Anesthesia for Drug Dependent Patients

Essay
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Mohamad Abdallah Mohamed

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ADH:	Anti-Diuretic Hormone
5-HT:	5-Hydroxy Tryptamine
ADH:	Alcohol Dehydrogenase
AIDS:	Auto Immune Deficiency Syndrome
AUC:	Area Under Curve
BEC:	Blood Ethanol Concentration
BZDs:	Benzodiazepines
C AMP:	Cyclic Adenosine Mono Phosphate
CAs:	Cyclic Anti-depressants
CBF:	Crebral Blood Flow
CMR:	Cerebral Metabolic Rate
CNS:	Central Nervous System
CYP:	Cytochrome P-450
DA:	Dopamine
DIC:	Disseminated Intravascular Coagulation
ECG:	Electro-Cardiography
FGA:	First Generation Anti-psychotic
FPM:	First Pass Metabolism
GABA:	Gamma Amino Butyric Acid
GIT:	Gastro-Intestinal Tract
HIV:	Human Immune Deficiency Virus
IV:	Intra Venous
LC:	Locus Ceroeleus
MAC:	Minimum Alveolar Concentration
MAOIs:	Mono Amine Oxidase Inhibitors
MH:	Malignant Hyperthermia
NA:	Nore Adrenaline

List of Abbreviations (Cont.)

N-Ac:	Nucleus Accumbens
NE:	Nore Epinephrine
NMS:	Neuroleptic Malignant Syndrome
NSAIDs:	Non Steroidal Anti-Inflammatory Drugs
Pa CO2:	Arterial Carbon Dioxide Pressure
RIMAs:	Reversible Inhibitors of Monoamine oxidase
ROD:	Rapid Opioid Detoxification
SGA:	Second Generation Anti-psychotic
SSRIs:	Selective Serotonin Re-uptake Inhibitors.
TCAs:	Tri-Cyclic Antidepressants.
UROD:	Ultra Rapid Opioid Detoxification
VTA:	Ventral Tegmental Area

Introduction

Substance abuse may be defined as self-administration of drugs that deviate from accepted medical or social use, which if sustained can lead to physical and psychological dependence ***(Roberta and Katherine, 2008).***

The drug abusing patient presents for surgery with problems common to all drug abuse, and some specific to the individual drugs. Drug dependence is a chronic relapsing neurophysiologic disease resulting from the prolonged effects of drugs on the brain. The neurochemical abnormalities resulting from chronic use are the underlying cause of many of the observed physical and behavioral aspects of addiction ***(Langford et al., 2003)***.

A patient with a history of current or previous drug addiction presents special challenge for the perioperative team. Although the prevalence of the drug addiction is significant (7%), there is limited information based on prospective clinical trials to guide perioperative management ***(Judith et al., 2001).***

Anesthesiologists encounter emergency cases in which 'party drugs' have clearly been used, and may also be anesthetizing patients in whom abuse is present but unrecognized. Understanding how illicit drugs interact with anesthetic agents is of paramount importance ***(Steadman and Birnbach, 2003).***

Introduction

Substance abuse has crossed social, economical, and geographic borders, and it remains one of the major problems facing society today.

Drug abuse may result in increased morbidity and mortality during intra and postoperative period, therefore, a thorough understanding of the consequences of drug abuse is essential for practicing anesthesiologists. ***(Rudra et al., 2008).***

Anesthesia and postoperative analgesia in patients dependent on psychoactive substances pose special problems. Often these patients suffer from severe medical and psychotic illness. In addition, drug-specific adaptations such as tolerance, physical dependence, and withdrawal may diminish the effectiveness of anesthetic and analgesic drugs. Therefore, problems resulting from substance intoxication, or recent ingestion of or exposure to a substance may arise in evaluation of patients for anesthesia. When patients present for anesthesia and surgery there is a mandate to detect, manifest mental problems, as well as covert substance use patterns that may impact response to anesthetic agents and the surgical procedures ***(Rudra et al., 2008).***

Even if there are few scientific data about preoperative management of drug addict patients, a careful anesthesiology evaluation facilitates the prevention of interactions between drugs and anesthetics and avoids intraoperative and postoperative complications. Moreover it is important to earn the confidence of patient in order to know the abuse substances consumed and the frequency of consumption. This knowledge is necessary to the anesthetist in order to manage possible withdrawal syndromes or overdoses, which are the two greatest dangers for a drug addict patient during the hospital stay ***(Cavaliere, 2005).***

Narcotic analgesics

The term "narcotic," derived from the Greek word for stupor, originally referred to a variety of substances that dulled the senses and relieved pain. Today, the term is used in a number of ways. Some individuals define narcotics as those substances that bind at opiate receptors while others refer to any illicit substance as a narcotic. In a legal context, narcotic refers to opium, opium derivatives, and their semi-synthetic substitutes. Cocaine and coca leaves, which are also classified as "narcotics", neither bind opiate receptors nor produce morphine-like effects, and are discussed in the section on stimulants ***(Storm et al., 2003).***

Narcotics are used therapeutically to treat pain, suppress cough, alleviate diarrhea, and induce anesthesia. Narcotics are administered in a variety of ways. Some are taken orally, transdermally (skin patches), or injected. They are also available in suppositories. As drugs of abuse, they are often smoked, sniffed, or injected. Drug effects depend heavily on the dose, route of administration, and previous exposure to the drug. Aside from their medical use, narcotics produce a general sense of well-being by reducing tension, anxiety, and aggression. These effects are helpful in a therapeutic setting but contribute to their abuse ***(Storm et al., 2003).***

Narcotic addiction

Definitions:

1. Tolerance:

Tolerance is defined as either a need for increased amounts of the substance to achieve the desired effect or a diminished effect with continued use of the same amount of the substance. Withdrawal is manifested by a characteristic syndrome with sudden abstinence, but it may be relieved or avoided if the same or a closely related substance is taken ***(Jefferey, 2007).***

2. *Cross-tolerance:*

Tolerance or resistance to a drug that develops through continued use of another drug with similar pharmacological action ***(Jefferey, 2007).***

3. Physical dependence:

Physical dependence is the physiological adaptation of the body to the presence of an opioid. It is defined by the development of withdrawal symptoms when opioids are discontinued, when the dose is reduced abruptly or when an antagonist (e.g., naloxone) or an agonist-antagonist (e.g., pentazocine) is administered ***(O'Brien, 1996).***

Physical dependence is a normal and expected response to continuous opioid therapy. Physical dependence may occur within a few days of dosing with opioids, although it varies among patients. Physical dependence

(indicated by withdrawal symptoms) does not mean that the patient is addicted ***(O'Brien, 1996).***

4. addiction:

While tolerance and physical dependence are physical changes in the body, addiction is defined by aberrant changes in behavior. Addiction is compulsive use of drugs for non-medical reasons; it is characterized by a craving for mood-altering drug effects, not pain relief. ***(O'Brien, 1996).***

5. Pseudo-addiction:

Pseudo-addiction is an iatrogenic phenomenon, e.g. it is when problems result from the treatment efforts of health professionals ***(Weissman and Haddox, 1989).***

Pseudo-addictive behavior may occur when analgesics are prescribed in inadequate doses or at dosing intervals that are longer than the duration of action of the drug ***(Weissman et al., 1996).*** Pseudo-addictive behaviors are more likely to occur in patient care settings where health care professionals are inadequately trained in pain management and the rational use of opioids. The appropriate clinical response to pseudo-addictive behaviors is to reassess the patient's pain and to treat the pain adequately ***(Weissman et al., 1996).***

Molecular mechanisms of addiction

Tolerance, dependence, and addiction are all manifestations of brain changes resulting from chronic substance abuse and involve different brain pathways than those subserving acute drug reinforcement. Acute drug reinforcement appears to share a final common dopaminergic pathway from the ventral tegmental area of the brain to the nucleus accumbens ***(Kleber et al., 2005).***

Origins of drug liking:

Many factors, both individual and environmental, influence whether a particular person who experiments with drugs will continue taking them long enough to become dependent or addicted. For individuals who do continue, the drug's ability to provide intense feelings of pleasure is a critical reason ***(Nestler, 2002).***

When abused drugs travel through the bloodstream to the brain, they attach to specialized proteins on the surface of neurons that may be receptors, transporters, or even structural elements of the neurons. For example, opiates such as heroin bind to mu opioid receptors, which are on the surfaces of opiate sensitive neurons, and have their effects by inhibiting the cyclic adenosine monophosphate (cyclic AMP) second messenger system. Inhibition occurs through a guanine nucleotide-binding (G)-protein-mediated coupling leading to a series of changes in phosphorylation for a wide range of intraneuronal proteins ***(Nestler, 2002).***

The linkage of heroin with the receptors imitates the linkage of endogenous opioids such as beta-endorphin with these same receptors and triggers the same biochemical brain processes that reward people with feelings of pleasure when they engage in activities that promote basic life functions, such as eating, sex and social interactions ***(Nestler, 2002).***

Mesolimbic (midbrain) reward system:

One of the brain circuits activated by opioids and most, if not all, abused drugs is the mesolimbic (midbrain) reward system. This system generates signals in a part of the brain called the ventral tegmental area (VTA) that result in the release of the chemical dopamine (DA) in another part of the brain, the nucleus accumbens (N-Ac) ***(Koob and Le Moal, 2001).***

This release of DA into the N-Ac causes feelings of pleasure. Other areas of the brain create a lasting record or memory that associates these good feelings with the circumstances and environment in which they occur. These memories, called "conditioned associations," often lead to the craving for drugs when the abuser reencounters those persons, places, or things, and they drive abusers to seek out more drugs in spite of many obstacles ***(Koob and Le Moal, 2001).***

Other abused drugs activate this same brain pathway, but via different mechanisms and by stimulating or inhibiting different neurons in this pathway.

For example, opioids and cannabinoids can inhibit activity in N-Ac directly, whereas stimulants such as cocaine and amphetamine act indirectly

by binding to various DA transporters and either inhibiting the reuptake of DA into the VTA neurons (cocaine) or actively pumping DA out of the VTA (amphetamine) at its synapse with the N-Ac neurons ***(Kosten, 2002; Stahl, 1998).***

Since stimulation of the DA D2 receptor inhibits the cyclic AMP system, this increase in DA in the synapse leads to relative inhibition of the N-Ac neuron. The mechanism is more complex than this, however, since the D1 receptor has the opposite effect on the cyclic AMP system (e.g., it increases the amount of cyclic AMP) and both D1 and D2 receptors are present on the N-Ac neurons ***(Stahl, 2002).***

The presumption is that the D2 receptor effects predominate perhaps simply due to more D2 receptors or due to a higher affinity of the D2 than the D1 receptors for DA. Other substances may be even more indirect in their stimulation. For example, nicotine and benzodiazepines stimulate ion channels for calcium/ sodium and chloride, respectively ***(Stahl, 2002).***

The calcium/sodium channel is a nicotinic receptor that normally binds acetylcholine, while the chloride channel is associated with a gamma-aminobutyric acid (GABA) receptor. The stimulation of these ion channels can lead to depolarization of the VTA neuron and release of DA into the synapse between the VTA and N-Ac. The entry of calcium into the VTA neuron can also directly facilitate the merging of the synaptic vesicles in the VTA with the cell membrane, leading to release of DA from these vesicles ***(Kosten, 2002).***

Particularly in the early stages of abuse, the drug's stimulation of the brain's reward system is a primary reason that some people take drugs