Vasoconstrictor for Local Anesthetics

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Dental local anesthetics contain vasoconstrictors, such as adrenaline and felypressin, to enhance the anesthetic effects and reduce bleeding in the surgical field.

Adrenaline has been used for long periods of time as an additive to local anesthetics. With a typical dose range of 10 - 12.5 μg /mL, adrenaline is believed to prolong duration by its vasoconstrictive properties that prevent systemic reabsorption of local anesthetics. Several reports have warned against the use of dental local anesthetics containing adrenaline in patients with cardiovascular diseases [1, 2].

On the other hand, felypressin, a non-catecholamine vasopressor that is chemically related to vasopressin, has been used as a safe vasoconstrictor in patients with compromised cardiovascular status in Japan and European Union nations [3]. However, there have been reports that, in a dog study, clinical doses of felypressin caused decreases in coronary blood flow [4]. But, in felypressin contained propitocaine, felypressin have maintained oxygen supply in myocardial tissues by maintained myocardial blood flow [5].

An α-2 adrenoceptor agonist, clonidine, combined with a local anesthetic, has been found to extend the duration of the peripheral nerve block. The action of clonidine was suggested to be due to local vasoconstriction [6]. But, clonidine is not particularly specific for α-2 adrenoceptors and acts via α-1 adrenoceptors at comparatively high concentrations. Thus, it is unclear whether it acts via α-2 adrenoceptors. And despite substantial study, it is not clear which doses of clonidine are optimal for prolongation of analgesia after peripheral nerve blocks. The use of perineural clonidine is not currently recommended for clinical use [7].

On the other hand, another α-2 adrenoceptor agonist, dexmedetomidine, acts more specifically against α-2 adrenoceptors and has more than 7-8 times the affinity for α-2 adrenoceptors of clonidine. Dexmedetomidine, which produce sympatholytic, sedative, analgesic, antihypertensive and bradycardiac effects when combined with a local anesthetic agent, have been found to cause vasoconstriction [8-10]. The α-2A, α-2B and α-2C adrenoceptors have been well identified pharmacologically and have been shown to cause vasoconstriction [8-10]. The α-2A, α-2B and α-2C adrenoceptors have been shown to cause vasoconstriction [8-10]. The α-2A, α-2B and α-2C adrenoceptors have been shown to cause vasoconstriction [8-10]. The α-2A, α-2B and α-2C adrenoceptors have been shown to cause vasoconstriction [8-10].

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flick and epidural animal model, we previously reported that prolongation of the local anesthetic effect by dexmedetomidine was concentration-dependent [12,13].

We studied dexmedetomidine for local anesthetic, now. Use of a new drug, dexmedetomidine, that can replace adrenaline and felypressin for the safe enhancement of local anesthesia effect in patients with cardiovascular diseases, may be recommended.

References