A review of the T2 segment of the brachial plexus

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ABSTRACT

Although the complex architecture of the brachial plexus (BP) has been described for decades, recent literature still aims to elucidate the variation in nerve root contributions to the BP. Understanding this variability in the nerve morphology of the BP may assist physicians and surgeons in the diagnosis and management of certain clinical conditions that involve the BP, either directly or indirectly due to its close association with neighbouring structures. In this article, we review the current anatomical knowledge of the BP, focusing especially on its T2 contribution, and discuss the causes and consequences of some relevant BP pathologies.

Keywords: brachial plexus, intercostobrachial plexus, neuropathies, upper limb

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INTRODUCTION

Variations in the architecture of the brachial plexus (BP) have often been observed, and terms such as cephalic and caudal, high and low, or prefixed and postfixed have been used to refer to the nerve composition of the BP.[1] One of the most common variations is the contributions from the C4 and T2 roots, which are also referred to as a prefixed and postfixed BP, respectively. The most typical description of the BP involves the union of the ventral rami of C5–C8 cervical nerves with most of the ventral ramus of T1. Often, C4 provides a branch to C5, and T2 may communicate with T1.[2] What remains unclear regarding this arrangement is the extent of the contributions from C4 and T2. In this article, we review the anatomy of the BP with emphasis on recent investigations into the anatomical communications between T2 and the BP. We also consider the possible implications of this T2 contribution in brachial plexopathies, BP anaesthetic blocks and other surgical procedures involving the axillary region.

ANATOMY

Standard anatomy textbooks provide a typical short description of the BP.[3-5] Localised between the first rib and the clavicle in the region of the thoracic outlet, the BP

is a complex network of nerves formed by the union of the cervical and thoracic spinal cord roots. From its origin in the posterior triangle of the neck, the BP passes distally to the upper extremity, dividing into rami, trunks, divisions, cords and terminal nerve branches. At the medial surface of the middle scalene muscle, the C5 and C6 ventral rami join to form the superior trunk of the plexus, the C7 ramus continues as the middle trunk, and the C8 and T1 rami join to form the inferior trunk. Reaching the lateral border of the first rib, the trunks of the BP branch into anterior and posterior divisions, also recognised functionally as the flexor and extensor divisions, respectively. The divisions further separate into cords. The lateral cord is formed by the union of the superior and middle trunks, the posterior cord, by the posterior divisions of all three trunks and the medial cord, by the anterior division of the inferior trunk. From the three cords arise terminal branches, two main terminal branches and a varying number of smaller branches from each cord. The lateral cord terminates as the musculocutaneous nerve and contributes the lateral root of the median nerve. The posterior cord terminates as the axillary and radial nerves. Finally, the medial cord terminates as the ulnar nerve and medial root of the median nerve.[6] Also arising from various components of the BP are intermediary branches which include the dorsal scapular nerve arising from the fifth cervical nerve to supply rhomboids and sometimes, the levator scapulae; the long thoracic nerve arising from C5–C7 rami to supply the serratus anterior; the suprascapular nerve arising from the upper trunk with contributions from C5 and C6 to supply the supraspinatus and infraspinatus muscles; the nerve to the subclavius muscle also arises from the upper trunk; the lateral pectoral nerve arises from the lateral cord with contributions from C5–C7 to supply the pectoralis major; the medial antebrachial cutaneous nerve (with contributions from C8 and T1), the medial brachial cutaneous nerve (with contributions from T1 via the intercostobrachial nerve) and the medial pectoral nerve (with contributions from C8 and T1) arise from the medial cord to supply the skin over the distal medial arm, the skin over the proximal medial forearm and the pectoralis major and minor muscles, respectively; and finally, the thoracodorsal nerve (with contributions from C5–C8) and the upper and lower subscapular nerves (with

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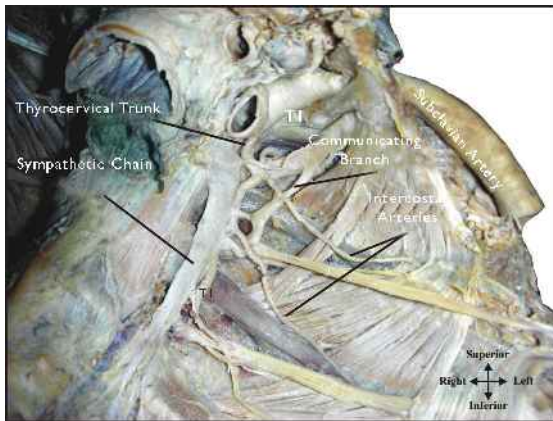
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Fig. 1 Photograph shows a cadaveric hemithorax from the left side. The lungs and endothoracic fascia have been removed to expose the neurovascular bundles. The subclavian artery is evident, giving off the thyrocervical trunk with several intercostal arteries. The T1, T2 and T3 spinal nerves are exposed with an evident communicating branch between T2 and T1.
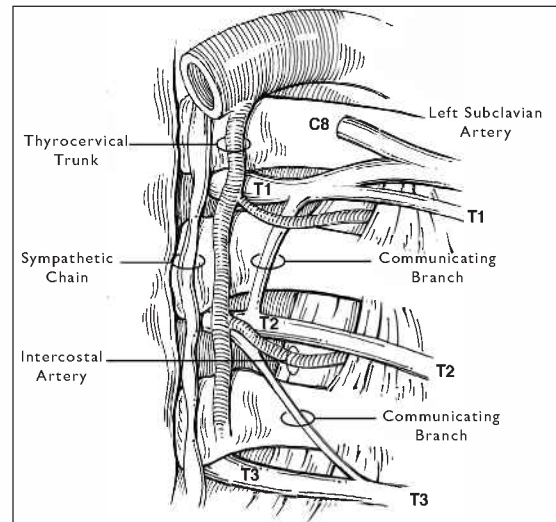
Fig. 2 Schematic representation of the communicating branch between T2 and T1 shows the left hemithorax. The lungs and endothoracic fascia have been removed to expose the neurovascular bundles. The subclavian artery is evident, giving off the thyrocervical trunk with several intercostal arteries.

contributions from C5 and C6) arise from the posterior cord to supply the latissimus dorsi, the subscapularis muscle and teres major, respectively.[7]

Despite the aforementioned typical representation of the BP, several authors have attempted to validate the degree of T2 contribution to the BP, which would broaden the textbook description of the C5 to T1 input into the BP. Kerr reported a postfixed plexus in 30% of his specimens.[1] Other researchers have obtained variable results in the incidence of postfixed plexuses. Cunningham identified a postfixed plexus in 72% of cases,[8] and Testut identified the contribution of T2 to the BP.[9] Paterson identified a postfixed plexus in 33% of cases,[10] Harman, in 58.33% of cases,[11] Hirasawa, in 16.5% of cases,[12] Brunelli and Brunelli, in 30% of cases,[13] Bonnel, in 4% of cases[14] and Uysal et al, in 2.5% of cases.[15] Slingluff et al also identified the contribution to the BP by T2 and highlighted recurring patterns in the microanatomical organisation of both prefixed and postfixed plexuses.[16] On the other hand, Aldolphi found T2 to be included in 73% of the plexuses that he examined.[17] Nevertheless, Aldolphi refuted the existence of a communication between T1 and T2 nerves, attributing such an occurrence to variations associated with the caudal displacement of the BP.[17] With regard to his own findings, Kerr claimed to have studied the caudal distribution of the BP in a limited number of specimens, and acknowledged that he was not able to find any reports where the C4 nerve entered the BP and the T2 nerve did not.[1] We can thus speculate that Kerr did not find any reports to confirm or deny the invariable contribution of T2 to the BP.

More recent studies by Loukas et al have expanded the schema of the BP further to include communicating branches between T2 and the BP in 100% of the specimens. The authors classified these communicating branches as intrathoracic and extrathoracic based on their point of origin. Intrathoracically, they observed that in 17.3% (n = 26) of the specimens, communicating branches always originate from the ventral primary ramus of T2 and travel distally towards T1; a finding that, to the authors' knowledge, had not been previously reported in the literature (Figs. 1 & 2).[18] In addition, Loukas et al described that extrathoracic communications occur in 86% (n = 129) of the specimens, arising from either the intercostobrachial nerve (ICBN) or one of its branches and communicating with the medial cord (35.6%, n = 46), medial antebrachial cutaneous nerve (25.5%, n = 33), or posterior antebrachial cutaneous nerve (24%, n = 31).[18] Furthermore, the majority of specimens (68.2%, n = 88) among these extrathoracic communications were observed to have only one extrathoracic branch, while the others (31.7%, n = 41) exhibited two branches. The authors concluded that all of the specimens contained a communicating branch between T2 and the BP, as those axillae lacking an extrathoracic contribution from the ICBN were always found to possess an intrathoracic communication.[18] In another report, Loukas et al studied the ICBN and its branching pattern in greater detail and described that in 25% (n = 50) of the specimens, the ICBN originated from T2 and provided a branch to the medial cord of the brachial plexus.[19] Whether the communicating branch between the BP and T2 represents BP branching or a T2 contribution to the BP is yet to be determined. In 1999, O'Rourke et al analysed the ICBN in 14 cadavers,

and reported communications with the BP in ten axillae.[20] Moreover, they noted that all the branches were from the BP to the ICBN, relying solely on the postulation that the angle of the connecting fibres between the ICBN and the BP was indicative of a course from the BP.[20] Caution must be exercised, however, in drawing function from morphology. As an example, Loukas et al raised the issue of the polarity of the communicating branches, stating the uncertainty of whether T2 is projecting fibres to the BP or if T2 is receiving fibres from the BP.[18] In other words, the authors were unclear as to the functional contribution of the communicating branches, if in fact, they contribute anything at all, to the BP. The efforts of all these authors must rightfully be acknowledged; it is, however, difficult to deduce more than merely anatomic significance from these reports. Further studies must be undertaken to elucidate the functional contribution of T2 to the BP, perhaps by way of animal experiments or electrophysiological studies.

CLINICAL RELEVANCE OF T2 CONTRIBUTION TO THE BRACHIAL PLEXUS

Clinically, the BP is extremely vulnerable to trauma as it is positioned between two moveable structures, the neck and upper extremity, and it is at a rather superficial location.[21] It is also worth mentioning that the BP lies in close proximity to structures such as the axillary artery, the apex of the lung, the breast and axillary lymph nodes, thus further increasing its susceptibility to traumatic injury and other disorders involving these adjacent structures. Although there are protective structures that oppose the traction of the cervical nerves, such as dural sleeves extending to the epineurium, fibrous attachments between the epineurium of C5–C7 and the cervical transverse processes, these arrangements are lacking at C8 and T1.[2] Consequently, these two caudal roots are at a greater risk of avulsion than the cervical roots C5 and C6.[1,22] Moreover, as the T1 root is of a shorter length, it is more prone to avulsion.[23] These features, therefore, emphasise the importance of elucidating the anatomical variation and functional contribution of T2 to the BP in order to fully comprehend the mechanism of action of avulsion injuries and devise effective diagnostic criteria and treatments for lower BP pathologies.

There are several examples of lower brachial plexopathies that stress the clinical relevance of T2 contribution to the BP. One such example is based on our experience and involves a patient with a lesion of the ulnar nerve, who retained function of the interosseous muscles; a finding that might be explained by the presence of a T2 contribution to the BP. Another example revisits the study by Loukas et al,[18] in which the authors highlighted the

importance of the intercostobrachial nerve in providing communication to the BP. This nerve is often injured during axillary lymphadenectomies, mastectomies or transaxillary procedures of the thoracic outlet region. The consequences of such a ligation include pain and paraesthesia in the axillary region and upper extremity. Based on their findings, the authors proposed the preservation of the ICBN in mastectomies and axillary node clearance and further speculated that the ICBN may also carry motor fibres.[18] Further research must be undertaken to test these hypotheses. Overall, Loukas et al's study has provided novel and critical information about understudied neuroanatomy with very plausible clinical relevance.

The lower BP segments also show clinical significance in avulsion injuries, as noted herein. The clinical literature suggests that avulsion injuries may be common, with avulsion of one or more roots occurring in nearly 70% of severe BP traction injuries.[24] Furthermore, avulsion injuries of the BP are not only physically traumatic and result in partial or complete loss of function of the upper extremity, but they also yield devastating emotional and socioeconomic consequences. This outcome is further complicated by the fact that innovative surgical techniques such as nerve transfers are able to restore only some function to the upper limb; however, complete recovery of function has not yet been reported.[25] As the nerve contribution to the BP is not fixed, it is difficult to interpret the recovery from avulsion injuries until more light is shed on the functional contribution of C4 and T2 to the BP.

Obstetric brachial plexopathies also occur as a result of a type of traction injury during both natural and Caesarean section deliveries, when the head and neck are deviated in excess to one side or the other in order to release the shoulder.[21] Focusing specifically on those obstetric brachial plexopathies that involve the caudal BP, Klumpke's palsy involves damage to C8 and T1 with the clinical manifestations of hand paralysis and Horner's syndrome.[21] As in adults, there is a poor prognosis in the recovery of these infants despite surgical intervention and physical therapy, thus further emphasising the need for research into the nerve fibre distribution of the BP.

Yet another relevant clinical consideration of the T2 nerve fibre contribution to the BP arises during anaesthesia of the region where the BP is contained. Neal et al, in their review, reported that the ICBN is blocked separately when anaesthetising the medial arm or axilla, or for the placement of an anterior portal during arthroscopic shoulder surgery.[6] Lanz et al also agreed that supplemental block of this nerve is necessary, and provided for their

reasoning the lack of evidence confirming that any of the current approaches to BP anaesthesia in fact block the T1–T2 segments.[26] In addition, BP nerve injury appears to be common during anaesthetic procedures. The American Society of Anaesthesiologists Closed Claims database reported that 16% of total claims are due to anaesthesia-related nerve injury. While 28% of these involve the ulnar nerve, 20% involve the BP. This high incidence of anaesthesia-related BP injury is perhaps a testament to the gap in our knowledge of the microanatomy of the BP and true functional nerve contributions to the BP.[27]

CONCLUSION

T2 contributions to the BP impart further complexity onto a structure that is already believed to be "the most complex structure in the peripheral nervous system".[21] Further research is required to characterise the nature of these contributions (number and type of fibres), confirm their directionality, compare these observations in animal studies, and more notably, to reveal their clinical relevance. It is important to fully understand the spectrum of diversity represented within the BP in order to assist clinicians and surgeons in the proper diagnosis and surgical treatment of brachial plexopathies, mastectomies and axillary lymphadenectomies. In light of the new and yet inconclusive knowledge of BP morphology, clinicians and surgeons should consider all possible variations of T2 contributions to the BP in the surgical treatment of breast cancers, lymph node clearance, anaesthetic nerve blocks, avulsion and traction injuries to the BP, and generally, in determining diagnostic criteria and effective treatments of other BP pathologies.

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