SEDATION WITH XYLAZINE (0.05mg/kg)

Xylazine, an alpha2-agonist non-narcotic compound, is a sedative and analgesic as well as muscle relaxant. Its sedative and analgesic activity is related to central nervous system depression. Its muscle relaxant effect is based on inhibition of the intraneural transmission of impulses in the central nervous system.

Xylazine is a popular choice for sedative in Large Animal Medicine because it has a wide margin of safety. Increasing the dose does not in general increase the degree of sedation but rather the duration of effect. Xylazine has proven to be a safe anesthetic adjunct when co-administered with ketamine to induce short periods of surgical anesthesia. When combined with ketamine, muscle relaxation and visceral analgesia are improved, and emergence from anesthesia is smoother. Other anesthetic agents (i.e. butorphanol, guaifenesin, benzodiazepines) have also been combined with xylazine to obtain surgical anesthesia.

Onset of action following IV injection occurs at 2 minutes, peak effect ocurrs in 5 minutes. Low doses (0.015 - 0.025 mg/kg IV or IM) will provide sedation without recumbency in cattle. Higher doses of xylazine (0.1 mg/kg IV or 0.2 mg/kg IM) will provide recumbency and light planes of general anesthesia in cattle for approximately 1hr. Duration of action lasts around 40 minutes. With intramuscular administration of xylazine, major effects develop in approximately 10 to 15 minutes after.

 When administered alone intravenously, xylazine induces bradycardia and a brief period of hypertension (5-10 minutes) followed by a longer period of decreased cardiac output and blood pressure.  The initial hypertension is caused by xylazine’s action at peripheral postsynaptic adrenergic receptors, which produces vascular smooth muscle contraction and vasoconstriction. Eventual reductions in blood pressure are due to decreased sympathetic tone resulting from xylazine’s activation of central and presynaptic sympathetic neuronal alpha2 adrenoceptors. Intramuscular administration of the drug does not produce this dramatic initial increase in blood pressure and vascular resistance.

The most commonly encountered arrhythmogenic effects of xylazine include sinoatrial block, atrioventricular block, bradycardia, first- and second-degree heart block, AV dissociation, and sinus arrhythmia. Respiratory rate decreases with the administration of xylazine. However, arterial pH, PaO2, and PaCO2 remain unchanged or are only minimally altered. Various alterations in gastrointestinal function have been reported following xylazine administration. These include hyper salivation, reduced reticuloruminal activity, reduced swallowing and reduced pharyngeal activity in ruminants; prolonged gastrointestinal transit time (mice), and vomiting (dogs and cats—due to central alpha2-adrenoceptor activation).

Xylazine causes transient hypoinsulinemia and hyperglycemia in several species. The magnitude and duration of these actions appear to be dose dependent. In ruminants, xylazine increases myometrial tone and intrauterine pressure.

Mydriasis is commonly observed after xylazine administration. This effect is caused by central inhibition of parasympathetic tone to the iris and/or direct sympathetic stimulation of alpha2 adrenoceptors located in the iris and CNS.