As the author states, “no single choice of anesthetic agent answers all the questions in all patients, whether for the alcoholic, addict, or frequent user of marijuana.” However, knowing the physical manifestations that these patients present and the resultant effects anesthesia will have on them influences the final decision-making process.

More than half of the trauma in the United States, principally caused by motor vehicle accidents, directly involves intoxicated drivers. Many of these drivers are influenced by the concomitant use of other drugs. Acute drug abuse or ethanol intoxication in a patient who is subsequently traumatized and who presents for emergency surgery poses many problems to the anesthetist. This article alerts the anesthetist to the adverse interactions which may occur when alcohol and/or “street drugs” are combined with general anesthesia.

The acutely intoxicated alcoholic is often injured while drunk. As a result, such a patient generally reaches the operating room mentally unprepared, and is usually belligerent if awake. The patient is just as likely to be stuporous or uncooperative, and probably will have a full stomach. The aim for the anesthetist is to isolate the airway with a cuffed endotracheal tube without promoting vomiting and aspiration, and induce anesthesia quickly. There are, however, several considerations about the alcoholic that the anesthetist must be aware of before giving the anesthetic.

Illnesses associated with alcoholism

Of the illness associated with alcoholism, hepatic dysfunction is the most common. While it takes 10 to 15 years of drinking to acquire cirrhosis, other nonreversible changes can occur earlier in the liver. In man, the mechanisms of liver changes have been attributed to fatty infiltrations, protein disturbance, and mitochondrial and membrane alterations.

Hepatic cirrhosis may be associated with hyperventilation and arterial oxygen desaturation, the latter due to shunting of blood from peri-esophageal and mediastinal veins to pulmonary veins. The chronic alcoholic is often a heavy smoker. Pathological conditions in the lungs may relate to frequent aspiration and emphysema. Some cardiac disease is common in the alcoholic as are pulmonary problems. In addition, arrhythmias are frequent, mainly atrial fibrillation and conduction disturbances. There is increased peripheral blood flow and vasodilation, and this may result in cardiac hypertrophy. There may be cardiomyopathy and congestive heart failure. Renal disease is not infrequent to the alcoholic, and the incidence of glomerulonephritis also is higher than usual.
The ethanol-induced widespread cutaneous vasodilatation renders acutely intoxicated individuals more susceptible to hypothermia than normal patients. In acute alcoholism, patients withstand shock badly due to vasodilation. Ethyl alcohol also contributes to fluid and electrolyte derangements both in the acutely intoxicated patient and in the chronic alcoholic. Alcohol inhibits the antidiuretic hormone (ADH) secretion resulting in free H₂O diuresis. The presence of decreased serum and total body magnesium levels in the chronic alcoholic has been a consistent finding, as is thiamine deficiency.

Alcohol intoxication and/or chronic alcoholism seems to modify the metabolism of a variety of pharmacologic agents. Ethyl alcohol is known to cause enzyme induction in chronic alcoholics, but when an intoxicating amount of alcohol is present in the blood, the hepatic biotransformation of many drugs may be inhibited, leading to increased therapeutic or toxic effects. This effect helps to explain what is often observed clinically. Acutely intoxicated patients respond to other depressants in an additive manner; while sober, chronic alcoholics seem resistant to depressive drugs, such as narcotics, analgesics, barbiturates, and major and minor tranquilizers.

Choice of agent

The choice of anesthetic agent and technic in the alcoholic elicits much debate. The multiplicity of diseases involving visceral organs makes regional anesthesia the preferred choice in the minds of some, though there is no evidence that the management of smooth general anesthesia has any deleterious effects. There is no single or right way to manage the task; each anesthetist must develop his or her own program.

In choosing the anesthetic, the anesthetist must keep in mind that the importance of maintaining hepatic blood flow in patients with liver disease is imperative. Severe hepatic dysfunction results in diminished plasma cholinesterase, and in prolongation of neuromuscular blockade following use of succinylcholine. There is diminished cholinesterase at the motor end plate which allows endogenous acetylcholine to accumulate. This would explain the often observed clinical impression that patients with hepatic dysfunction seem to be resistant to nondepolarizing muscle relaxants. Another factor may be the reversal of the albumin to globin ratio found in liver disease. This alteration of plasma proteins could result in increased binding of curare with resistance.

Although increased tolerance for anesthesia in alcoholics has never been firmly established, most anesthetists are convinced that the alcoholic "takes more" anesthesia. Although the association between liver necrosis and halothane may never be entirely clear, there is no strong evidence contraindicating the use of halothane. Impending delirium tremors may resemble the signs of central nervous system (CNS) irritability caused by enflurane, and control of depth of awareness is difficult with balanced technics. The signs of withdrawal should be looked for postoperatively, with careful sedation of the patient being the key to successful therapy. Tranquilizers such as diazepam are most useful.

Anesthesia for the drug abuser

Do drug abusers who require surgery present drug related anesthetic problems? In the patient with physiological dependence on drugs the anesthetist must be aware of a number of coexisting problems. These include the tolerance of drugs employed for anesthetic purposes, the possibility of withdrawal signs, hepatic dysfunction, and various cardiopulmonary problems, including endocarditis.

Amphetamines and lysergic acid diethylamide (LSD) produce sympathomimetic effects which are dose-related, including tachycardia, hypertension, arrhythmias, and fever. In view of these cardiovascular changes, pulse, blood
pressure, and respiratory rates may be unreliable in assessing the blood loss or the anesthetic level. Narcotic, barbiturate, and amphetamine drug users frequently resort to intravenous routes of administration. This eventually results in destruction of superficial veins, rendering intravenous therapy limited. Acute pulmonary edema following heroin overdosage has been observed for some time. Whether this condition is due to the heroin or the additives used to dilute the street drugs is not known.¹

Hypotension during surgery, which may be a manifestation of withdrawal, has occurred in narcotic addicts. Drugs frequently used in anesthesia addicts will potentiate the depressive effects of barbiturates and opiates, which may result in fatal respiration depression in the postanesthetic period.¹

Anesthetic management

Anesthetic management starts with establishing the diagnosis. Some patients are cooperative and admit to their addiction. For patients on methadone maintenance, it appears wise to prescribe the usual dose of methadone on the day of surgery, in addition to the usual premedication. Resumption of methadone postoperatively is advisable.⁵ Nalorphine must be avoided in addicts to the opiates or pethidine, since its use may precipitate acute withdrawal symptoms of a shock-like nature.

Although a patient may not be actively taking morphine or other opiates, morphine should be avoided. Even a single injection may reactivate psychological dependence. Meperidine can be used to supplement the therapeutic dose of methadone. Fentanyl is probably useful, but too little information is available to make any definite statement with relation to the addict. Pentazocine should never be used because it will precipitate an acute withdrawal syndrome. If the injured patient is suffering from an overdose and circulation is depressed, appropriate naloxone reversal is indicated.²

The choice of anesthetic agents is not limited. Reviews of hepatitis and anesthetic agents fail to uncover any contraindications to halothane in the addict. The major liver problem is the possibility of subclinical hepatitis. Shunting through the lung is common in the sick addict, and hypoxemia should always be considered. Postoperatively, meperidine is an excellent analgesic from which addicts can be rapidly weaned back to their methadone or other therapeutic regimen.²

Considerations for the marijuana user

The principal pharmacologically active component of marijuana is delta-9-tetrahydrocannabinol (THC). Reports of analgesia, sedation, and prolonged barbiturate sleeping time after THC administration suggests that this compound might have anesthetic-like properties. As many as 20 million Americans may have smoked marijuana. It is conceivable that some might undergo anesthesia and operations shortly after smoking marijuana, and any alteration in anesthetic needs should be appreciated.⁶

Factors other than anesthetic requirements may influence the response of the marijuana user to anesthesia. Smoking marijuana produces tachycardia and increased peripheral blood flow; consequently, subsequent responses to vasoactive drugs and anesthetics could be altered. THC may act as a cholinesterase inhibitor and increase the hazard of toxicity following neostigmine. If smoked in combination with the ingestion of alcohol, the additive depressant effects of this combination might decrease anesthetic requirements. Enzyme induction after chronic marijuana smoking may alter biotransformation and potential toxicity of other drugs.⁶

Marijuana has been shown to produce additive effects with halothane and cyclopropane, in humans, thereby creating a potential hazardous condition.⁴ Experiments indicate that the patient

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who has used marijuana before receiving an anesthetic may be already “pre-
medicated.”

There have been other warnings of dangerous drug interactions between mari-
jjuana and anesthetic medications. Marijuana alone causes an increase in pulse rate which is potentiated by atro-
pine sulfate and epinephrine. Marijuana has been shown to double the ketamine sleep-
ing time and to quadruple thiopental duration. The combination of in-
creased cardiac demand from tachy-
cardia with sustained catecholamine elevation has led some authors to warn that the use of marijuana in elderly or cardiac patients may be dangerous. It has been shown that smoking marijuana significantly decreases exercise toler-
ance.

Potential drug interactions occurring many hours, or even days, after mari-
jjuana smoking appear quite possible in light of the catabolism and excre-
tion of marijuana. A range of 20% to 35% of active cannabinols may remain in the tissues for longer than 72 hours after a single administration.

Conclusion

In conclusion, no single choice of anesthetic agent answers all questions in all patients, whether for the alcoholic, the addict, or the frequent user of mari-
jjuana. Preoperative evaluation may be incomplete due to poor patient co-
operation. Adequate history may be, and often is, unobtainable. Physical findings and neurologic status may be difficult to interpret. An organized plan of approach is more important than the choice of therapeutic agent.

REFERENCES


AUTHOR

Sandra L. Weiss, CRNA, BA, is a 1974 graduate of the Hurley Medical Center School of Nursing, and a 1977 graduate of Hurley's Anesthesia School, where she is currently a didactic and clinical instructor. She received her bachelor's degree from Ot-
tawa University, Kansas City in Health and Education. This work was prepared by Miss Weiss with the recommendation of Howard Stickney, CRNA, BA, the Program Director at Hurley Medical Center, Flint, Michigan.