Letters to the Editor

Articaine hydrochloride: is it the solution?

In a recently published, well-written review paper by Kakroudi *et al* the clinical safety of articaine (hydrochloride) pertaining to the claims that it is particularly neurotoxic (ie causing paraesthesia or, more correctly, hypoaesthesia) has been briefly discussed.[1] The authors correctly state that this focus on paraesthesia largely stems from postmarketing observational research done in Denmark, but supported by similar observations elsewhere after the introduction of articaine for dental use.[2,3] Most cases examined articaine used for inferior alveolar nerve blocks (IANB).

Two important questions arise from the paper by Kakroudi *et al*. The first question is why does articaine cause paraesthesia, in spite of the fact that its cytotoxic potential compared to the other commonly used local anaesthetic agents in dentistry, is among the lowest?[4,5] The second question is, why do we see a sudden increase in paraesthesias following the introduction of articaine local anaesthesia to dentists in countries which previously did not have access to it, while in Germany, where articaine was introduced to dentistry in 1976, they apparently do not have a high/abnormal rate of articaine-induced paraesthesia?

In this journal, Wells and Becket, in 2008, drew attention to a possible link between the concentration of articaine 4% and nerve damage following a focused literature review[6] which is supported by much earlier, and possibly transiently forgotten, research that neurotoxicity from articaine[7] and lignocaine[8] was dose-dependent and almost similar, in equal concentrations.[7] Further, Oertel *et al* showed that articaine hydrolysis by serum esterases was saturable by high articaine concentrations.[9]

One embarrassing point from a dental view, which has not been discussed, is how well dentists actually do IANBs and, more importantly, understand the basic pharmacological principles for the drugs they use. We propose that the absence of comprehension of simple pharmacological principles (ie dose-dependent effects) is a causative factor in the unnecessary increase in articaine-induced paraesthesia.

The important point is the second of the before-mentioned questions. Before the introduction of articaine to the new dental markets outside Germany, lignocaine 2% with different epinephrine concentrations was the 'standard' dental local anaesthetic with a relatively low incidence of pharmacodynamic adverse effects. It was not uncommon to inject relatively large volumes (eg two cartridges of 1.8 ml) for IANBs in the belief that this increased the ratio of anaesthetic onset. Ignorance of the simple fact that a local anaesthetic concentration of 4% is double that of 2%, and expecting identical pharmacodynamic properties when the drugs are more or less equipotent, is lack of basic pharmacological knowledge.

Articaine is a most welcome addition to the local anaesthetic armamentarium, but respect the dose-response profile of articaine. Do not inject large volumes of articaine when doing IANBs. Half the dose used with lignocaine is often more than enough for articaine IANBs.

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Authors' response

The authors thank Professors Vigen and Skoglund for their letter adding further information about articaine following our recent paper (Kakroudi *et al* *Dent Update* 2015; **42**: 88–93). We agree with the points raised and concur that reducing the volume of articaine administered would be expected to reduce the incidence of parasthesia following an IANB. However, avoidance or a reduction in the use of the IANB would be an even more significant factor in reducing the risk of paraesthesia. The beneficial properties of articaine, as outlined in our paper, allows alternative, and easier, techniques to be considered.

Dose is, indeed, a complex issue as, due to the difference in molecular weight between articaine and lidocaine, there are not twice as many molecules in articaine 4% as in lidocaine 2%; there are actually less than double and it is the number of molecules that is relevant to the efficacy of a dose-dependent drug. The key message for clinicians is that much less articaine is required.

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