Delirium in the Intensive Care Unit

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Abstract

The most common cause of behavioral disturbances in the Intensive Care Unit (ICU) is delirium. Its manifestations are: changes in awareness, disturbance of the sleep-awake cycle, deficit of attention and concentration, disorganized thought originating incoherent speech, disturbances of perception like delusions and/or hallucinations, disorientation in time and space, agitation or reduced psychomotor activity and disturbed memory. The major causes of delirium in the ICU are: systemic and metabolic diseases, for instances, sepsis, renal failure and hepatic failure; exogenous toxic agents, e.g. some drugs; withdrawal from substances upon which the patient has become dependent, like alcohol; and primary intracranial diseases such as infections of the central nervous system. Other factors often coexist like sleep deprivation, previous cognitive deficits, fear, anxiety and the patient's personality. Treatment includes the correction of metabolic and systemic disturbances, the suspension of toxics and/or the use of antidotes, the withdrawal treatment, the use of haloperidol and benzodiazepines, and non-pharmacological actions that reduce the environmental stress and promote the physical and mental well-being.

Key words: delirium, ICU, haloperidol, benzodiazepines.

Introduction

In recent decades, the considerable advances in knowledge and medical technology have enabled conditions to be reverted that in the past, inevitably led to death. In the intensive care units (ICU), the medical care available today has been optimized, whether the accurate monitoring of various biological parameters, aggressive treatment of various organic disorders, or the use of technology to support vital functions.

Alongside these developments, doctors and nurses are being specially trained to treat potentially fatal cardiac and respiratory problems, ensuring the success of high risk surgeries, curing severe burns, maintaining the viability of a transplanted organ, etc. The severity of the situations treated in the ICU, and the skill of the respective personnel to carry out a range of dramatic interventions, aimed at saving patients' lives, have combined to give these places an air of danger that must surely affect the patients.

This development is relatively recent, therefore it is little wonder that just 30 years ago, Dr. McKegney used the term "ICU syndrome" to describe the acute alterations in behavior and mental state of patients admitted to intensive care units, considering it a new sickness.[1] For this and other authors, the environmental stress that is prevalent in the ICU can lead to alterations in the patient's mental state. Subsequently, ICU syndrome, designated by various authors as ICU psychosis - because hallucinations are very common among these patients – has been the object of various studies, which conclude that the most important causal factors are sleep deprivation, sensory deprivation or overload, and the monotony to which ICU patients are subject.[2,3,4,5] More recently, it was demonstrated that although these factors do contribute to the appearance of alterations in mental state, there are others that are certainly more important, such as systemic and metabolic disturbances, the toxic effect of medications, and withdrawal from alcohol or drugs.[6,7,8] The expression ICU psychosis was considered inadequate, because it does not reflect the range of symptoms observed in confused patients in an ICU, and because it suggests a causal relationship between being in the ICU and becoming psychotic.

The expression acute confusional state (ACS) appears to be more appropriate to describe the mental alterations commonly observed in ICU patients.[6,7,8] These include: disturbances of the waking-sleeping cycle, changes in awareness, disorientation in relation

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TABLE I

List of the main entities that can cause ACS in the ICU

System/problem	Etiological factors
a) Primary intercranial disease	**Infection**
	Encephalopathy HIV
	Meningitis/encephalitis
	Neurosyphilis
	Neoplasia
	Lesion occupying space
	Epileptic crisis
	Post-critical states
	Complex partial state
	Vascular
	Hypertensive encephalopathy
	Intercranial hemorrhage
	Vasculitis
	Stroke
	Miscellaneous
	Normal pressure hydrocephalus
b) Systemic diseases with secondary effect on the brain	**Cardiopulmonary**
	Cardiac arrest
	Congestive cardiac insufficiency
	Resp. insufficiency
	Shock
	Endocrine/metabolic
	Acid-base disturbance
	Suprarenal dysfunction
	Hydro-electrolytic imbalance
	Diabetic ketoacidosis
	Hypoglycemia
	Hepatic insufficiency
	Eenal insufficiency
	Parathyroid dysfunction
	Thyroid dysfunction
	Porphyria
	Infection
	Sepsis
	Subacute bact. endocarditis

TABLE I

(Continuation)

System/problem	Etiological factors
	Neoplasia
	Paraneoplastic syndromes
	Nutritional deficiencies
	Folic acid
	Miacin
	Thiamine
	Vit. B12
c) exogenic toxic agents	**Dependency drugs**
	Alcohol
	Amphetamines
	Cocaine
	LSD
	Phencyclidine
	Non medicamentous
	Carbon monoxide
	Heavy metals
	Medications (see table 3)
d) Drug withdrawal	**Alcohol**
	Propanediol
	Chloral hydrate
	Meprobamate
	Sedative-hypnotic agents
	Barbiturates
	Benzodiazepine
	Narcotics

Adapted from Lipawski, Z.J. – Delirium in the elderly patient – New England Journal of Medicine, 320:578, 1989 and Ludwig, A.M. – Principles of Clinical Psychiatry.

to time and space, disorganized thoughts, illusions and hallucinations, paranoid ideas, and often, agitation.

ACS is potentially serious for the patient, because it is often associated with hyperactivity of the sympathetic nervous system, with a consequent increase in energy consumption and deregulation of the body's homeostasis, and also because agitation is manifested as repeated attempts to get out of bed, pull out cathe-

TABLE II

Most frequent causes of ACS in the ICU

Sepsis
Medications
Metabolic causes
Hepatic insufficiency
Renal insufficiency
Endocrinal anomalies
Thyroid (hypothyroidism, hyperthyroidism)
Parathyroid (hypoparathyroidism, hyperparathyroidism)
Fever
Cranial traumatism
Cerebral hemorrhage
Vasculitis and other diseases of the collagen

Modified from McCartney, J.R.; Boland, R.J. – Understanding and managing behavioral disturbances in the ICU – The Journal of Critical Illness, Vol. 8, No. 1, January 1993.

ters or endotracheal tubes, and often, poor adaptation to mechanical ventilation and a lack of cooperation with the medical personnel.[8,9]

Definition

According to the DSM III-R, the diagnostic criteria of ACS are as follows:

• Reduced ability to focus the attention on external stimuli (e.g.: questions have to be repeated because of wavering attention) and to appropriately shift the attention in response to new stimuli (e.g.: the patient keeps answering the previous question).

• Disorganized thoughts, manifested as disjointed, irrelevant or incoherent speech.

• At least two of the following:

a) reduced level of wakefulness, for example, difficulty staying awake during the observation.

b) disturbances in perception: errors of interpretation, delusions or hallucinations.

c) disturbances in the waking-sleeping cycle, with insomnia and daytime drowsiness.

d) increased or decreased psychomotor activity.

e) disorientation in relation to space, time and people.

f) memory problems, for example, inability to learn new things, such as the names of several unrelated objects, after five minutes, or to recall past events, such as the history of the present disease.

• The symptoms appear within a short space of time (generally hours or days) and tend to fluctuate throughout the day.

• One of the following (a or b):

a) data from the history, physical examination or laboratory tests, showing one or more specific organic factors that are believed to have caused the mental disturbance.

b) in the absence of such data, the existence of an organic etiological factor can be assumed, if the disturbance is not attributable to a non organic mental disease, for example a manic episode, as the cause of agitation and sleep disturbance.

Neurobiology

ACS appears to be the result of a dysfunction in the dopaminergic and acetylcholinergic systems of the brain.[10,11]

Various studies have demonstrated that the use of anticholinergic drugs can cause cognitive dysfunction, and slow down the EEG in Humans.[10] It is not rare, for example, to see patients with ACS resulting from the use of tricyclic antidepressants. Studies on primates also show that scopolamine causes a slowing down of the EEG and inability to learn.[10]

On the other hand, it appears that dysfunction of the mesocortical limbic dopaminergic system leads to the increased agitation and hallucinations seen in patients with A.C.S.[10] Dopamine controls the processes of association and learning which, if over-accelerated by an excess of dopamine, can lead to delirium and agitation. Indirect evidence of the fact that excessive dopamine produces these symptoms are the efficacy of haloperidol, a dopaminergic blocker, in controlling agitation and delirium. In addition, some drugs with central dopaminergic effect, like amphetamines and cocaine, cause anxiety, agitation, panic, hypervigilance and paranoia.

In short, ACS may result from disruption of the neurotransmitter systems: excessive dopamine and/or acetylcholine deficit. The agitation that often accompanies severe ACS is generally associated with the activation of the noradrenergic axis. This activation causes insomnia, panic, hypervigilance, and autonomic hyperactivity. The increase in levels of catecholamines leads to an increase in arterial pressure, heart rate and respiratory frequency, affecting the metabolism at neurohumoural or musculoskeletal levels. Muscle hypermetabolism produces metabolic acidosis, which

potentiates the occurrence of cardiac arrhythmias and an imbalance in oxygen distribution to the tissues. Muscle hyperactivity can lead to rhabdomyolysis with myoglobinuria and renal insufficiency.[6]

The acute organic responses to physical and psychological stress represent essential adaptive responses for survival, in a potentially harmful and uncertain environment. These responses can, however, occur in patients hospitalized in the ICU, and in these cases, they act to the patient's detriment, and need to be blocked as selectively and fully as possible.

Incidence

The frequency of ACS depends on the nature and severity of the primary disease, the type of treatment, the type of ICU and the criteria used for its diagnosis.

It occurs with higher frequency in surgical ICUs, followed, in decreasing order, by medical ICUs, coronary ICUs, medical wards, and surgical wards.[10,12,13]

The incidence of ACS following surgical intervention, for example, is no more than 0.1%, while in the elderly, this percentage rises to 10% or 15%.[8]

Etiology

May organic disturbances have been considered as factors that cause ACS According to Lipowski, the majority of these disturbances can be grouped into the following classes: a) primary intercranial disease b) systemic diseases with secondary effect on the brain; c) exogenic toxic agents; d) drug withdrawal.

Differential diagnosis

ACS should be distinguished from functional psychoses, secondary mania, dementia, complex partial crises and psychogenic dissociative disturbances.[8,12,13]

In functional psychoses there are no variations in wakefulness, and there is generally a previous history of psychotic symptoms and clearly systematized episodes of delirium. In ACS, the episodes of delirium are unstructured, and when hallucinations occur, these are generally visible, tactile or kinesthetic. Mood disturbances are often associated with A.C.S., which can make it difficult or impossible to diagnosis affective disturbances (depression, manic depressive disturbance, secondary mania) in the presence of ACS.

In the presence of mania in an ICU patient, an organic or toxic cause should be ruled out, such as medications (corticoids, isoniazid, levodopa), metabolic disturbances, infection, neoplasia of the SNC and right temporal lobe epilepsy.

The previous history is essential for distinguishing between dementia and ACS, since both are characterized by a global cognitive dysfunction. In practice, ACS often appears alongside dementia, causing a deterioration that is added to the patient's existing cognitive impairment.

Complex partial crises coming from foci in the limbic system can produce abnormal psychic and behavioral phenomena that can be confused with ACS. The EEG can be decisive in this situation.

TABLE III

Numerous medications have been associated with ACS The list below shows the main ones:

Class	Agent
Anesthetics	All
Anticholinergics	Atropine sulphate
Anticonvulsants	Barbiturates
	Carbamazepine
	Phenytoin
Non-selective	
Aantihistamines	Diphenhydramine
	Promethazine
H2 blockers	Cimetidine
	Ranitidine
Benzodiazepines	All
Cardiac agents	
Antiarrhythmics	Lidocaine
β blockers	Propranolol
	Metoprolol
Cardiac glycosides	Digitalics
Corticosteroids	All
Narcotics	All
Antibiotics	Penicillin
	Rifampicin
	Imipenem
Antidepressants	Tricyclics

Adapted from McCartney, J.R.; Boland, R.J. – Understanding and managing behavioral disturbances in the ICU – The Journal of Critical Illness, Vol. 8, No. 1, January 1993.

Although rare in the ICU, dissociative psychogenic states may resemble ACS. Patients with psychogenic amnesia often display a global cognitive dysfunction; however, the deficits are invariably inconsistent, and include the patient's inability to identify themselves (this inability that is generally not present in ACS and in transitory global amnesia).

EEG is a particularly sensitive method for the diagnosis of ACS, although it is non-specific. In the presence of ACS, EEG is diffuse and generally slow, and the base heart rate usually decreases with the severity of the ACS. In some particular cases, like *delirium tremens* and atropine poisoning, rapid heart rate may occur. Electroencephalographic alterations generally precede the clinical manifestations of ACS, and often continue for some time after the resolution of the symptoms. EEG is generally normal in functional psychoses and dissociative psychogenic disorders.

Treatment

The treatment of ACS is based on the following principles:[3,4,17]

A) correcting the metabolic and systemic alterations;
B) eliminating the toxicity of the drugs;
C) treating the withdrawal;
D) use of neuroleptic medication.

A) Correcting the metabolic and systemic alterations: Meticulous examination of the clinical situation and analyses of the patient are the first steps in the attempt to find a specific anomaly that can be selectively treated. By way of example, some more common situations can be mentioned: treating infections, maintaining normal perfusion pressure, correcting electrolytic disturbances, maintaining normal blood volume, and adequate oxygenation of the blood

B) Eliminating the toxicity of the drugs: When there is a strong suspicion of drug toxicity, the administration of the drug responsible for the ACS should be suspended or reduced, or where possible, a specific antidote used. Narcotics and agents with anticholinergic properties are among the drugs most commonly associated with ACS in the ICU. Physostigmine can be used at a dose of 1 to 2 mg IV in slow administration, or in continuous infusion, to revert the ACS associated with anticholinergic agents.[8] Naloxone at a dose of 0.4 mg IV can be used in the treatment of ACS resulting from the ingestion of narcotics, and it is sometimes necessary to repeat this dose to reverse the effects of narcotics which have a long half-life.[17] Flumazenil at doses of 1 to 2 mg IV has been successfully used to revert ACS resulting from ingestion of benzodiazepines. It is sometimes necessary to use flumazenil in continuous infusion at a dose of 0.4 mg to 1 mg per hour, due to its short-lived effect.[8]

C) Treating the withdrawal: The diagnosis of ACS caused by withdrawal presupposes a high level of suspicion, for various reasons. Firstly, the urgent nature of admissions to the ICU means the sudden interruption of drug abuse. Secondly, ICU patients are often incapable of communicating effectively, which makes it impossible to gather data on the history of abuse of a substance. Thirdly, the clinical signs of withdrawal, such as fever, tremor and other signs of sympathetic hyperactivity, are non-specific and are often present for other reasons. Finally, there are no laboratory tests that can confirm a diagnosis of withdrawal. The clinical exam can sometimes provide some indications, such as the presence of signs of needle injection or stigma of chronic alcohol abuse.

ACS arising from privation is invariably associated with intense agitation, terrorizing visual hallucinations and manifestations of sympathetic hyperactivity, such as fever, tremor, tachycardia, tachypnoea and mydriasis. The substances most commonly named in withdrawal are alcohol, sedative-hypnotic agents and opiates. The general principle of treatment of withdrawal consists in administering a substance with the same mechanism of action as the substance of the abuse. When the dysautonomic manifestations are marked, clonidine has been used with good results, at doses of 0.150 mg to 0.300 mg, divided into two administrations. The cardiovascular effects of this medication should be carefully monitored, as undesirable hypotension may occur.[8]

Alcohol – Benzodiazepines are the treatment of choice for all phases of alcohol withdrawal. It is believed that its effectiveness, in this situation, is due to its gabaminergic effect, which is similar to that of alcohol.[6,15] Effectively, benzodiazepines act on the central nervous system, through the binding of the benzodiazepine receptors that are part of the GABA (A) receptors. By binding to these receptors, benzodiazepines facilitate the effect of the GABA, which promotes the opening of the chlorine channels, stimulating hyperpolarization of the nerve cell and making it more resistant to

excitation. Diazepam, administered via intravenous, is the benzodiazepine of choice among us, and the dose varies from one case to another. Some patients respond to doses of 50 mg in 24 hours, while others require doses of more than 800 mg in the same period of time.[8] In patients with hepatic insufficiency, high serum levels of diazepam or its metabolite, nordiazepam, may occur. In this situation, the use is justified of another benzodiazepine, which has short half-life, or which suffers little hepatic metabolization, such as midazolam and lorazepam, respectively. If this therapy is unsuccessful, paraldehyde may be used.[8] In our experience, concomitant use of haloperidol, in continuous infusion, at a dose of 30 to 150 mg/24 hours, has been useful in the treatment of alcohol withdrawal, particularly as a therapeutic adjuvant in cases of motor agitation and hallucinatory syndrome.

Sedative-hypnotic agents – The sudden interruption of consumption of these agents can cause ACS, the initial severity of which will depend on the pharmacokinetic properties of the agent in question.[8] Generally, the interval between interruption of the drug and the start of withdrawal depends on the half-life of the drug. Diazepam withdrawal, for example, may occur only at the end of a week, due to the long half-life of this compound and its active metabolites. On the other hand, alprazolam, oxazepam and lorazepam withdrawal may occur within several hours of the last administration. Withdrawal from barbiturates is potentially a very serous situation, and if it is not recognized and treated, can cause worsening of the ACS, convulsions, and even death. The treatment consists of the administration of an agent of the same pharmacological family, but with a longer half-life, which enables a slow, gradual weaning. Another situation in which careful attention to the possible occurrence of withdrawal syndrome in ICU patients is required, is in cases where sedatives, like benzodiazepines or barbiturates, are administered in continuous infusion.

Opiates – Withdrawal from opiates does not generally cause major confusion syndrome. The treatment consists of replacement of the opiate by narcotics. Methadone is the drug generally used for this purpose, due to its long half-life, although it is not widely used in general hospitals. The therapeutic regimen that we use consists of the association with clonidine, an anti-spasmodic, chlorpromazine, at a dose of 25 to 50 mg every 6 hours, and a benzodiazepine.

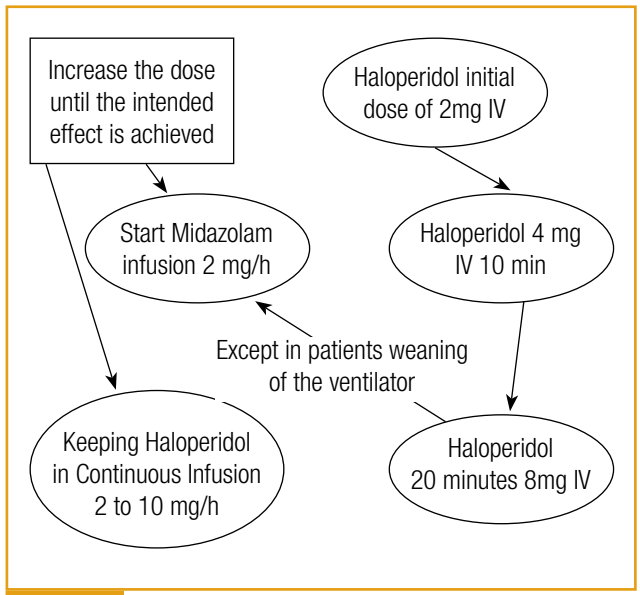

FIG. 1

D) Pharmacological treatment of non-specific ACS: When a specific cause of ACS is not found, or cannot be corrected, a neuroleptic agent like haloperidol is recommended.[6,7,8,15] Haloperidol is a highly potent neuroleptic butyrophenone, which acts by blocking the dopamine receptors of the Central Nervous System.

It has negligible effects on cardiovascular and respiratory function, and hypotension has only been recorded in hypovolaemic patients. Unlike other neuroleptics, it is rarely associated with convulsions, and the extrapyramidal effects are minimal when it is administered by the intravenous route.[6,15] The combination of benzodiazepines is very well-tolerated, and appears to be very effective in the treatment of ACS. Haloperidol should be given at an initial dose of 2 mg IV bolus, which can be increased to double every 10 minutes, until the desired effect is obtained.[6] Many authors associate midazolam in continuous infusion when the first 14 mg of haloperidol are ineffective (*Fig. 1*), but only in patients who are not in the process of being taken off the ventilator.[6] When it is possible to calm the patient, the administration of haloperidol should be maintained, in continuous infusion, and it is generally possible to reduce the dose. The dose and pace of administration should be adjusted, according to the effect on the patient. This medication is maintained for as long as the cause or the circumstances that led to the ACS persist. Although experience shows that the sudden interruption of

TABLE IV

Causes of sleep deprivation in the ICU

PATIENT FACTORS
SEVERITY OF THE DISEASE
MEDICATIONS
Drugs that suppress REM sleep
Narcotics
Barbiturates
Antidepressants
Drugs that do not suppress REM sleep
Benzodiazepines
FEVER
PAIN
LOSS OF CONTROL (restrictions, treatments)
FEAR, ANXIETY AND STRESS
FACTORS RELATED TO THE MEDICAL PERSONNEL
DIAGNOSTIC TESTS
NURSING INTERVENTIONS
INVASIVE PROCEDURES
ENVIRONMENTAL FACTORS
LIGHTS
NOISE
Mechanical devises, including ventilators and alarms (45-76 dB)
Background noise (55-72 dB)
Respirator or nursing care (55-83 dB)
Conversation between the medical personnel (60-74 dB)
UNPLEASANT SMELLS

Adapted from Schwab, R.J. – Disturbances of sleep in the Intensive Care Unit – in Critical Care Clinics.

the administration of haloperidol has no unpleasant consequences, it is prudent to gradually decrease the dose, to avoid the reappearance of the ACS or the appearance of withdrawal dyskinesias. This treatment regimen is adequate for patients without respiratory difficulty, and for those who will be connected to a ventilator. In patients connected to a ventilator, but who are being gradually removed from the assisted ventilation, medications with a myorelaxant effect, such as benzodiazepines, should not be used. Therefore, in these cases, the medical treatment of ACS should be based on haloperidol alone.

Other factors that contribute to the genesis of ACS

A) Sleep deprivation: Numerous studies have shown that sleep deprivation in healthy individuals for 2 to 5 days causes irritability, disorientation, slow speech, and sometimes, hallucinations and paranoia.[5] These symptoms are similar in some ways to those manifested by patients in an ICU, when presenting ACS, which has led some authors to state that sleep deprivation is the main cause of ACS in the ICU.[5] Currently, there appears to be a general consensus that sleep deprivation contributes to the onset of ACS, but it is rarely the main cause. There are various reasons for the occurrence of sleep deprivation in the ICU *(Table 4)*. The main one, according to the majority of studies, is noise.

Thus, the first actions to be taken in the prevention of sleep deprivation are those that seek to reduce noise in the ICU, particularly at night. Another measure that is equally important is trying to maintain day/night cycles, and favoring natural light over artificial light. Often it becomes necessary to use hypnotic medications, and the choice is generally benzodiazepines. These medications are almost always hypnotics, but unfortunately, they suppress phases 3 and 4 of sleep, and have other secondary actions that are often prejudicial to the patient's treatment. Recently, a new hypnotic medication called zolpidem was introduced to the market, which despite being unrelated to benzodiazepines, selectively binds only to one of the benzodiazepine receptors in the brain (type 1 receptor). For this reason, it does not have myorelaxant, anticonvulsant or anxiolytic action, and it does not cause anterograde amnesia as other benzodiazepines do. Its most important characteristic is that it does not suppress any of the phases of sleep, as well as the fact that its effects do not continue into the next day.[5]

If these drugs are not effective, or side effects occur, thioridazine may be used as an alternative, at a dose of 10 to 40 mg, or cloral hydrate at a dose of 60 mg.

Hypnotics should only be given after the hour of silence, and when the lights are low. Patients who receive haloperidol with therapeutic action in the initial phases of ACS generally do not require hypnotic agents.

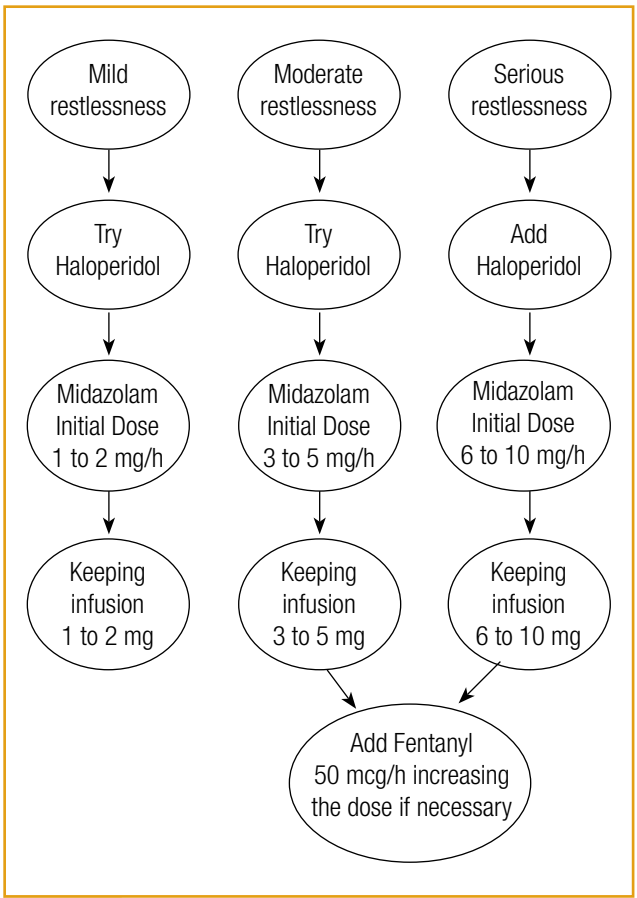

FIG. 2

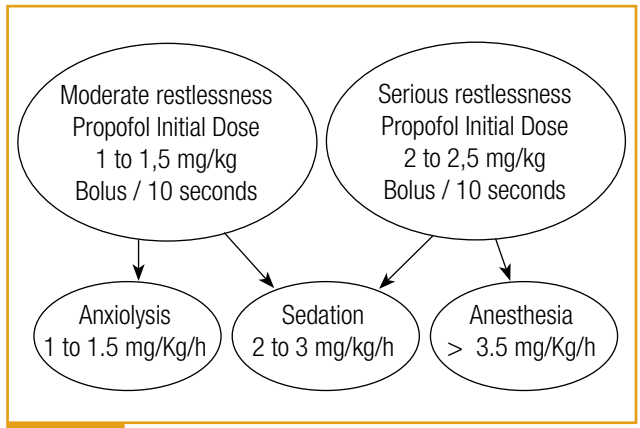

FIG. 3

B) Dementia: Nowadays, the admission of elderly patients to the ICU is increasingly common. Some present global cognitive deficits of greater or lesser importance, and when subject to a systemic disease, and the environmental stress of admission to an ICU, these cognitive deficits often become more acute, and they may present a range of symptoms that are indistinguishable from ACS. We can say, perhaps, that the limit of tolerance to cerebral noxae is lower in this patient population, but that acute confusional states are more common.

C) Personality, fear and anxiety are other factors involved in the genesis of ACS, although these factors alone are more frequently associated with agitation, defined by the American Psychiatric Association as "excessive motor activity, generally without purpose and associated with nervous tension". Individuals with so-called type A personality, for example, can adapt poorly to a situation where they have no control or decision-making power over the therapeutic measures to which they are submitted. On the other hand, individuals with dependant personalities find it easier to accept being in the ICU, but become agitated and anxious when told that they will be transferred to the normal ward, where the care and attention are less intensive.[1,16,17]

Fear and anxiety are natural reactions in ICU patients, but when exacerbated, due to the patient's personality or the characteristics of the ICU and the disease, they can lead to great agitation and panic. It is very important to recognize these factors early on, and intervene with pharmacological and non-pharmacological measures. The pharmacological treatment of choice in cases of acute agitation is the use of sedatives, like benzodiazepines, in doses that are adjusted for each individual case (*Fig. 2*).[9,17] Alternatively, in patients in the process of being taken off ventilation, it is preferable to use haloperidol, as benzodiazepines have a marked myorelaxant effect. In cases of severe agitation, propofol may be used which is a fast-acting anesthetic agent with a very short half-life (*Fig. 3*).[6,9] In cases of agitation where pain appears to be the determining factor, fentanyl should be used, in a single dose of 50 to 300 mcg, in intravenous bolus, followed by continuous infusion at a dose of 50 to 300 mcg/hour.[18]

Non-pharmacological treatment of ACS

Various circumstances inherent to the ICU can also contribute to the genesis of ACS. A range of actions is therefore justified, aimed at minimizing the conducive factors and behavioral alterations in ICU patients.[4] Some of these measures are shown in *Table 5*.

TABLE V

Objective	Methods
Improve cognitive function	Reorientate frequently; place a clock, calendar, television and/or radio in the room; give clarifications and explanations
Maximize comfort	Control the pain adequately, mobilize the patient, allow them to rest, respect their sleep.
Give support and confidence	Transmit empathy and encouragement, allow ventilation
Decrease environmental stress	Give pre-surgery education, offer sensorial stimulation, limit the noise of alarms and equipment, preserve the day-night cycles
Increase communication between the patient, the family and the medical personnel	Use written communication or signs if the patient is intubated, and encourage the family to visit
Avoid physical self-harm and aggression towards the personnel	Use the minimum restriction

Summary

ACS in ICU patients is characterized by:
• Changes in wakefulness;
• Disturbance of the waking-sleeping cycl;
• Attention and concentration deficits;
• Disorganized thoughts (incoherent speech);
• Disturbances in perception (delusions, hallucinations);
• Disorientation in relation to time, space or people alterations in psychomotor behavior (agitation or decrease in psychomotor activity);
• Memory problems.

Designating this situation ICU psychosis is inadequate, because: It is not a true psychosis; the mental alteration is not a direct consequence of hospitalization in the ICU; and it does not make a clinical distinction with situations observed in other types of ward.

The main causes of ACS in the ICU are: metabolic/systemic diseases and disorders; exogenic toxic agents; withdrawal from substance abuse; and primarily intercranial diseases.

Often, other important factors coexist, such as: sleep deprivation, previous cognitive deficits, fear, anxiety and the patient's personality.

The treatment includes: correction of metabolic/systemic disturbances; suspension of toxics; treatment of the withdrawal; use of haloperidol with or without benzodiazepines; and non-pharmacological measures to reduce environmental stress and promote physical and mental well-being. ■

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