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This chapter describes the clinical anatomy of the brachial plexus and the nerves derived from it. The brachial plexus is the source of innervation for the entire upper extremity, including the upper extremity joints. Branches come off the plexus roots, trunks, and cords. Major terminal nerves from the plexus include the median, radial, ulnar, and musculocutaneous nerves. The information in this chapter will provide anatomical background relevant to blocks described in Chaps. 19, 20, 21, 22, and 23. Institutional subscriptions Chapter 45. Pectoral girdle and upper limb: overview and surface anatomy. In: Standring S, Johnson D, editors. Gray's anatomy, 40th ed. London: Churchill Livingstone; 2008. p. 780–90. Google Scholar Chapter 45. Pectoral girdle, shoulder region and axilla. In: Standring S, Johnson D, editors. Gray's anatomy, 40th ed. London: Churchill Livingstone; 2008. p. 818–22. Google Scholar Schuenke M, Schulte E, Schumacher U. The brachial plexus. In: Ross LM, Lamperti ED, editors. Thieme atlas of anatomy: general anatomy and musculoskeletal system. Stuttgart: Georg Thieme Verlag; 2006. p. 314–27. Google Scholar Download references The brachial plexus is a network of nerve fibres that supplies the skin and musculature of the upper limb. It begins in the root of the neck, passes through the axilla, and runs through the entire upper extremity. The plexus is formed by the anterior rami (divisions) of cervical spinal nerves C5, C6, C7 and C8, and the first thoracic spinal nerve, T1. In this article, we shall look at the anatomy of the brachial plexus – its formation and anatomical course through the body. The brachial plexus is divided into five parts; roots, trunks, divisions, cords and branches (a good mnemonic for this is Read That Damn Cadaver Book). There are no functional differences between these divisions – they are simply used to aid explanation of the brachial plexus. The 'roots' refer the anterior rami of the spinal nerves that comprise the brachial plexus. These are the anterior rami of spinal nerves C5, C6, C7, C8, and T1. At each vertebral level, paired spinal nerves arise. They leave the spinal cord via the intervertebral foramina of the vertebral column. Each spinal nerve then divides into an anterior and a posterior ramus. The roots of the brachial plexus are formed by the anterior rami of spinal nerves C5-T1 (the posterior divisions innervate the skin and musculature of the intrinsic back muscles). After their formation, these nerves pass between the anterior and medial scalene muscles to enter the base of the neck. Fig 1Proximal portion of the brachial plexus in the neck. Fig 2The spinal cord outflow at each vertebral level. The anterior rami of vertebral levels C5-C8 and T1 make up the roots of the brachial plexus. The root of the neck. The trunks of the brachial plexus are visible emerging between the anterior and middle scalene muscles. The root of the neck. The trunks of the brachial plexus are visible emerging between the anterior and middle scalene muscles. At the base of the neck, the roots of the brachial plexus converge to form three trunks. These structures are named by their relative anatomical location: Superior trunk – a combination of C5 and C6 roots. Middle trunk – continuation of C7. Inferior trunk – combination of C8 and T1 roots. The trunks traverse laterally, crossing the posterior triangle of the neck. Each trunk divides into two branches within the posterior triangle of the neck. One division moves anteriorly (toward the front of the body) and the other posteriorly (towards the back of the body). Thus, they are known as the anterior and posterior divisions. We now have three anterior and three posterior nerve fibres. These divisions leave the posterior triangle and pass into the axilla. They recombine into the cords of the brachial plexus. Once the anterior and posterior divisions have entered the axilla, they combine together to form three cords, named by their position relative to the axillary artery. The lateral cord is formed by: The anterior division of the superior trunk The anterior division of the middle trunk The posterior cord is formed by: The posterior division of the superior trunk The posterior division of the middle trunk The posterior division of the inferior trunk The medial cord is formed by: The anterior division of the inferior trunk. The cords give rise to the major branches of the brachial plexus. Fig 3Diagrammatic representation of the brachial plexus. For simplicity, the smaller branches of the brachial plexus are not shown. The posterior divisions are shown in yellow, and anterior divisions in black. In the axilla and the proximal aspect of the upper limb, the three cords give rise to five major branches. These nerves continue into the upper limb to provide innervation to the muscles and skin present. In this section, we shall concentrate on these five nerves. Note: these are only brief notes on the function of the nerves – for more detailed information click on the title to visit their respective pages. Musculocutaneous Nerve See more detailed information here. Roots: C5, C6, C7. Motor Functions: Innervates the brachialis, biceps brachii and coracobrachialis muscles. Sensory Functions: Gives off the lateral cutaneous branch of the forearm, which innervates the lateral half of the anterior forearm, and a small lateral portion of the posterior forearm. Fig 4The derivation of the musculocutaneous nerve from the brachial plexus Axillary Nerve See more detailed information here. Roots: C5 and C6. Motor Functions: Innervates the teres minor and deltoid muscles. Sensory Functions: Gives off the superior lateral cutaneous nerve of arm, which innervates the inferior region of the deltoid ("regimental badge area"). Fig 5The derivation of the axillary nerve from the brachial plexus. Median Nerve See more detailed information here. Roots: C6 – T1. (Also contains fibres from C5 in some individuals). Motor Functions: Innervates most of the flexor muscles in the forearm, the thenar muscles, and the two lateral lumbricals associated with the index and middle fingers. Sensory Functions: Gives off the palmar cutaneous branch, which innervates the lateral part of the palm, and the digital cutaneous branch, which innervates the lateral three and a half fingers on the anterior (palmar) surface of the hand. Fig 6The derivation of the median nerve from the brachial plexus Radial Nerve See more detailed information here. Roots: C5 – T1. 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Hyponyms: brachial plexus, cardiac plexus, celiac plexus, choroid plexus, Exner's plexus, internal carotid plexus, pelvic plexus, solar plexus, venous plexus 1934, Henry Miller, Tropic of Cancer, Grove Press, published 1961:I have the sensation of being immersed in the very plexus of life, focal from whatever place, position or attitude I take my stance. An interwoven combination of parts or elements in a structure or system. (mathematics) The system of equations required for the complete expression of the relations which exist between a set of quantities. 1866, Brande & C, A Dictionary of Science, Literature, & Art[1], page 935:Plexus (Lat.). The system of equations required for the complete expression of the relations which exist between a set of quantities. plexus m (plural plexussen) plexus m (uncountable) Perfect passive participle of plectō ("to plait, braid, interweave"). plexus (feminine plexa, neuter plexum); first/second-declension participle This entry needs quotations to illustrate usage. If you come across any interesting, durably archived quotes then please add them! First/second-declension adjective. From plectō ("to plait, braid, interweave") + -tus (action noun suffix). plexus m (genitive plexūs); fourth declension (poetic) a twining, plaiting, braid This entry needs quotations to illustrate usage. If you come across any interesting, durably archived quotes then please add them! Fourth-declension noun. "plexus", in Charlton T. Lewis and Charles Short (1879) A Latin Dictionary, Oxford: Clarendon Press plexus in Gaffiot, Félix (1934) Dictionnaire illustré latin-français, Hachette. The brachial plexus is a network of nerves responsible for the motor and sensory innervation of the upper extremity and upper chest. Pathologies affecting this structure can lead to significant morbidity, ranging from pain and sensory deficits to profound motor impairment. MR imaging is the modality of choice for imaging the brachial plexus. This review provides an overview of brachial plexus imaging, emphasizing practical aspects, anatomic landmarks, and imaging protocols, as well as a wide range of pathologies ranging from traumatic injury to inflammatory, idiopathic, hypertrophic, and neoplastic conditions. • MRI is the preferred modality for brachial plexus evaluation due to high resolution, multiplanar imaging, and superior soft tissue contrast. • Brachial plexus trauma causes pre- or postganglionic injury. Classification by severity or location helps identify patients who may benefit from conservative treatment or surgical intervention. • Schwannomas and neurofibromas are the most common primary brachial plexus tumors, but secondary neoplasms, spreading via direct extension or metastasis, are more frequently encountered. • Viral and vector-borne bacterial infections of the BP are often classified as Parsonage-Turner syndrome, typically presenting with a characteristic history and prodrome. • Radiation-induced plexopathy, the most common non-traumatic plexopathy, often occurs after radiation therapy for apical lung, breast, or head and neck cancers. 2 D 2 dimensional 3D 3 dimensional BP brachial plexus CIDP chronic inflammatory demyelinating polyneuropathy CMT Charcot-Marie-Tooth disease COVID-19 coronavirus disease 2019 CT computed tomography DTI diffusion tensor imaging EMG electromyography MPNST malignant PNST NCS nerve conduction studies NF neurofibromatosis PNSTs peripheral nerve sheath tumors SSFP steady-state free precession STIR short-tau inversion recovery T1WI T1-weighted images T2WI T2-weighted images TBPI traumatic brachial plexus injury The brachial plexus (BP) is a network of nerves responsible for the motor and sensory innervation of the upper extremity and upper chest. Pathologies affecting this structure can lead to significant morbidity, ranging from pain and sensory deficits to profound motor impairment. A thorough understanding of the anatomy, pathology, and imaging techniques is essential for accurate diagnosis and management. MR imaging is the modality of choice for imaging the BP. The BP typically arises from the ventral rami of the C5, C6, C7, C8, and T1 spinal nerves as they exit the neural foramina. , The BP consists of 5 ventral rami of spinal nerves, 3 trunks, 6 divisions, 3 cords, and 5 terminal branches (Figs. 1 and 2). These segments are often remembered using the mnemonic "Radiology Technologists Drink Cold Beverages" or the variation "Some Technologists Don't Carry Beepers," which uses the correct terminology for spinal nerves instead of "roots." , Fig. 1 The normal anatomy of the brachial plexus. (Created by Illustrator: Ienosuke Sato.) Fig. 2 Coronal oblique T2W image (A) shows the normal anatomy of the brachial plexus. The spinal nerves (S) and trunks (T) travel anterior to the middle scalene muscle (asterisk), the divisions (D) just above the inner margin of the clavicle (curved arrow), and the cords (C) and branches (B) on both sides of the coracoid process of the scapula (⋀). Coronal oblique T1W images (B and C) show the relationship of the cords (C) with the subclavian artery and vein (arrows), and the coracoid process (⋀). Normal anatomic variants are present in up to 50% of the population, with the most common being the course of C5 to C6 anterior to the scalene muscle. , Other variations, such as a prefixed (C4–C8) or postfixed (C6–T2) plexus, should also be recognized to avoid misinterpretation. These clinically significant variations may influence predisposition to certain pathologies and impact surgical planning. Anatomically, as well as on imaging, the BP can be divided into 3 key sections: supraclavicular, retroclavicular, and infraclavicular. Important anatomic and imaging landmarks define these sections as described in Table 1 . Brachial plexus anatomic landmarks and segments Sections Segments Anatomical Landmarks Supraclavicular Spinal nerves (roots) The ventral rami of the spinal nerves pass through the interscalene triangle after exiting the neural foramen . This triangle is formed by the anterior and middle scalene muscles and is bounded inferiorly by the first rib Trunks At the lateral border of the middle scalene muscle , the roots form 3 trunks : • Upper trunk : C5 and C6 roots • Middle trunk : C7 root • Lower trunk : C8 and T1 roots These trunks course posterior to the subclavian artery . Each one divides into 2 to give an anterior and a posterior division near the lateral border of the first rib Retroclavicular Divisions Triangular cluster posterior to the midclavicle and superior to the subclavian artery • 3 anterior divisions • 3 posterior divisions Infraclavicular Cords The divisions coalesce to form 3 cords , named based on their position with respect to the axillary artery , and located inferolaterally to the medial margin of the coracoid process : • Lateral cord : Formed by the anterior divisions of the upper and middle trunks • Medial cord : A continuation of the anterior division of the lower trunk • Posterior cord : Formed by the posterior divisions of all trunks Terminal branches The cords divide into the 5 terminal branches at the lateral border of the pectoralis minor muscle , the final anatomic landmark: • Median nerve • Ulnar nerve • Musculocutaneous nerve • Radial nerve • Axillary nerve Various diagnostic tools can be used to assess BP abnormalities, both invasive and noninvasive. Initial examination may include a radiograph to provide an overview of the spine, shoulder, and chest, allowing the detection of abnormalities such as a cervical rib, elongated C7 transverse process, or tumors like Pancoast. Ultrasound allows for dynamic assessment of the different segments of the BP during various neck and shoulder movements, and can be used as a guide for treatment options. Computed tomography (CT) is useful for evaluating osseous structures, particularly in cases of trauma or neoplastic bone involvement. CT angiography can be tailored for assessing thoracic outlet syndrome, while CT myelography can aid in evaluating preganglionic injuries in trauma. , , However, MR imaging is the preferred imaging modality for BP evaluation due to its high resolution, multiplanar capabilities, and superior visualization of soft tissues, nerves, and muscles. It enables detailed assessment of abnormalities from the spinal cord and intradural rootlets to the spinal nerves and terminal branches. , On MR imaging, the different segments of the BP are isointense to muscle on T1-weighted images (T1WI) and isointense to slightly hyperintense compared to muscle on T2-weighted images (T2WI; see Fig. 2). It is always important to compare the BP to the contralateral side, as they should be symmetric. Advanced techniques, including MR neurography and diffusion tensor imaging (DTI), provide improved visualization of nerve integrity and microstructure. , , DTI, in particular, enables 3 dimensional (3D) reconstruction of nerve fibers by analyzing water diffusion in multiple directions. Additionally, artificial intelligence is showing promise in automating segmentation and improving diagnostic accuracy. , Electrophysiologic testing, including nerve conduction studies (NCS) and electromyography (EMG), is used to confirm the diagnosis, localize the lesion level, and assess the severity of axon loss. MR imaging of the BP provides high spatial resolution of small anatomic structures in the head and neck region. Imaging protocols for the BP should be customized according to the clinical suspicion to optimize diagnostic accuracy. It can be performed on both 1.5 Tesla (1.5 T) and 3T scanners, with a combination of 2 dimensional (2D) and 3D sequences. , While 2D sequences offer superior in-plane resolution and faster acquisition times, 3D isotropic sequences allow for multiplanar reconstructions and detailed visualization of nerve pathways. The use of flexible surface coils or custom coil arrays ensures optimal coverage of the BP. , However, dedicated high-resolution multichannel phased array surface coils are preferred. Imaging protocol for BP MR imaging can vary between institutions due to differences in equipment, software capabilities, and specific clinical requirements (Table 2). MR imaging protocol example for the assessment of brachial plexus used at authors' institution, including a 3 plane localizer and a surface coil Primary Sequences FOV (~mm) Slice Thickness (mm) a TR TE Flip Angle Coronal T2W high resolution, with multiplanar oblique reformats Shoulder to shoulder Oblique axial , sagittal and coronal on the affected side 1 3800 191 120 Coronal STIR FSE Shoulder to shoulder 1–2 3800 195 120 Coronal T1WI TSE 2D Shoulder to shoulder 3–4 643 13 160 Sagittal oblique T1WI TSE Affected side (220–280) 3–4 730 12 130 If contrast required, multiplanar T1WI fat-suppressed sequences are obtained after contrast administration. Of note, coronal should cover shoulder to shoulder, and oblique reformats the affected side For imaging planning, axial oblique and coronal oblique images are preferred, with coronal and sagittal planes optimally oriented orthogonally to the neurovascular bundle. , This also helps reduce motion artifacts, as the heart and lungs are avoided. T1W non–fat-suppressed sequences are valuable for visualizing nerves within perineural fat. T2W fat-suppressed sequences help detect nerve abnormalities, muscular edema, and soft tissue changes and are useful to distinguish tumors and other pathologic processes. Short-tau inversion recovery (STIR) is a reliable method for fat suppression across scanners but has drawbacks like low signal-to-noise ratio, pulsation artifacts, and enhanced nerve signal. Spectral adiabatic inversion recovery offers a viable alternative when available. Intravenous contrast material should not be routinely used and should be reserved for specific cases, such as neoplastic conditions or infectious plexitis. If administered, T1W fat-suppressed sequences should be obtained. The BP can be affected by a variety of pathologic conditions, which can be broadly divided into 2 major categories: traumatic and nontraumatic brachial plexopathies. MR imaging plays a critical role in evaluating these pathologies, providing detailed visualization of structural changes and facilitating both diagnosis and treatment planning. Adult traumatic brachial plexus injury (TBPI) results in functional deficits and debilitating pain and may have mental health implications and significant economic impact. The annual incidence of TBPI worldwide ranges from 0.17 to 1.6 per 100,000. , These injuries most frequently occur in young adult male individuals who are involved in traffic accidents. , Classifying TBPI helps identify patients who may benefit from conservative or surgical treatment. TBPI is traditionally categorized using the Seddon and Sunderland classifications (Table 3). , According to the Seddon classification, the mildest nerve injury type is neurapraxia. This condition involves a temporary block in the conduction of nerve impulses, which typically resolves on its own. On the other hand, axonotmesis refers to damage to the axon and the supporting connective tissue framework, although these structures remain partially or fully intact. The likelihood of spontaneous recovery in cases of axonotmesis depends on the extent of the damage (see Table 3). Neurotmesis is the most severe type of nerve injury, characterized by a complete disruption of the nerve and its connective tissue components. This type of injury usually requires surgical intervention. Sunderland further developed Seddon's classification of nerve injuries by dividing them into 5 categories based on the number of compromised connective tissue layers (see Table 3). Degree of nerve injury, classification, and management Anatomical Structure Injury Classification of Injury Seddon Sunderland Management Axon Focal segmental demyelination Neuropraxia ("praxis" means "nonaction") Grade I Conservative Axon + connective tissue (lesion in continuity) Axon damaged with intact endoneurium Axonotmesis ("tmesis" means "cutting") Grade II Conservative Axon + endoneurium damaged with intact perineurium Axonotmesis Grade III Conservative Axon + endoneurium + perineurium damaged with intact epineurium Axonotmesis Grade IV Nerve reconstruction Axon + connective tissue + anatomic nerve division Complete nerve transection Neurotmesis Grade V Nerve reconstruction Based on the dorsal root ganglion, the BP can be categorized into preganglionic and postganglionic segments. A preganglionic injury occurs when nerve rootlets are avulsed from the spinal cord or when there is a rupture in the preganglionic root zone. This severe type of injury typically affects the lower C8 to T1 nerve roots. Spontaneous nerve recovery is not possible in cases of preganglionic injuries, which necessitate surgical intervention. Postganglionic injuries are further divided into supraclavicular and infraclavicular injuries. Supraclavicular injuries involving the roots, trunks, or divisions are typically associated with high-energy trauma. Compared to infraclavicular injuries, supraclavicular injuries are generally more severe, are more likely to require surgical nerve repair, and are associated with a worse prognosis. In contrast, infraclavicular injuries, affecting the cords and terminal branches, typically do not require surgery. They most commonly involve the C5 to C7 nerve roots and are often associated with other injuries, such as anterior shoulder dislocation or vascular dissection. , TBPI is often linked to high-energy blunt trauma resulting from falls from heights, motor vehicle accidents, and sports injuries. In certain populations, low-energy or gunshot wounds are significant injury mechanisms. MR imaging is the imaging method of choice for evaluating the BP as it facilitates surgical planning and patient prognostication. The imaging findings depend on the location of injury and time elapsed after injury onset. CT myelography serves as an alternative imaging method when MR imaging is not available or when MR imaging findings are unclear and is specifically limited to assessing preganglionic TBPI. MR imaging may reveal discontinuity of the ventral or dorsal nerve rootlets from the spinal cord (Fig. 3), indicating root avulsion, localized cord edema, and the presence of subarachnoid hemorrhage. , , Secondary imaging findings include the presence of a pseudomeningocele, a cerebrospinal fluid-filled nerve root sleeve with a cystic appearance, thickening of the dura matter (known as the black line sign), edema in the ipsilateral paraspinal muscles suggestive of denervation myopathy, and lateral displacement of the cord toward the side of injury due to scarring (see Fig. 3 ; Fig. 4). , MR imaging is most accurate for the detection of preganglionic injury when performed 3 to 4 weeks after the injury. This timing allows for the resolution of edema and hemorrhage and for the development of secondary imaging findings, such as pseudomeningocele. Of note, up to 23% of root avulsions do not have an associated pseudomeningocele. Fig. 3 MR findings in preganglionic injury. Axial high-resolution 3D steady-state free precession (SSFP; A and B), coronal STIR (C), and axial T2W fat-saturated (FS) (D) images, show normal appearance of the ventral and dorsal nerve rootlets from the spinal cord (arrows in A), which are not visualized (arrowheads in B) in preganglionic injury, indicative of nerve root avulsion. Other imaging findings associated with a preganglionic injury include pseudomeningocele (asterisk in C and D) and high T2 signal of the muscles, suggestive of denervation myopathy (curved arrow in D). Fig. 4 A 38 year old man with paralysis of the upper arm after sustaining a motor vehicle accident. Coronal STIR images (A–C) show a small pseudomeningocele within the right C7 to T1 neural foramen (arrowhead in A), indicative of a preganglionic injury (nerve avulsion). Associated hyperintensity and thickening of the postganglionic nerves (arrow in B) consistent with stretch injury, and high signal within the muscles suggestive of denervation myopathy (curved arrows in C). Only gold members can continue reading. Log In or Register to continue The brachial plexus is a network of nerve fibres that supplies the skin and musculature of the upper limb. It begins in the root of the neck, passes through the axilla, and runs through the entire upper extremity. The plexus is formed by the anterior rami (divisions) of cervical spinal nerves C5, C6, C7 and C8, and the first thoracic spinal nerve, T1. In this article, we shall look at the anatomy of the brachial plexus – its formation and anatomical course through the body. The brachial plexus is divided into five parts; roots, trunks, divisions, cords and branches (a good mnemonic for this is Read That Damn Cadaver Book). There are no functional differences between these divisions – they are simply used to aid explanation of the brachial plexus. The 'roots' refer the anterior rami of the spinal nerves that comprise the brachial plexus. These are the anterior rami of spinal nerves C5, C6, C7, C8, and T1. At each vertebral level, paired spinal nerves arise. They leave the spinal cord via the intervertebral foramina of the vertebral column. Each spinal nerve then divides into an anterior and a posterior ramus. The roots of the brachial plexus are formed by the anterior rami of spinal nerves C5-T1 (the posterior divisions innervate the skin and musculature of the intrinsic back muscles). After their formation, these nerves pass between the anterior and medial scalene muscles to enter the base of the neck. Fig 1Proximal portion of the brachial plexus in the neck. Fig 2The spinal cord outflow at each vertebral level. The anterior rami of vertebral levels C5-C8 and T1 make up the roots of the brachial plexus. The root of the neck. The trunks of the brachial plexus are visible emerging between the anterior and middle scalene muscles. The root of the neck. The trunks of the brachial plexus are visible emerging between the anterior and middle scalene muscles. At the base of the neck, the roots of the brachial plexus converge to form three trunks. These structures are named by their relative anatomical location: Superior trunk – a combination of C5 and C6 roots. Middle trunk – continuation of C7. Inferior trunk – combination of C8 and T1 roots. The trunks traverse laterally, crossing the posterior triangle of the neck. Each trunk divides into two branches within the posterior triangle of the neck. One division moves anteriorly (toward the front of the body) and the other posteriorly (towards the back of the body). Thus, they are known as the anterior and posterior divisions. We now have three anterior and three posterior nerve fibres. These divisions leave the posterior triangle and pass into the axilla. They recombine into the cords of the brachial plexus. Once the anterior and posterior divisions have entered the axilla, they combine together to form three cords, named by their position relative to the axillary artery. The lateral cord is formed by: The anterior division of the superior trunk The anterior division of the middle trunk The posterior cord is formed by: The posterior division of the superior trunk The posterior division of the middle trunk The posterior division of the inferior trunk The medial cord is formed by: The anterior division of the inferior trunk. The cords give rise to the major branches of the brachial plexus. Fig 3Diagrammatic representation of the brachial plexus. For simplicity, the smaller branches of the brachial plexus are not shown. The posterior divisions are shown in yellow, and anterior divisions in black. In the axilla and the proximal aspect of the upper limb, the three cords give rise to five major branches. These nerves continue into the upper limb to provide innervation to the muscles and skin present. In this section, we shall concentrate on these five nerves. Note: these are only brief notes on the function of the nerves – for more detailed information click on the title to visit their respective pages. Musculocutaneous Nerve See more detailed information here. Roots: C5, C6, C7. Motor Functions: Innervates the brachialis, biceps brachii and coracobrachialis muscles. Sensory Functions: Gives off the lateral cutaneous branch of the forearm, which innervates the lateral half of the anterior forearm, and a small lateral portion of the posterior forearm. Fig 4The derivation of the musculocutaneous nerve from the brachial plexus Axillary Nerve See more detailed information here. Roots: C5 and C6. Motor Functions: Innervates the teres minor and deltoid muscles. Sensory Functions: Gives off the superior lateral cutaneous nerve of arm, which innervates the inferior region of the deltoid ("regimental badge area"). Fig 5The derivation of the axillary nerve from the brachial plexus. Median Nerve See more detailed information here. Roots: C6 – T1. (Also contains fibres from C5 in some individuals). Motor Functions: Innervates most of the flexor muscles in the forearm, the thenar muscles, and the two lateral lumbricals associated with the index and middle fingers. Sensory Functions: Gives off the palmar cutaneous branch, which innervates the lateral part of the palm, and the digital cutaneous branch, which innervates the lateral three and a half fingers on the anterior (palmar) surface of the hand. Fig 6The derivation of the median nerve from the brachial plexus Radial Nerve See more detailed information here. Roots: C5 – T1. Motor Functions: Innervates the triceps brachii, and the muscles in the posterior compartment of the forearm (which are primarily, but not exclusively, extensors of the wrist and fingers). Sensory Functions: Innervates the posterior aspect of the arm and forearm, and the posterolateral aspect of the hand. Fig 7The derivation of the radial nerve from the brachial plexus. Ulnar Nerve See more detailed information here Roots: C8 and T1. Motor Functions: Innervates the muscles of the hand (apart from the thenar muscles and two lateral lumbricals), flexor carpi ulnaris and medial half of flexor digitorum profundus. Sensory Functions: Innervates the anterior and posterior surfaces of the medial one and half fingers, and associated palm area. Fig 8The derivation of the ulnar nerve from the brachial plexus. When dissecting the upper limb, it can be difficult to recognise what part of the brachial plexus you are at – it can just look like a mass of nerves. The important structure to look for is an 'M' shape. This is formed by the musculocutaneous, median, and ulnar nerves, usually superficial to the axillary artery. This shape is usually consistent between cadavers. It can help you get your bearings, and you can work backwards to identify the cords, divisions and branches. Fig 9The 'M' shape, consisting of the ulnar, median and musculocutaneous nerves. In addition to the five major branches of the brachial plexus, there are a number of smaller nerves that arise. They do so from all five parts of the brachial plexus, and are listed below: Roots Trunks Lateral cord Medial cord Posterior cord Dorsal scapular nerve Long thoracic nerve Suprascapular nerve Nerve to subclavius Lateral pectoral nerve Medial pectoral nerve Medial cutaneous nerve of arm Medial cutaneous nerve of forearm Superior subscapular nerve Thoracodorsal nerve Inferior subscapular nerve Erb's palsy refers to an injury to the upper ro

most common lesion in nerve injuries, especially in BPIs related to sport activity [37]. In more severe traumas, axonal structures have been damaged [38]. The portion of the axon distal to the site of lesion degenerates (Wallerian degeneration) and its muscle fibers lose innervation and trophic stimuli, determining muscle atrophy related to denervation mechanisms. These “orphan” muscle fibers could be reinnervated mainly through two mechanisms [39]. Axons from the proximal part of the damaged nerve may regrow and regain their muscle fibers (regrowth reinnervation) or adjacent axons can “adopt” orphan muscle fibers through collateral sprouting of axonal terminals within the muscle (collateral reinnervation). Regrowth of the axons is related to the distance from the site of lesion and the target muscle and it is usually a difficult and slow process. Moreover, connective structures have to be intact to allow regrowth and reinnervation. In any case, the functional relevance of the reinnervation processes is difficult to foresee, even depending on the number of axons involved. These kinds of lesions have a difficult prognosis and they should be monitored carefully; they can show sufficient functional spontaneous recovery or poor rehabilitative results. The most dramatic scenario is represented by neurotmesis [40]. In these lesions, all structures of the nerve are involved (myelin, axon, and connective tissue). Sometimes the nerve should be separated with a gap between the two nerve extremities. A stump neuroma should be present, as tentative regrowth by the nerve. Obviously, motor and sensory fibers are completely interrupted as a result in this case, and all muscle fibers managed by the nerve lose motor innervation and trophic stimuli, determining a severe atrophy and loss of function. In this situation, spontaneous recovery is impossible and surgical treatment is required. These lesions are common in severe traumatic BPIs unfortunately (i.e., cervical roots avulsions). Therefore, in each patient with a nerve injury it is necessary to define the etiology behind the lesion, localize the lesion carefully, and define the type of lesion. Indeed, this is unavoidably related to treatment and prognosis. Injuries of peripheral nerves can have a wide spectrum of etiology. Some diseases could be classified as primary diseases of the peripheral nerve, i.e., hereditary neuropathy [41]. In other cases, systemic diseases could determine peripheral nerve damage, as in metabolic neuropathies [42] (i.e., diabetes, renal insufficiency, amyloidosis, and others). Other systemic causes of neuropathy are toxic [43] (i.e., alcoholic neuropathy), related to nutritional deficiency [44] (i.e., hypovitaminosis) or to specific medical treatments (chemotherapy-induced neuropathy, post-actinic neuropathy) or correlated to some infectious diseases [45] (i.e., HIV, Lyme disease). Autoimmune mechanisms justify the onset of a large number of neuropathies [46], some with acute onset (i.e., Guillain-Barrè syndrome), and other with chronic manifestations (i.e., CIDP: chronic inflammatory demyelinating polyneuropathies). Some forms could be characterized by sensory symptoms; some others could determine motor sign uniquely (i.e., multifocal motor neuropathy). These diseases are systemic and determine the involvement of several peripheral nerves usually, affecting structures of the brachial plexus also. Specific characteristics of these forms are out of the scope of the present paper, but they should be taken in account during the examination of a patient with signs of involvement of peripheral nerve structures. Some peripheral nerve injuries could be iatrogenic [47], such as nerve damage during surgical interventions or related to medical procedures. These forms of nerve injuries, in particular those regarding the brachial plexus, are relevant in sport medicine as they are a non-rare complication of several orthopedic treatments like shoulder surgery and shoulder dislocation reduction. Primary (i.e., neurinoma) or secondary (i.e., brachial plexus involvement in lung tumors) neoplastic involvement of peripheral nerves is possible [48] and should be taken into account according to the clinical history referred by the patient and to the signs collected during the clinical evaluation. Nevertheless, the involvement of brachial plexus structures due to these systemic and localized diseases is rare. Indeed, in the epidemiology of the etiology of isolated BPIs, traumatic events remain the main cause of injury [49]. Usually, male subjects of working age are involved in traumatic BPIs, with a male/female ratio of 13.3:1 [5]. A closed lesion of the brachial plexus, mainly related to traction of peripheral nerves, represents 93% of traumatic BPIs [50]. The other reasons are gunshots lesions, lacerations, and iatrogenic lesions. Supraclavicular structures of brachial plexus (i.e., roots and trunks) are more involved (90%). Complete injuries of brachial plexus are quite common during traumatic lesions (53%). Nevertheless, the upper structures are more affected than lower [5]. Motorcycle accidents are the main cause of traumatic BPIs, accounting for 67% of them [5]. In motorbike or cycling accidents, the arm undergoes a sudden and violent traction caused by the fall and usually the higher the velocity the more severe the clinical presentation of the injury [49]. In occupational or sport activities, data from previous studies reported a pooled prevalence of 10% of traumatic BPIs [5]. Some more detailed data about sport activities show an incidence of BPIs among nearly 4% of trauma related to winter sports [51]. Obviously, BPIs are a common injury in contact sports [49] but their exact incidence is difficult to estimate. Indeed, lesions with neuroapraxia, commonly known as “burners and stingers syndrome” are common in these sports and related to impact (i.e., kick in martial arts) on the supraclavicular region. Likely, the complete functional recovery associated to these lesions determines an underestimation of their exact rate of incidence. A similar concept should be considered is some sports, even in relation to the equipment. In American football, the use of helmets has reduced the number of severe concussions, but it has caused a higher incidence of trauma in the supraclavicular region with the involvement of the brachial plexus [52]. Even in motorcycle accidents, the adoption of helmets permits the management of severe BPIs often requiring a surgical intervention. Probably, the increase of these cases is related to a higher survival rate for the use of the helmet itself [53]. Moreover, a reporting bias should be considered in the epidemiology of BPIs related to sport activities. Indeed, almost all studies reported the incidence and prevalence of traumatic BPIs that required surgical treatment [54]. Several lesions of brachial plexus showed a less dramatic involvement of nerve structures, with neuroapraxia and partial axonotmesis. In these cases, a conservative approach could determine a successful and sometimes complete recovery. The incidence of these lesions is not quantified in published studies and their prevalence could be underestimated. Some data show that 30–40% [55] of rugby players have suffered a “stinger” or “burner” at least once. In a Canadian football study, a “stinger” was reported by the 26% of players, during the 2010 football season [56] and only 59% of them were referred to medical staff. Moreover, the history of “stinger” is a risk factor to sustain a new “stinger” and a correlation with the years of sport practice was detected too. In an American football study, the lifetime rate of BPIs (“stinger”) was 50.3% [57] and the incidence seems to be related to the role in the team (highest incidence in running backs and defensive linemen). Therefore, while incidence rates of severe BPIs seem to be low, the incidence of simpler lesions of BPIs, like burners and stingers, depicts a worrying and an unclear scenario in sport medicine [37]. BPIs should be suspected in each athlete presenting with symptoms of peripheral nerve involvement [58]. As previously stated, most of the BPIs are associated to indirect trauma, with traction of cervical spinal nerves due to forced movements of the arm or of the cervical spine. Therefore, a clinical examination aiming to exclude an involvement of cervical spinal cord is mandatory. Radiographs should be performed to rule out cervical vertebral involvement [59]. Any sign of weakness or numbness involving lower limbs or territories distal to upper limbs should be evaluated carefully. After having excluded a cervical spinal cord involvement, the evaluation of segmental strength is useful to localize the level of lesion within the plexus [58]. Localization of the lesion should be difficult, even due to the high degree of anatomical variability between, and even within, subjects [60]. Anatomical knowledge could guide the localization of the lesion. Based on the time from the lesion, the visual inspection could demonstrate selective atrophy in specific muscular groups [61]. Indeed, after axonotmesis or neurotmesis, several days are needed to permit muscular atrophy. Moreover, visual inspection could make visible two relevant signs able to localize lesion: winged scapula and Bernard-Horner syndrome. Winged scapula [62] could be associated to several nerve injuries. In particular, lesion of three nerves could determine winged scapula: long thoracic nerve, dorsal scapular nerve, and spinal accessory nerve. Some clinical characteristics, out of the scope of the present paper, help in identification of nerves involved among them [63]. Nevertheless, the long thoracic nerve lesion is the most common form. Its involvement should be isolated and related to several diseases. Interestingly, in a patient with BPIs, the presence of a winged scapula due to a lesion of long thoracic nerve should be investigated carefully. Indeed, the long thoracic nerve arises from C5-C6-C7 at roots level and is the most proximal nerve arising from the brachial plexus [20]. Therefore, its clinical deficit is compatible with a very proximal lesion of brachial plexus, usually at roots level, as roots avulsion. Moreover, Bernard-Horner syndrome is characterized by homolateral miosis, ptosis, and enophthalmos [64]. This clinical presentation is common in proximal involvement of the lower portion of the brachial plexus. Indeed, lesion of C8-T1 roots determines the involvement of the sympathetic trunk with lesions of fibers that from the spinal cord in cervical and thoracic regions go back to head and cranial structures [59]. Although several central localizations could justify this syndrome, its occurrence in a patient suffering from BPIs is indicative of involvement of distal structures of the plexus. Manual muscle testing, grading voluntary recruitment of skeletal muscles from zero (no movement or perceived muscular contraction) to five (normal strength against gravity and full manual resistance) is commonly used to assess strength [59]. The involvement of the upper trunk is common and determines weakness of proximal muscles (i.e., scapular muscles and muscles of shoulder), while hand muscle function is preserved (Erb-Duchenne paralysis) [65]. Conversely, involvement of the lower trunk is associated to a motor deficit of the intrinsic hand muscles, with normal strength in proximal muscles (Klumpke paralysis) [66]. Ventral rami of spinal nerves, trunks, divisions, cords, and terminal branches of the brachial plexus are mixed nerves, including some sensory fibers. Therefore, BPIs are also associated to sensory symptoms in specific cutaneous areas. Sensory symptoms could include neuropathic pain, numbness, hyperalgesia, allodynia, hypoesthesia, and anesthesia, according to the time from the lesion and to the type of lesion [67]. A high variability of the sensory cutaneous pattern of innervation exists. However, the cutaneous sensory involvement has to be consistent to the pattern of weakness to permit an exact localization of the lesion. It is useful to specify that sensory cutaneous innervation of brachial plexus structures is different from dermatomeric innervation. Indeed, the dermatome is the cutaneous area innervated by a specific dorsal root of a specific spinal nerve. On the other hand, the lesion of a brachial plexus structure implies the involvement of cutaneous regions served by specific fibers of different spinal nerves travelling in the same structure of the brachial plexus [68]. The osteotendinous reflexes appear reduced or absent, according to the anatomical distribution [59]. For example, the bicipital reflex is conveyed through musculocutaneous nerve, lateral cord, upper trunk, and C6 root [69]. Each lesion along this pathway could determine the abolition of the reflex. In addition, changes in the deep tendon reflexes have to be consistent with the patterns of weakness and sensory involvement. A detailed clinical examination should be sufficient to localize the lesion in most of cases and to orient the following steps in the management of patients suffering from BPIs. Electrodiagnostic studies and imaging techniques are useful to confirm the diagnosis, to better characterize the lesion, to define the prognosis, and to guide the treatment. In BPIs, electrodiagnostic studies include electroneurography and needle electromyography, commonly [70]. Evoked potentials are not useful as routine examinations. Electroneurography [71] permits the analysis of compound motor action potentials (CMAPs) and of sensory action potentials (SAPs) through electrical stimulation of peripheral nerves and surface recording from muscles or skin areas, respectively. Motor and sensory conduction velocities can be measured. The main rule of electroneurography in BPIs is to confirm a pathological involvement of SAPs [68]. Indeed, due to the anatomical localization of cell bodies of sensory neurons (ganglion of dorsal root), the persistence of normal sensory action potentials is non-compatible with a plexus lesion (SAPs remain normal in patients with BPIs only if an avulsion of dorsal root is present: pre-ganglionic lesion). Conversely, any damage along the nerve reduces the amplitude of sensory action potentials (post-ganglionic lesions). Moreover, the analysis of CMAPs permits restriction of the possible localization of the lesion and exclusion other diseases. Motor conduction blocks are the electroneurographic manifestations of myelin dysfunction and represent the main finding in neuroapraxial lesions. Needle electromyography is sensitive in detecting denervation [72]. Therefore, it is useful to demonstrate axonal damage. Hence, it is necessary to demonstrate axonotmesis and neurotmesis. Moreover, through electromyography, it is possible to investigate active recruitment of the muscle and to investigate the morphology of motor unit potentials (MUPs) [59]. Indeed, MUPs analysis could also demonstrate reinnervation progression, as well as it can add information about the prognosis. Thus, electrodiagnostic studies are able to localize the lesion and to provide functional information; moreover, they permit definition of the type of lesion and follow up of the reinnervation process [59,73]. Unfortunately, they require a specific timing that has to be taken into account. Conduction blocks are immediately detectable through motor conduction studies. After axonal lesions, reduction of CMAPs and SAPs amplitude requires several days. During the first days, the amplitude of the potentials recorded though electrical stimulation distally to the lesion site is still normal and decrement begins according to Wallerian degeneration. CMAPs nadir is reached in 4–5 days, while SAPs nadir requires 7–8 days. According to the time from the lesion and to the distance of the target muscles, denervation signs are detectable with needle electromyography after several days and usually requires 2–3 weeks to became evident in more distal muscles (21 days) [74]. Imaging techniques to investigate peripheral nerves have become more common in the last few years. Indeed, improvements in the equipment allow better investigation of these structures. X-ray and MRI of the cervical spine are commonly performed and are useful to rule out vertebral fractures, spinal cord involvement, and to assess some indexes like the Mean Subaxial Cervical Space Available for the Cord (MSCSAC): the antero-posterior diameter of spinal canal minus the antero-posterior diameter of spinal cord, measured in a sagittal plane. Indeed, some previous results showed an association between risk of BPIs and cervical canal stenosis assessed through this method in athletes [75]. Modern magnetic resonance (MRI) is useful to define the nerves and their relationship with normal or pathological surrounding structures [76]. Nowadays, MRI could provide visualization and investigation of brachial plexus structures directly and could give some information about the neurogenic muscular changes, like denervation (Figure 3). 3D T2 STIR SPACE sequence—coronal images of normal Brachial Plexus. (a) Dotted rectangle includes cervical spinal nerves emerging from the intervertebral foramina in the supraclavicular laterocervical region; (b) dotted rectangle includes the brachial plexus structures passing through the cervicoaxillary canal.Regarding ultrasound (US), the diffusion of high-frequencies probes has allowed the exploration of small superficial structures as peripheral nerves. Ultrasounds show some advantages [60]. They are not invasive and allow performing dynamic maneuverers able to investigate stability of peripheral nerves during active and passive movements. US also allows exploration of some parameters of inflammation as vascularization, such as visualizing the ultrastructure of the nerve and the fascicular architecture. Furthermore, US allows undertaking of some measurements like cross-sectional area of the nerve, following the course of several nerves, and, finally, studying their relationship with the other anatomical structures. Nevertheless, due to the anatomical complexity of the region, imaging investigation of BPIs is difficult and a high-degree of competence is required [58]. Supraclavicular portions of the brachial plexus could be investigated in lateral neck regions. Spinal nerves could be identified and followed until trunk formation. Some anatomical landmarks, such as anterior and posterior tuberculi of transverse processes of cervical vertebrae, and scalenus muscles (anterior and middle), could be useful in order to localize these structures [77] (Figure 4). Ultrasound image of the interscalene groove in supraclavicular laterocervical region. Cervical spinal nerves (ventral rami) forming the brachial plexus pass through the anatomical space between anterior and middle scalene muscles, before they merge to form the upper, middle, and lower trunks. (a) Ultrasound image. White arrows indicate cervical spinal nerves (ventral rami); (b) schematic representation of the interscalene groove. C5 is the most superficial cervical spinal nerve, immediately below the sternocleidomastoid muscle. C5: cervical spinal nerve 5. C6: cervical spinal nerve 6. C7: cervical spinal nerve 7. C8: cervical spinal nerve 8. T1: thoracic spinal nerve 1. SCM: sternocleidomastoid muscle, AS: anterior scalene muscle, MS: middle scalene muscle.Divisions are not commonly explorable as they are located behind the clavicle and their ultrasound exploration is impossible. Cords and terminal branches are explorable distally to the clavicle and they are commonly visualized through an axillary approach. A relevant anatomical vascular reference, in this case, is due to their relationship with the axillary artery. Even US could give qualitative information about muscles, identifying denervated muscles in which the contractile tissue is substituted with connective and adipose tissues after denervation [77]. Although functional information is not obtained through imaging studies or should be considered “indirect” (i.e., denervation in a muscle could indicate axonal damage of a specific nerve), numerous anatomical and morphological information is provided. Therefore, in most cases, electrodiagnostic studies and imaging techniques are useful to collect numerous morphological and functional information about the BPIs. Therapeutic options are defined according to the type of lesion [61]. Neuroapraxia requires conservative treatment, and the recovery is complete. Neurotmesis requires early surgical treatment. Axonotmesis requires a conservative approach and an accurate follow up to evaluate the reinnervation process. In the case of insufficient restoration of the function, surgical treatment could be required. The main goals or rehabilitation are (i) protection of the injured area, avoiding other damage of nerve structures, (ii) pain control and management of sensory symptoms, (iii) limitation of muscular atrophy, (iv) improvement of strength, (v) restoration of function. In order to obtain these goals, several techniques are adopted. It has been demonstrated that stretching maneuvers are able to maintain flexibility of skeletal muscles [78]. Moreover, molecular evidence shows that passive stretching promotes the stimulation of mechanoreceptors and slow down the degradation process of proteins, representing the molecular basis of atrophy [79]. Stretching is also relevant to maintain a correct range of motion of the joints functionally correlated to the involved muscles. Electrical stimulation determines the passive contraction of the denervated muscles. Several types of electrical stimulation have been proposed [80]. Morphology of the waves, duration of electrical stimulation, and frequency of stimulation and position of the electrodes are parameters that should be considered in the treatment protocol. Nevertheless, no evidence supports a specific protocol of intervention about electrical stimulation and several approaches exist [3]. Transcutaneous nerve electrical stimulation (TENS) is commonly adopted to interfere with sensory perception and exerts an antalgic effect [81]. In partial axonal lesions, the active recruitment of the involved muscles could be useful to promote reinnervation and to increase strength and hypertrophy of survival motor units. This goal could be achieved through several rehabilitative techniques (i.e., neuromuscular proprioceptive facilitations [82]). It has been proposed that exercises against external resistance increase the strength of the muscles, through isometric, isotonic, and auxotonique contractions (elastic resistance) [83]. The education of the patient is also important, especially if a relevant sensory involvement is present. The deafferentation of skin tissue is the main cause of skin lesions (cuts, burns, and others) that could facilitate other diseases like infections and orthopedic traumas [84]. Rehabilitative approaches are important to maintain proprioception of deafferented structures also, limiting the impact on central motor representation and control. Some orthoses are useful to facilitate movement or to permit strengthening of specific muscle groups, like hand muscles. Some BPIs could be very painful and pain management is a difficult topic. Some drugs, like opioids, should be used in neuropathic pains. Some antiepileptic drugs, like pregabalin and gabapentin [85], have shown positive effects in post-traumatic neuropathic pain relief, although some side effects are reported (i.e., dizziness) and discontinuation of treatment is frequent. As in other musculoskeletal conditions [86], some multivitamin supplements and neurotrophic agents are commonly adopted, especially in neuroapraxia and axonotmesis, even if their effectiveness is debated and even if they are commonly used in sports population behind nerve injuries [87]. Perineural steroid and anesthetic injections at the site of nerve injury, commonly adopted for short relief of the pain in other several musculoskeletal conditions [88], are not routinely performed in the management of neuropathic pain but they have shown some positive results [89]. The timing of surgical treatment has been extensively discussed in scientific literature [36,54]. Some BPIs (i.e., gunshots injuries) are associated to vascular injuries or open lesions (i.e., cut lesions) and require acute surgical treatment [90]. Neurorrhaphy is often performed, and the anatomical continuity of the peripheral nerve is restored. In case of a significant damage of neural tissue, a simple neurorrhaphy cannot be performed and a nerve graft is required. Usually, the sural nerve (a pure sensory nerve of the leg) is used as donor. Preganglionic lesions, like roots avulsions, have no spontaneous recovery and a delayed surgical treatment is not useful. Therefore, different surgical approaches are performed as soon as possible [90]. In the large part of BPIs, the mechanism of lesion is mainly due to excessive traction along the peripheral nerve with axonal damage and a variable amount of neuroapraxia is commonly present [91]. In this case, the surgical approach is often delayed for 3–4 months. During this period, diagnostic procedures and rehabilitative treatments were carried out: clinical evaluations and electrodiagnostic studies could be useful to attest progression in recovery of denervated muscles. In the case of poor restoration of function, surgical exploration of brachial plexus structures is required. Sometimes, it is difficult to restore all functions of muscles innervated by damaged nerves. Therefore, a “strategy” of priority in recovery is necessary. Restoring the function of elbow flexors and shoulder stabilizers is often considered as priority [91]. Several peripheral nerve procedures have been proposed during recent years. Neurotization is commonly adopted [92]. This technique consists of a motor nerve transfer. A donor spare motor nerve is transferred and sutured to a damaged nerve. Intra-plexus neurotization provides transfer of a nerve from the brachial plexus in favor of another nerve within the brachial plexus. For example, some motor branches of the radial nerve directed to the long head of triceps are transferred to a damaged axillary nerve to restore the function of deltoid muscle. In other cases, some extra-plexus nerves could be used. As example, the intercostal nerve could be transferred in favor of the musculocutaneous nerve or the spinal accessory nerve could be used to reinnervate muscles supplied by the suprascapular nerve [93]. In some cases, terminal branches of a spared motor nerve could be inserted directly in the target muscles to promote the generation of new neuromuscular junctions. Some procedures provide the transfer of a specific fasciculus of a nerve, distal to the brachial plexus, in favor of another nerve. For example, in the procedure described by Oberlin et al. [94], the ulnar fasciculus directed to flexor carpis ulnaris is transferred in favor of motor branches for biceps brachii innervation. Several orthopedic procedures were used in recent years, like shoulder arthrodesis, wrist arthrodesis, or even upper limb amputation. Improvements in surgical peripheral nerve procedures have reduced their utility dramatically. In specific cases, in which surgery was performed too late, and nerve restoration is not possible, tendon transfers have become a valuable palliative surgical treatment [95]. In these cases, the target of the surgery is the transfer of a tendinous structure of normally innervated muscles. Just as an example, trapezius muscles attachment could be transferred to humerus to restore abduction or external rotation, the pectoralis major’s tendon could be transferred in favor of biceps tendon to restore elbow flexion. The triceps tendon could be transferred in favor of biceps tendon to restore elbow flexion. In the late 70s some authors [96] described a technique of free muscle transfer. Some muscles far from the site of lesion, are transferred to restore some biomechanical functions. For example, gracilis muscle is transferred in favor of elbow flexors. This technique requires microvascular sutures and it is technically difficult. In the last years, a bionic reconstruction approach has been described in patients with global BPIs including lower root avulsions. These patients have a severe impairment of hand function, and they have received previous surgical brachial plexus reconstruction without significant restoration of distal muscles voluntary contraction. Therefore, they have no other treatment options available. This approach has been conducted in two stages: first useful electromiographic signals for prosthetic control were identified and/or created through assessment of voluntary recruitment of some forearm muscles or by a free muscle transfer of gracilis muscle. After a dedicated rehabilitation program to improve the control of these muscles, an elective amputation of hand has been performed and a replacement with a mechatronic prosthesis was conducted. Although the results are restricted to a limited cohort of patients and this technique is indicated in BPIs without other treatment options, a significant functional improvement has been proved [97,98]. As previously described, some sport activities are associated to BPIs with different degree of motor and sensory impairment. “Stinger” involving the upper trunk of brachial plexus represents the most common BPIs in athlete. This condition also represents the most common upper extremity neurologic injury in the athlete [99]. They are acutely involved and could have different clinical presentation according to the degree of nerve damage. In the most favorable scenario, some forms of stingers are not reported to physician. This high percentage of “missed” athletes could be justified by the lack of long-lasting symptoms. Indeed, some athletes could experience only self-limiting sharp and burning pain, radiating down the arm with paresthesia and numbness lasting for few seconds to minutes. Therefore, even if the time lost in athletic activities is 2.9 days on average, 79.3% of athletes continue a normal training program after a simple stinger [100]. Muscular strength could be reduced, and weakness can persist for few hours to 6 weeks or more. [101]. As described, superior cervical roots (C5 and C6) and upper trunk are the most affected structures of the brachial plexus. This could be related to several factors such as (i) vulnerability of upper trunk at Erb’s point (supraclavicular region); (ii) direct trauma on nerve structures; (iii) anatomical variability in intervertebral foramina (with smallest at C4-C5 level); (iv) mechanism of injury further reducing the diameter of foramina, like hyperextension with ipsilateral side bending and contralateral cervical spine rotation [99,102,103]. Sideline evaluation of athlete with a suspected BPIs is difficult. In the severe cases, the athlete is assessed during competition and the main clinical problem to rule out is the involvement of cervical spinal cord. The helmet should not be removed and/or a rigid collar should be applied. Bilateral arm signs or involvement of lower limbs are not in agreement with BPIs and should increase the suspicion of a spinal cord involvement. The less severe athletes are commonly assessed after the competition. Muscular strength of C5-C6 muscles should be assessed, such as deltoid, biceps brachii, supraspinatus, and brachioradialis. These muscles are innervated through different nerves raised from brachial plexus and “share” a unique anatomical connection through the upper trunk. The involvement of serratus anterior muscle (long thoracic nerve) and/or rhomboideus muscles and levator scapulae muscle (dorsal scapular nerve) could help to distinguish between lesion of C5-C6 roots and upper trunk. [59] In the most common anatomy, triceps muscle has to be normal (as innervated through middle trunk containing fibers of root C7) and hand muscles too. Numbness is commonly reported in the deltoid region and on the lateral aspect of the arm and forearm to the thumb. Bicipital and brachioradial reflexes are commonly reduced or absent. Patients with muscular weakness and with persistent symptoms should be clinically assessed and followed for at least two weeks. Indeed, in BPIs characterized by neuroapraxia, a complete restoration of functions is usually evident during this period. In case of persistent pain and severe weakness, imaging technique and electrodiagnostic studies should be performed. These data could determine the exact localization of the lesion and are essential to guide the treatment in this phase. Indeed, in axonotmesis and neurotmesis, needle electromyography is able to demonstrate active denervation. Effective reinnervation processes could be recorded in the recovery phase of axonotmesis. In neurotmesis, like root avulsions, imaging techniques are more useful for demonstrating the anatomical damage and to avoid needless delay in surgical treatment. In athletes with axonotmesis, the rehabilitation procedures should be undertaken as soon as possible. The clinical evaluation should guide the assessment of the athlete. Indeed, needle electromyography could demonstrate changes for several months after nerve damage and cannot be used as a method to determine the time to return to play [104]. In hyper acute phase, if the athlete presents transient BPIs lasting for a few seconds and with a complete restoration of motor and sensory symptoms, sport activity can be permitted. If athlete complains of persistent symptoms, he/she should be assessed routinely. Return to play is permitted when sensory symptoms are solved, and muscular strength is restored. It is important to take into consideration the high rate of reoccurrence of stinger, especially in athletes with anatomical abnormalities, like intervertebral foramina stenosis. In these athletes, the signs and symptoms could last longer than usual, and sport activity should be limited. Obviously, in athletes with persistent weakness, intractable pain and severe limitations of cervical spine range of motion, sport activity is prohibited. In severe cases, in which neurotmesis or root avulsion are evident, the treatment is mainly based on a surgical approach. The choice of the equipment, the type of sport activity performed, the role in the team, and some technical advice are useful strategies of prevention that should be practiced in sports at high-risk of BPIs. Severe BPIs are a rare condition in sport medicine. Instead, milder forms of brachial plexus involvement seem more common among athletes, according to the type of sport, the role in the team, and the sporting history, and they should be related to some specific equipment. Severe BPIs are a difficult topic and their management requires a multi-professional team. Clinical investigation is the cornerstone in the definition and localization of injury. Electrodiagnostic studies and imaging techniques provide valuable morphological and functional information able to guide the treatment and to define the prognosis. Rehabilitative approaches should be structured and should include several options of treatment aimed to control pain and to promote the restoration of motor and sensory functions. In most severe cases, specialistic neurosurgical approaches should be performed on peripheral nerves or on tendinous structures to restore functions, which could not be regained conservatively. Conceptualization, I.B., S.P. and F.S.; methodology, I.B., S.P. and A.M.S.; data curation, S.P., B.C. and M.Z.; writing—original draft preparation, I.B., V.R. and B.C.; writing—review and editing, F.S. and M.Z.; visualization, S.P. and F.S.; supervision, S.P. and F.S. All authors have read and agreed to the published version of the manuscript. This research received no external funding. The authors declare no conflict of interest.

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