The Cervical Plexus: An Evolution Shift in the Accessory Innervation Theory

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The cervical plexus (CP) has haunted clinicians and scientists for several years. Anatomists, general dentists, endodontists, periodontists, neurologists, pain physicians, anaesthesiologists, researchers and a variety of surgeons have been frustrated by the cranial nerves “accessory innervation” (AI) from the CP. Indeed, AI by the CP likely contributes not only to failed dental anaesthesia in the adult posterior mandible but also to a vast array of unusual head and neck clinical presentations, pain syndromes and postoperative complications. It is certainly disconcerting to fail to induce profound anaesthesia when a patient is in the chair expecting a painless procedure. From an estimated 300 million anaesthetic cartridges used annually in the US alone, anaesthetic fails in 13% (n=39 million) injections overall, with 88% (n=34.32 million) occurring with the inferior alveolar nerve (IAN) block (1). In endodontics, it has been reported that as much as 45% of IAN block fails, especially in mandibular molars with symptomatic irreversible pulpitis (1). The IAN block has the highest failure rate not only in dental local anaesthesia but also among all local anaesthetic blocks in medicine (2). Previous research illustrated several explanations for this, including the central core theory, lowered pH of inflamed tissues, nerve altered resting potentials, anaesthetic-resistant sodium channels, anaesthetic composition, recreational drugs, Ehlers-Danlos syndrome and having red hair. Accessory mental nerves and mylohyoid nerve branches have also been implicated in anaesthetic failures (3).

The AI to the IAN theory states that incidents of unsuccessful anaesthesia may result from inner- ventilations of the adult mandible arising from the CP in addition to the auriculotemporal, buccal, mental, incisive, mylohyoid and lingual nerves. This theory had not been universally accepted due to the lack of anatomical evidence demonstrating that the CP nerve can extend to the mandible (4). Previous research highlighted that the difficulty in identifying such superficial branches during dissections could be attributed to the small size and thickness of the mandibular accessory foramina and CP, as well as to the dissection technique used (3). However, a three-dimensional nerve mapping method investigation through human cadaver microdissection, tissue transparency, and Sihler’s technique for nerve staining presented the first evidence recorded in the literature of the transverse cervical nerve (TCN) from the CP entering the mandible (3). In animal models using primates and cats, it was revealed that the CP entered the mandible and provided direct innervation to the teeth and dental pulps (1, 3, 4). In addition to previous clinical evidence since the AI to the IAN theory introduced by Nevin, 1922, contemporary microdissection technology results confirmed that the CP supplied AI to the inferior border of the posterior mandible in 97% of cases through the TCN and great auricular nerve (4, 5).

On the basis of a systematic literature review, human cadaver dissections, and a randomised control trial clinical study completed by our research group, there is evidence for the contribution of the CP to cranial nerves, which could be responsible for anaesthetic failures during root canal procedures in the posterior adult mandible (4). A recent systematic review concluded that the anatomy of the craniocervical nerve plexus needs to be investigated further (4). These future investigations could answer questions, test hypotheses and design and develop techniques relevant to the implications of the CP and cranial nerves AI theory to address potential ways for managing persistent pain in mandibular molars scheduled for root canal treatment procedures (1, 3, 4).
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REFERENCES