antibiotics may be more effective.

5. In all patients except those in extremis, do gastric lavage. This is important because a portion of the drug may lie dormant in the stomach, in deep coma and later be absorbed as recovery begins.

6. Fluid balance as in any unconscious patient.

7. Ninety-five per cent O₂, five per cent CO₂—with care when the patient is in deep coma.
8. Observation for cardiac arrhythmias. This is especially important in patients receiving caffeine or amphetamine. The dose limits proposed by Reed, et al, should not be exceeded.

9. Treatment of circulatory collapse may require the use of pressoramines in the event that the other symptomatic measures, oxygen and analeptics, do not restore the blood pressure.

Nikethamide and disodium succinate have not been found to be effective. Electrostimulation\(^4,5\) has been reported as a successful mode of therapy in a few cases but further results must be reported before final evaluation can be made.

BIBLIOGRAPHY


