

# Insight into the effects of dexmedetomidine on intraoperative hemodynamics and postanesthetic recovery speed

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Dexmedetomidine is a potent alpha-2-adrenergic agonist, more selective than clonidine, with widespread actions that include anxiolysis, sedation, anesthetic-sparing, analgesia and sympatholytic properties. Since its release in the US market in late 1999, it has gained remarkable attention in the adult, pediatric and geriatric populations, predominantly because of its minimal respiratory depression. A large body of recent work supports its favorable profile in many other clinical scenarios, including neuroprotection, cardioprotection and renoprotection, with promising results [1].

Even though dexmedetomidine displays various favorable pharmacological actions, it is believed that its routine use on patients undergoing surgery is not the current trend. The reason for this can be summed up in two ways. First, intraoperative hemodynamic instability including hypotension and/or bradycardia due to sympatholysis may be problematic in some patients. Such hemodynamic effects of dexmedetomidine may be expected to be more pronounced in hypovolemic patients and in those with diabetes mellitus or chronic hypertension and in the elderly. Secondly, a delay in postanesthetic recovery is expected due to dexmedetomidine's relatively long duration of action compared to other short-acting anesthetics. This disadvantage means that dexmedetomidine is not suitable for day surgery patients.

An interesting study has been carried out associated with the effects of dexmedetomidine on intraoperative hemodynamics and postoperative recovery in this issue of Korean Journal of Anesthesiology [2]. Kang et al. [2] found that the changes in mean arterial pressure (MAP) during the operation in the dexmedetomidine group were significantly less than

those in the control group, which means that intraoperative administration of dexmedetomidine produced more stable hemodynamics than in the control. In addition, the authors claimed that dexmedetomidine was not associated with prolongation of extubation time or compromised recovery profile. Although their study was small-scaled, with only 10 patients involved in each group, their results are interesting in that they presented some evidence to dispel our concerns about dexmedetomidine-induced intraoperative hypotension and delay in postanesthetic recovery. These concerns have made us reluctant to the routine use of dexmedetomidine in the operating room.

The hemodynamic effects of dexmedetomidine are somewhat controversial. Hypotension and/or bradycardia are not unusual, because dexmedetomidine decreases sympathetic nervous system activity. On the other hand, transient hypertension has been observed primarily during the loading dose in association with the initial peripheral vasoconstrictive effects, although treatment of transient hypertension has generally not been necessary. Investigations in both humans and animals have revealed significant cerebral vasoconstrictive effects of dexmedetomidine [3,4]. Also, dexmedetomidine is known to be associated with higher MAP than midazolam in patients under epidural anesthesia [5]. Such controversies regarding hemodynamic effect are thought to be caused by biphasic action of dexmedetomidine, characterized by an initial short-term increase in MAP followed by a longer lasting hypotension. The biphasic action of dexmedetomidine is sometimes displayed in a dose-dependent manner, i.e., lower dosages reduce norepinephrine release, resulting in decrease

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in vascular tone and hypotension, and higher dosages produce alpha-2B-mediated vasoconstriction and hypertension [6]. One factor that should be remembered is that the hemodynamic effect of dexmedetomidine during anesthesia is, to some extent, implicated with its anesthetic sparing effect, because dexmedetomidine reduces the required amount of anesthetics necessary to maintain adequate depth of anesthesia.

Dexmedetomidine is basically a central nervous system depressant with a relatively long duration of action, and thus it causes delayed postanesthetic recovery. As was revealed in the Bührer et al. [7], dexmedetomidine reduced the thiopental dose requirement for electroencephalographic burst suppression by 30%, which was not the result of a pharmacodynamic interaction but rather of a dexmedetomidine-induced decrease in thiopental distribution volume and distribution clearances. Also, dexmedetomidine decreases the BIS value itself [8]. These characteristics are considered to be responsible for delayed recovery from anesthesia after dexmedetomidine. On the other hand, some investigators reported that dexmedetomidine promoted an earlier emergence and reduced the length of stay in the postanesthetic care unit [9,10]. In these reports, they ascribed shorter recovery time to less amount of anesthetics needed when dexmedetomidine was co-administered.

On the whole, the Kang et al. [2] results of the stable intraoperative hemodynamics and the acceptable postanesthetic recovery speed in the dexmedetomidine group are thought to be related to the anesthetics-sparing effect of dexmedetomidine, not solely to the pharmacologic effect of dexmedetomidine itself. In their study, the  $C_e$  (effecter-site concentration) of TCI-propofol was controlled to the minimum dosage needed to maintain BIS of 45–55, while the  $C_p$  (plasma concentration) of TCI-remifentanyl was fixed to be 10 ng/ml during the operation. They found that the mean infusion rate of propofol as supplement anesthetics was about 30% lower by co-administration of dexmedetomidine than in the control. At the end of surgery, the  $C_e$  of propofol in the dexmedetomidine group was relatively lower than that in the control group. As a result, less propofol administration was responsible for less propofol-induced vasodilatory effect, which in turn maintained relatively stable intraoperative MAP, offsetting dexmedetomidine-induced delayed recovery.

Again, it should be reemphasized that the favorable hemo-

dynamics during anesthesia and the comparable speed of recovery after anesthesia is attributed to dexmedetomidine's anesthetics sparing effect. Nevertheless, for the anesthetic management, dexmedetomidine-induced anesthetics sparing is meaningful in that a fundamental concept of anesthetic management is to minimize the side effects of anesthetic drugs by administering the lowest dose necessary to maintain an adequate depth of anesthesia while ensuring a rapid recovery.

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