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The anesthetic propofol for cardioprotection in diabetes: An update

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Diabetes mellitus is the fifth leading cause of death worldwide. Diabetic patients often die from diabetic complications, the majority of which are connected with cardiovascular disease. Following Surgery, patients with diabetes suffer higher rates of perioperative cardiovascular morbidity, mortality, and a lower rate of survival than non-diabetic patients. Anesthetics may change the mode of ventricular filling and often modify and attenuate the stabilization of cardiac hemodynamics during surgery in patients. Increase in oxidative stress under pathological conditions has been shown to make the heart more prone to insults. In diabetes, hyperglycemia increases reactive oxygen species (ROS) production and induces oxidative stress, which has been recognized as a major mechanism of myocardial injury. Thus, cardiovascular responses to anesthesia in diabetic patients can be less predictable, making these patients more susceptible to cardiovascular complications during surgery which may be in part as a consequence of increased oxidative stress, and impaired endogenous myocardial protective pathway. Increasing evidence shows that propofol, a commonly used general anesthetic with antioxidant potential, can confer cardioprotection in patients undergoing either cardiac or non-cardiac surgery. However, the cardioprotective effects of propofol in patients with diabetes are debatable. Although conventional low doses of propofol have not been clinically effective in reducing postoperative cardiac injury or improving cardiac function, our *in vivo* study suggest that high doses of propofol can improve cardiac function and inhibit oxidative stress in diabetic rats. In line with our results, Crespo et al (Diab Vasc Dis Res. 2011 Oct;8(4):299-302) reported that propofol (50mg/kg IP) can increase cardiac output and systolic blood pressure (SBP), which should improve tissue perfusion in diabetic patients undergoing surgical procedures. Propofol is known to cause vasodilation by increasing nitric oxide (NO)-mediated relaxation secondary to eNOS activation. In diabetic rats, however, NO-mediated relaxation is impaired, and oxidative stress is increased. However, Wickley and colleagues suggest that propofol should be administered cautiously to diabetic patients with limited inotropic reserve and /or diastolic dysfunction because propofol can cause a decrease in myofilament Ca^{2+} sensitivity via protein kinase C and nitric oxide synthase-dependent pathway in diabetic cardiomyocytes, which may subsequently lead to hemodynamic instability. Therefore, it seems necessary that large-scale randomized clinical trials in diabetic patients be performed in the near future in order to answer whether or not propofol can confer cardioprotection in diabetic patients.

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