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Magnesium in Intensive Care Unit: A Review

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ABSTRACT

Aim of the work: there is an increased interest in the role of magnesium ions in clinical medicine, nutrition and physiology. Magnesium affects many cellular functions, including transport of potassium and calcium ions and modulates signal transduction, energy metabolism and cell proliferation. Magnesium deficiency is not uncommon among the general population: its intake has decreased over the years. Magnesium derangement results in various symptoms and signs; magnesium supplementation or intravenous infusion may be beneficial in various diseased states; so this review aimed to highlight the physiology of magnesium in humans, the derangement of magnesium in the form of hyper and hypomagnesemia , their clinical picture and the clinical and therapeutic uses of magnesium in the critical ill patients .

Methods: references were obtained from Medline, Google Scholar and Ovid from 1960 to 2017. All categories of articles (clinical trials, reviews, or metaanalyses) on this topic were selected.

Conclusion: magnesium is a critical physiological ion; it has many known indications in anesthesiology and intensive care because of its interactions with drugs used in intensive care. Intensive care specialists need to have a clear understanding of the role of this important cation. Magnesium is gaining recognition as a clinically important electrolyte in intensive care and emergency medicine .Recent clinical trials and case reports increase interest of magnesium as an effective therapeutic agent for potentially life-threatening problems such as torsade de pointes, digitalis toxicity, bronchospasm and alcohol withdrawal, subarachnoid hemorrhage, acute myocardial infarction, preecalmsia , eclamsia ,hypertension , diabetes , metabolic syndrome and cardiac arrhthymias .

Keywords: physiology of magnesium, hyper and hypomagnesemia , magnesium in ICU

INTRODUCTION

There is an increased interest in the role of magnesium ions in clinical medicine, nutrition and physiology. Magnesium affects many cellular functions including: transport of potassium and calcium ions and modulates signal transduction, energy metabolism and cell proliferation. Magnesium deficiency is not uncommon among the general population; its intake has decreased over the years especially. The magnesium supplementation or intravenous infusion may be beneficial in various diseased states [(1)]. The Mg ion, the second most abundant intracellular cation after potassium, plays essential roles in the structure and function of the human body; it is an essential cofactor in a wide variety in the physiological processes , including protein synthesis and stability, neuromuscular excitability and the conduction of neural impulses, stimulus-contraction coupling and muscular contraction, magnesium is an indispensable part of the activated Mg ATP complex and it is required for adenosine triphosphate (ATP) synthesis in the mitochondria .Also, magnesium is an important cofactor in over 300 enzymatic reactions and it is required for the activity of all rate limiting glycolytic enzymes, protein kinases and more generally , all ATP and phosphate trans-associated enzymes; magnesium may also bind the enzymes directly (i.e RNA and DNA polymerases) and alter their structure therefore , the availability of an adequate quantity Mg may be considered as a critical factor for normal body and cellular homeostasis and function [(2)] .Magnesium is a critical physiological ion and its deficiency might contribute to the development of pre-eclampsia to impair neonatal development and to metabolic problems extending into adult life. Pharmacologically, magnesium is a calcium antagonist with substantial vasodilator properties but without myocardial depression. Cardiac output usually increases following magnesium administration, compensating for the vasodilatation and minimizing hypotension.

Neurologically, the inhibition of calcium channels and antagonism of the N-methyl-D-aspartic acid (NMDA) receptor raises the possibility of neuronal protection. It is the first-line anticonvulsant for the management of preeclampsia and eclampsia and it should be administered to all patients with severe preeclampsia or eclampsia [(3)].

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Physiology of magnesium

About 99% of the total body magnesium is located in bone, muscles and non-muscular soft tissue. Approximately 50–60% of magnesium resides as surface substituents of the hydroxyapatite mineral component of bone. Most of the remaining magnesium is contained in skeletal muscle and soft tissues. The magnesium content of bone decreases with age and its store in this way is not completely bioavailable during magnesium deprivation. Bone provides a large exchangeable pool to buffer acute changes in serum magnesium concentration. Overall, one third of skeletal magnesium is exchangeable, serving as a reservoir for maintaining physiological extracellular magnesium levels. Intracellular magnesium concentrations range from 5 to 20 mmol/L; 5% is ionized , the remainder is bound to proteins, negatively charged molecules and adenosine triphosphate (ATP) [(4)]. Extracellular magnesium accounts for ~1% of total body magnesium which is primarily found in serum and red blood cells (RBCs). Serum magnesium can -just like calcium- be categorized into three fractions. It is either free / ionized, bound to protein or complexes with anions such as phosphate, bicarbonate and citrate or sulphate. Ionized magnesium has the greatest biological activity [(5)] .Magnesium is primarily found within the cell where it acts as a counter ion for the energy-rich ATP and nuclear acids. Magnesium is a cofactor in >300 enzymatic reactions. Magnesium critically stabilizes enzymes, including many ATP-generating reactions. ATP is required universally for glucose utilization, synthesis of fat, proteins, nucleic acids and coenzymes, muscle contraction, methyl group transfer and many other processes. Thus, one should keep in mind that ATP metabolism, muscle contraction and relaxation, normal neurological function and release of neurotransmitters are all magnesium dependent. It is also important to note that magnesium contributes to the regulation of vascular tone, heart rhythm, platelet-activated thrombosis and bone formation [(6).] In muscle contraction, for example, magnesium stimulates calcium re-uptake by the calcium-activated ATPase of the sarcoplasmic reticulum. Magnesium also modulates insulin signal transduction and cell proliferation and it is important for cell adhesion and trans-membrane transporting including transport of potassium and calcium ions. It also maintains the conformation of nucleic acids and it is essential for the structural function of proteins and mitochondria [(7)] . Magnesium homeostasis is maintained by the intestine, the bone and the kidneys [(8)] . Magnesium just like calcium is absorbed in the gut and stored in bone mineral and excess magnesium is excreted by the kidneys and the feces. Magnesium is mainly absorbed in the small intestine although it is also taken up via the large intestine. Two transport systems for magnesium in the gut are known [(9)]. The majority of magnesium is absorbed in the small intestine by a passive paracellular mechanism, which is driven by an electrochemical gradient and solvent drag [(10)] . Of the total dietary magnesium consumed, only about 24:76% is absorbed in the gut and the rest is eliminated in the feces. Intestinal absorption is not directly proportional to magnesium intake, but is dependent mainly on magnesium status. The lower the magnesium level, the more of this element is absorbed in the gut, thus relative magnesium absorption is high when intake is low and vice versa. When intestinal magnesium concentration is low, active trans-cellular transport prevails, primarily in the distal small intestine and the colon[(9)].

Dysmagnesemia, clinical picture and management

Normal plasma magnesium concentration is 1.8 to 2.3 mg/dL (0.75 to 0.95 mmol/L, 1.5 to 1.9 mEq/L). Magnesium exists in three states: ionized magnesium (60% of total magnesium), protein bound (30%, mostly albumin) and complexes to serum anions (10%). Attempts to predict the ionized magnesium concentration from changes in albumin or pH have generally been unsuccessful. Currently, the clinical role of measuring ionized Mg^{2+} is unclear, and, unlike ionized calcium, measurement is not standard practice [(11)]. Clinical squealae of altered magnesium content is more dependent on tissue magnesium levels rather than blood magnesium concentration. It is often difficult to consistently correlate symptoms to specific plasma magnesium levels [(12)].

Hypomagnesemia is common. It was found in 10% (Mg^{2+} <1.6 mg/dL) of patients admitted to a geriatric facility. When present, hypomagnesemia is usually undetected. In a prospective study, 47% of patients undergoing clinical blood testing for electrolyte concentrations had hypomagnesemia. Among ICU patients, the prevalence of hypomagnesemia arranges from 11% to 65% [(13)].

Clinical sequelae of hypomagnesemia

Determining the clinical consequences of isolated hypomagnesemia is difficult because patients with hypomagnesemia typically also have hypokalemia, hypocalcemia, and hyponatremia. A reasonable approach used in numerous case reports is to attribute symptoms to low magnesium levels only after the symptoms are resistant to the correction of other electrolyte abnormalities and abolished following magnesium repletion. Symptoms due to hypomagnesemia have been reported at modest degrees of depletion, but in general symptoms become more common as serum magnesium fall below1.2 mg/dL [(14)].

Table 3. Manifestations of altered serum magnesium concentrations [15]

Manifestation	Magnesium level		
	mmol/L	mEq/L	mg/dL
Tetany ,Seizures or arrhythmias	<0.5	<1	<1.2
Neuromuscular irritability, Hypocalcemia or Hypokalemia	0.5–0.75	1.0–1.5	1.2–1.8
Normal magnesium level	0.75–1.05	1.5–2.1	1.8–2.5
Typically asymptomatic	1.05–2.1	2.1–4.2	2.5–5.0
Lethargy, Drowsiness, Flushing or diminished deep tendon reflex	2.1–2.9	4.2–5	5.0–7.0
Somnolence , loss of deep tendon reflexes , hypotension or ECG changes	2.9–5	5.8–10	7.0–12
Complete heart block, Cardiac arrest , Apnea , Paralysis or Coma	>5	>10	>12

Treatment of hypomagnesemia

Patients with symptomatic hypomagnesemia should be treated with intravenous magnesium. The most common formulation Is MgSO4. 1 gram of Magnesium sulfate contains 0.1 grams of elemental magnesium. No trials have been done to determine the optimal regimen for magnesium replacement, but consensus statements suggest administration of 8 to 12 g of magnesium sulfate in the first 24 hours followed by 4 to 6 g per day for 3 or 4 days to replete body stores. [12] .The American College of Cardiology and the American Heart Association recommend 1 to 2 grams of magnesium sulfate as an IV bolus over five minutes for torsades de pointes therapy. Emerging data suggests that magnesium may also have a role in reducing reperfusion injury and decreasing infarct size in acute myocardial infarction (AMI). Animal studies in multiple species consistently show a decrease in infarct size in subjects given magnesium prior to reperfusion. It appears that the benefits of magnesium are nearly eliminated by administration of magnesium after reperfusion, indicating that magnesium may specifically prevent reperfusion injury[16] . Magnesium replacement should be done cautiously in patients with renal insufficiency. Recommendations vary between 50 to 75% dose reductions. Patients should be monitored closely during infusions for decreased deep tendon reflexes and magnesium levels should be checked at regular intervals. Oral supplementation has been shown to successfully correct increased magnesium retention. In a double blind study of patients with normal serum Mg^{2+} but Mg^{2+} retention of >25%, administration of 360 mg of elemental magnesium per day (divided tid) lowered the magnesium retention to less than 10%. Lower dose magnesium repletion has been shown to be ineffective [17].

Hypermagnesemia

Normally the kidney excretes only 2–4% of the filtered magnesium, but it is capable of increasing fractional excretion to nearly 100% in the face of decreased GFR or increased serum magnesium levels .Because of this reserve of magnesium excretion potential, hypermagnesemia is rarely seen [18].

Clinical manifestations of hypermagnesemia

Neuromuscular toxicity . Magnesium can block synaptic transmission of nerve impulses. Hypermagnesemia causes the initial loss of deep tendon reflexes and may lead to flaccid paralysis and apnea[19]. Neuromuscular toxicity also effects smooth muscle, resulting in ileus and urinary retention. In cases of oral intoxication, the development of ileus can slow intestinal transit times, further increasing absorption of magnesium [20]. Hypermagnesemia has also been reported to cause a parasympathetic blockade resulting in fixed and dilated pupils, mimicking brainstem herniation. Other neurologic signs include lethargy, confusion and coma [21].

CV toxicity

Magnesium acts as a calcium channel blocker and in cardiac tissue also blocks potassium channels needed for repolarization. Cardiac manifestations of hypermagnesemia initially include bradycardia and hypotension. Higher magnesium levels cause PR interval prolongation, increased QRS duration and prolonged QT interval [22]. Extreme cases can result in complete heart block or cardiac arrest. One case of ventricular fibrillation has been reported with a Mg^{2+} level of 9.7 mg/dL[23]

Metabolic disturbances

Hyperkalemia has variably been reported in hypermagnesemic case series. This may be due to decreased renal potassium clearance from blockade of potassium channels in the apical membranes of principal cells in the cortical collecting duct. In addition, multiple studies have found suppression of plasma renin activity in pregnant patients being treated with magnesium sulfate [(24)].

Treatment of hypermagnesemia

The first principle of treatment is prevention. Patients with renal insufficiency should not be given magnesium containing antacids or cathartics. In cases of hypermagnesemia, stopping the infusion or supply of magnesium will allow patients with intact renal function to recover. Calcium salts can reverse hypotension and respiratory depression [(25.] Patients are typically given 100 to 200 mg of elemental calcium intravenously over 5 to 10 minutes. To speed the renal clearance of magnesium, loop diuretics and saline diuresis are intuitive options, though we could find no literature or evidence to support their use. Clark and colleagues suggests adding calcium gluconate (15 mg/kg over 4 hours) to augment renal clearance of magnesium [(25)]. In patients with severe renal dysfunction, dialysis offers a way to rapidly clear magnesium. Both peritoneal and hemodialysis are effective at lowering magnesium levels. In one case series of 43 hypermagnesemic patients, 5 were dialyzed. Two patients received peritoneal and both died. Three patients received hemodialysis and two survived [(26)].

Clinical and therapeutic uses of magnesium in intensive care

Magnesium has many known indications in anesthesiology and intensive care. Because of its interactions with drugs used in intensive care. Intensive care specialists need to have a clear understanding of the role of this important cation. Magnesium is gaining recognition as a clinically important electrolyte in intensive care and emergency medicine [(27)].The fact that hypomagnesemia is frequent postoperatively and in the intensive care and needs to be detected and corrected to prevent increased morbidity and mortality. Magnesium reduces catecholamine release and thus allows better control of adrenergic response during intubation or pheochromocytoma surgery. It also decreases the frequency of post operative rhythm disorders in cardiac surgery as well as convulsive seizures in preeclampsia and their recurrence in eclampsia. The use of adjuvant magnesium during perioperative analgesia may be beneficial for its antagonist effects on N-methyl-D-aspartate receptors. The precise role of magnesium in the treatment of asthmatic attacks and myocardial infarction in emergency conditions needs to be determined [(28).]

Magnesium Sulfate for the Treatment of Eclampsia

The effect of magnesium sulfate in the prevention of eclampsia is likely multi-factorial. Magnesium sulfate may act as a vasodilator, with actions in the peripheral vasculature or the cerebrovasculature, to decrease peripheral vascular resistance or relieve vasoconstriction. Additionally, magnesium sulfate may also protect the blood-brain barrier and limit cerebral edema formation, or it may act through a central anticonvulsant action [(29)] . Empirical evidence supports the effectiveness of MgSO4 in preventing and treating eclamptic seizures. For eclamptic seizure prophylaxis in preeclamptic women, MgSO4 is superior to phenytoin, nimodipine, diazepam and placebo [30).] Although the effectiveness of MgSO4 in treating and preventing eclampsia has been established, questions still exist as to its safety. There are concerns regarding the possibility of hypermagnesemia toxicity in eclampsia treatment. Normal serum concentrations of Mg^{2+} are 1.5 to 2.5 mEq/L (1.8 to 3.0 mg/dL), with one-third to one-half bound to plasma proteins[(31)].Total magnesium serum concentrations advocated for the treatment of eclamptic convulsions are 3.5 to 7 mEq/L (4.2 to 8.4 mg/dL), which can be obtained by administering it intramuscularly (6 g loading dose followed by 2 g/h), intravenously (2 to 4 g dose up to 1 g/h), or a combination of both. Areflexia, particularly loss of the patellar deep tendon reflex, has been observed at 8 to 10 mEq/L and respiratory paralysis seen at 13mEq/L.Progressively higher serum magnesium levels can ultimately lead to cardiac arrest [(32)].

Anticonvulsant activity of magnesium in eclampsia

Seizures are thought to be mediated at least in part by stimulation of glutamate receptors, such as the NMDA receptor. In addition, systemic treatment with MgSO4 causes a significant reduction in the NMDA receptor binding capacity in the brain [(32)]. MgSO4 has been shown to be an effective treatment option for the prevention of eclampsia. Its mechanism of action is likely multi-factorial, encompassing both vascular and neurological mechanisms. Being a calcium antagonist, its effect on vascular smooth muscle to promote relaxation and vasodilation may have a role in lowering total peripheral vascular resistance. In addition, MgSO4 may have an effect on the cerebral endothelium to limit vasogenic edema by decreasing stress fiber contraction and

paracellular permeability via calcium-dependent second messenger systems such as MLC kinase. Lastly, MgSO4 may also act centrally to inhibit NMDA receptors, providing anticonvulsant activity by increasing the seizure threshold. A more complete understanding of the effects of MgSO4 will likely promote safer and more effective treatments of eclampsia[29].

Potentials of magnesium treatment in subarachnoid hemorrhage

Subarachnoid hemorrhage accounts for only 3 % of all strokes. It occurs at a younger age and carries a worse prognosis than other types of stroke. The clinical hallmark of subarachnoid hemorrhage (SAH), mostly caused by a rupture of an intracranial aneurysm, is a history of unusually severe headache that started suddenly. Because of the young age at which SAH occurs and its poor prognosis, the loss of productive life years from SAH is as large as that from ischaemic stroke, the most frequent subtype of stroke [35]. Magnesium inhibits many agonists of platelet aggregation and adhesion, like thromboxane A_2 and beta-thromboglobin, most probably due to inhibition of intracellular Ca^{2+} mobilisation . As a consequence, magnesium inhibits platelet aggregation and the platelet-dependent thrombus formation and this effect is independent of, and additive to, that of aspirin . Magnesium stimulates synthesis and release of the potent vasodilator prostacyclin and has a vasorelaxing effect in prostaglandin induced vasospasm [36].

Hypomagnesemia in subarachnoid hemorrhage

The elevated levels of catecholamines after SAH may play a mediating role in the increased intracellular shift of Mg^{2+}. Catecholamines stimulate lipolysis through the β2-receptor with liberation of free fatty acids. This leads to an intracellular deposit of Mg^{2+} as an insoluble soap. The consequent decrease of intracellular Mg^{2+} may lead to Mg^{2+} influx. It has been shown that adrenaline causes a rapid fall in the plasma magnesium concentrations [37]. Another possible cause for the increased intracellular shift is the glutamate-stimulated Mg^{2+} influx by NMDA activated ion channels, which takes place in the absence of extracellular Na^{+} and Ca^{2+}. Intracellular Mg^{2+} levels are indeed increased in SAH. However, 90 % of the intracellular Mg^{2+} is complexed with ATP and the increase of intracellular Mg^{2+} during ischaemia may also be the result of the release of Mg^{2+} from this complex. ATP binds with Mg^{2+} with an associate constant of 4, while binding affinity with ADP is about 2 times smaller. The cytosolic and mitochondrial Mg^{2+} concentrations will increase in cells with a poor energy state and less ATP [3]. Within red cells there is an increase in Mg^{2+} concentration after deoxygenation on the basis of the greater affinity of deoxy-Hb than oxy-Hb for the cell Mg^{2+} buffers ATP, ADP and BPG. There is, however, also a direct binding of Mg^{2+} to Hb. Although the interaction is of low affinity, it becomes prominent at high concentrations of Mg^{2+} and Hb. This Mg^{2+} buffer capacity of hemoglobin might be an additional reason why serum magnesium is decreased in SAH . It may be an additional reason for the vasoconstrictive potency of oxyhaemoglobin as hypomagnesemia causes vasoconstriction[38] .The diminished availability, and subsequent decreased extracellular Mg^{2+} after SAH, results in significantly increased intracellular free Ca^{2+} in cerebral vascular muscle cells and type -2 astrocytes. This may cause cerebral microvascular constriction, followed by a pro-inflammatory response, inducing vascular smooth muscle, endothelial and neuronal cell damage [39]. Magnesium has the potency to attenuate cerebral ischaemia after SAH by its neuroprotective and vasodilatory effect and could thus ameliorate the clinical outcome in patients suffering SAH. Importantly, magnesium is safe, cheap and commonly available, and there is overwhelming clinical experience with magnesium treatment in a variety of disorders [40].

Uses of magnesium in treatment of acute myocardial infarction

Magnesium has properties of myocardial protection, the pathophysiological explanations of which in acute myocardial infarction include prevention of arrhythmia, antiplatelet effect, prevention of reperfusion injury, and coronary vasodilation. Although several studies have evaluated the role of magnesium administration in patients with acute myocardium infarction, the clinical impact of such therapy in this condition has been controversial, largely as a result of conflicting data from randomized controlled trials. The data available to date do not favor the routine administration of intravenous magnesium in patients with myocardial infarction, but this should not preclude magnesium administration to replenish low serum magnesium concentrations or use of magnesium sulfate for treatment of torsade de pointes in patients with myocardial infarction [41].

Use of magnesium in bronchial asthma: a new approach to an old problem

Asthma is a significant public health burden that affects more than 200 million people

worldwide. Asthma is a disease characterized by episodes of reversible narrowing of the airways in response to a wide range of endogenous and environmental triggers. It occurs in individuals who are predisposed to develop the disease as a result of genetic and environmental factors. Symptoms, caused by inflammation and smooth muscle contraction in the bronchioles, can vary from mild chest tightness, shortness of breath, and coughing or wheezing to respiratory failure and death. Currently, there is no cure for asthma. The disease is managed by taking appropriate medication and minimizing contact with environmental triggers such as air pollution, tobacco smoke, pets, dust mites, cockroach allergens, and mold. Other common causes of asthma exacerbations include respiratory infections, stress and even exercise [(42)] .

Acute asthma is common and severe exacerbations may result in hospitalization, need for endotracheal intubation and, rarely and death. Standard emergency care includes systemic corticosteroids and bronchodilators, but additional treatments may be required in severe cases. Nowadays there is asubstantial evidence regarding the role of MgSO4 in acute asthma and apotential benefit in chronic asthma[(43).]

Mechanisms of magnesium dilation to the smooth muscles of the bronchi inhibition of cholinergic neuromuscular transmission with decreased sensitivity to the depolarizing action of acetylocholine ,stabilization of mast cells and T lymphocytes ,stimulation of nitric oxide and dilatation prostacyclins ,stimulation of reticulum calcium uptake and stimulation of Ca-ATPase activity and the calcium drive into the reticulum[(44)] . The pathophysiology of asthma is a complex disease with genetic, immune system, environmental, behavioral, and nutritional components. Epidemiologic studies have implicated Mg in asthma management as the mineral is associated with bronchodilation,immune function, and anti-inflammatory properties. While, studies of Mg supplementation have shown mixed results in ability to modify Mg status and control of asthma symptoms, Mg status is not clinically easy to detect, and increased need for Mg may exist in people with asthma. Mg may be a useful complement to medical treatment of asthma as it may reduce airway hyper-responsiveness, increase PEFR, and result in a subjective perception of improved quality of life and asthma control[(45)] .Recommendations of the British Thoracic Society allow one dose of magnesium sulfate to patients with acute severe asthma exacerbation and inadequate initial response to broncho-dilating inhalation treatment (evidence category A). Future investigations should help to establish the indications for magnesium use in the treatment of acute asthma exacerbations as well as the magnesium dose and the scheme of its administration anticonvulsant action of Mg [(46)].

Surgery for pheochromocytoma

The use of Mg in pheochromocytoma surgery is based on the inhibitory action of catecholamine reuptake by the adrenal medulla, which reduces the sensitivity of adrenergic receptors in the context of peripheral vasodilatation and decreases antiarrhythmic effects [(47)] . Several clinical cases have demonstrated the advantage of supplementing conventional treatment with Mg sulfate to improve control of arterial pressure and heart rate. Hemodynamic balance was achieved during anesthesia induction and intubation, but frequently remained unstable during tumor resection [(48)].

Magnesium in anaesthesia and Perioperative analgesia

Several animal and human studies reported antinociceptive effects of magnesium when administered intravenously or intrathecally. Suggested mechanisms underlying these antinociceptive effects include the inhibition of calcium influx (calcium channel blockers augment morphine-induced analgesia and decreased total opioid consumption), antagonism of NMDA receptors and prevention of enhanced ligand-induced NMDA signaling in a state of hypomagnesemia_In addition; magnesium seems to attenuate or even prevent central sensitization after peripheral tissue injury or inflammation because of inhibition of dorsal horn NMDA receptors [(49)]. Mg blockade at the neuromuscular junction is the result of (i) a reduction of the amount of acetylcholine released from motor nerve terminals; (ii) a decrease in the depolarizing action of acetylcholine at the endplate; or (iii) depression of muscle fibre membrane excitability. In a rat phrenic nerve-diaphragm preparation, Mg potentiated neuromuscular blockade produced by rocuronium, decamethonium and succinylcholine. Prolonged curarization may require more frequent recourse to anticholinesterases to counteract neuromuscular blockade [(49)] .

Torsades de Pointes

Magnesium attenuates pathologic changes by inhibiting calcium currents, as shown by a variety of experimental and clinical data ,As an urgent measure, 2 g MgSO4(25–50 mg/kg in children) should be the drug of choice, followed by electrolyte stabilization and efforts to accelerate the basic heart rate [(52)] .

Digoxin-induced Arrhythmias

Magnesium is well established in the management of digoxin-induced tachyarrhythmias. Digoxin antibodies are the basic treatment, but in hypomagnesemic patients, especially those susceptible to digoxin-induced arrhythmias, intravenous administration of magnesium should be part of the immediate standard therapy until Fab antibodies are available (Class IIa, Level of Evidence B, AHA) [(53)] .

Conclusion: Magnesium has many known indications in anesthesiology and intensive care. Intensive care specialists need to have a clear understanding the role of this important cation. Magnesium is gaining recognition as a clinically important electrolyte in intensive care and emergency medicine .Recent clinical trials and case reports increasing interest of magnesium as an effective therapeutic agent for potentially life-threatening problems such as torsade de pointes, digitalis toxicity, bronchospasm, and alcohol withdrawal, subarachnoid hemorrhage, , preecalmsia , eclamsia ,acute myocardial infarction and cardiac arrhythmias, hypertension , diabetes, metabolic syndrome, correction of hypokalemia , induction of anesthesia and surgery of pheochromocytoma in order to control adrenergic response and an adjuvant in perioperative analgesia.

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