EDITORIAL

UNUSUAL RESPONSE TO MUSCLE RELAXANTS IN THE PREGNANT WOMAN UNDERGOING CESAREAN SECTION

Succinylcholine is usually recommended for tracheal intubation in the pregnant woman scheduled for Cesarean section under general anesthesia, while nondepolarising relaxant is used to maintain muscular relaxation throughout surgery. However, the use of both muscle relaxants may be complicated by the physiological and/or the pathological changes associated with pregnancy.

Because the parturient should be always considered as a patient with a full stomach and a possible difficult airway, succinylcholine is considered the muscle relaxant of choice for rapid sequence induction of general anesthesia. During pregnancy, the level of plasma cholinesterase which hydrolyses succinylcholine is moderately decreased, and hence hydrolysis of succinylcholine remains within the normal range. Also, the duration of action of succinylcholine will be only moderately prolonged in the heterozygote atypical parturient. However, in the parturient inheriting homozygote atypical esterase, hydrolysis of the injected succinylcholine is negligible with a consequent very prolonged neuromuscular block, which allows the transmission of the unhydrolysed succinylcholine across the placenta to the fetal circulation. This can be complicated by neuromuscular block in the fetus who may have inherited a homozygote atypical plasma cholinesterase if the father is homozygote or heterozygote carrier of the enzyme.

In addition to the possible prolonged neuromuscular block, succinylcholine administration may result in rare but very serious complications such as anaphylactic reactions. Also, it can trigger excessive potassium release in the parturients suffering from denervation conditions such as the quadriplegic patient or patients suffering from neurologic diseases such as guillan Barré syndrome.

The abnormal response to muscle relaxants during pregnancy is not limited to succinylcholine, but can also complicate nondepolarising muscle relaxants. Administration of nondepolarising relaxants during repeated Cesarean section can result in seriousIgE-mediated anaphylactic reaction which may culminate in maternal cardiac arrest. Fortunately, the placenta plays an important role in protecting the fetus against drug-induced anaphylactic reaction in the parturient. The placental barrier will prevent crossing of the high molecules-weight IgE antibodies to the fetus. Also, the high diamine oxidase of the maternal decidua will catalyze the oxidative deamination of histamine and other related endogenous amines released during anaphylaxis. Several case reports document the successful resuscitation of pregnant women in cardiac arrest after perimortem Cesarean delivery. The time interval from cardiac arrest to delivery is probably the single most important factor for fetal survival. If the fetus is delivered within 5 minutes, intact neurological survival is increased for both the mother and the newborn.

Abnormal response to nondepolarizing muscle relaxants during pregnancy can also occur in the undiagnosed myasthenia gravis parturient. Myasthenia gravis is easily missed during pregnancy. Misdiagnosis might lead to serious complications affecting both the mother and the neonate. The mother given the normal dose of nondepolarizing muscle relaxant can develop a prolonged and profound neuromuscular block which cannot be adequately reversed by neostigmine. Also, the neonate may develop transient myasthenia gravis symptoms. The onset of neonatal myasthenic

symptoms occur within the first two hours following delivery in 75% of the cases for a mean duration of 18 days.

Whenever nondepolarising relaxants are used in ecclamptic patients receiving magnesium sulphate therapy, its dose should be carefully titrated. The anticonvulsant effect of magnesium is usually attributed to both its central depressant action and its peripheral depressant effect on neuromuscular transmission. At the neuromuscular junction, magnesium deceases the presynaptic release of acetylcholine, as well as reduces the sensitivity of the post-junctional membrane to the liberated acetylcholine, and decreases the excitability of the muscle fibers membrane. These effects will potentiate the action of the neuromuscular blocking drugs, particularly the neuromuscular block of nondepolarising relaxants. Also, whenever magnesium is administered postoperatively to toxemic patients recovering from general anesthesia including muscle relaxants, its dose should be carefully titrated to avoid post operative recurarization.

Pheochromocytoma during pregnancy may mimic the usual symptoms and signs of preeclampsia. Paroxysmal attacks may be precipitated by postural changes, the mechanical effect of the gravid uterus in the last trimester, uterine contractions during labor and increased fetal movements. These signs and symptoms may mimic that of preeclampsia and is therefore often missed, and misdiagnosed as preeclampsia. However, hypertension associated with pheochromocytoma is seldom accompanied by oedema or proteinuria, while glycosuria is often present. Fortunately, the use of intravenous labetalol or hydralazine as well established in preeclampsia, and its use in combination with magnesium sulphate has been recommended in patients with pheochromocytoma. In this situation, the interaction of magnesium sulphate with muscle relaxants must be considered.

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