Published online: 2019-08-13

Prof. Mira Sen (Banerjee) CME Article

Debates to personal conclusion in peripheral nerve injury and reconstruction: A 30-year experience at Chang Gung Memorial Hospital

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ABSTRACT

Significant progress has been achieved in the science and management of peripheral nerve injuries over the past 40 years. Yet there are many questions and few answers. The author, with 30 years of experience in treating them at the Chang Gung Memorial Hospital, addresses debates on various issues with personal conclusions. These include: (1) Degree of peripheral nerve injury, (2) Timing of nerve repair, (3)Technique of nerve repair, (4) Level of brachial plexus injury,(5) Level of radial nerve injury,(6) Traction avulsion amputation of major limb, (7) Proximal Vs distal nerve transfers in brachial plexus injuries and (8) Post paralysis facial synkinesis.

KEY WORDS

Brachial plexus; microsurgery; nerve injury; nerve repair; peripheral nerve

INTRODUCTION

There has been much improvement in peripheral nerve injury and reconstruction since 1970 after the application of the operative microscopy. Improvement includes new technologies of imaging study (computed tomography and/or magnetic resonance imaging), electrodiagnosis (preoperatively and/or intraoperatively), chemistry (histochemical, immunochemical and biochemical), molecular biology (e.g., neurotrophism and neurotropism), pathology, pharmacology. Further advances include; better understanding of peripheral nerve structures (microanatomy), pathophysiology of nerve and muscle (denervation and reinnervation), better magnification (surgical loops and microscope), better instruments (micro-instruments and sutures materials), microsurgical neurovascular skills, and nerve reconstruction strategies such as nerve transfers and functioning free muscle transplantation (FFMT), and more respect of post-operative rehabilitation. The therapeutic approach of the peripheral nerve injury has thus, significantly changed with more optimism in results. However, there are still many questions but few answers to scientists, and many debates, but few conclusions to clinicians in the peripheral nerve science.

I started my microsurgical career in peripheral nerve injury and reconstruction in 1984, after I had 1 year fellowship training with Julia K. Terzis. Till now, I have

Access this article online	
Quick Response Code:	Website: www.ijps.org
	DOI: 10.4103/0970-0358.191295

How to cite this article: Chuang DC. Debates to personal conclusion in peripheral nerve injury and reconstruction: A 30-year experience at Chang Gung Memorial Hospital. Indian J Plast Surg 2016;49:144-50.

performed many reconstructive microsurgeries for peripheral nerve injuries, including over 2000 cases of adult brachial plexus exploration and reconstruction, over 1000 cases of FFMT for different regions (the face, upper and lower limbs), more than 500 cases of obstetrical brachial plexus palsy (OBPP) reconstruction (including early nerve reconstruction and late palliative reconstruction) and enterovirus brachial plexus neuritis treatment. Thousands of surgical cases related to the peripheral nerve injury and reconstruction have been performed, including peripheral nerve sheath tumors, facial paralysis, compression neuropathy such as thoracic outlet syndrome, cubital tunnel syndrome, carpal tunnel syndrome and many peripheral nerve injuries such as radial nerve, median, ulnar, femoral, posterior tibial nerve and common peroneal nerve. As a clinician and scientist, from a rookie to a recognised expert, it takes me about 30 years.

In this CME, I will deal with some debates in the field of peripheral nerve science and make my personal conclusions. Hopefully, this conclusion can partly be your conclusion in the future.

DEBATE 1: DEGREE OF PERIPHERAL NERVE INJURY

It was originally introduced by Seddon in 1943, and was later amplified by Sunderland in 1968 based on the disrupted internal structures. The prognosis for functional return was highly correlated with the degree of nerve injury. Sir Herbert Seddon made the classification of peripheral nerve injury into three types.

Neurapraxia

Neurapraxia affects the myelin sheath largely, causing segmental demyelination, but, no loss of axonal continuity, no Wallerian degeneration, no detectable morphological changes, only a localised conduction block. The, nerve conduction proximal and distal to the lesion is preserved, recovery is rapid and impulse conduction return is completed within days or weeks.

Axonotmesis

Axonotmesis leads to loss of axonal continuity but basal lamina is preserved. The changes show; chromatolysis of cell body, retrograde axonal degeneration for a few millimeters proximal to the lesioned site, Wallerian degeneration of the axons distal to the lesion, complete conduction block, Schwann cell proliferation to form cellular columns (Bands of Büngner) to guide the regenerating axon within the basal lamina tube, nerve sprouts but no neuroma and complete nerve recovery occurs within months.

Neurotmesis

Neurotmesis leads to complete anatomic severance of the nerve, no recovery is expected without surgical co-aptation. There is distal degeneration and some degree of proximal degeneration with neuroma formation.

Sir Sydney Sunderland expanded the Seddon's axonotmesis into two separate degrees of injury based on the ability of the nerve to recover (i.e., completely or partially). He also expanded the Seddon's neurotmesis into other two separate degrees of injury based on nerve lesion in continuity. There were five degrees of peripheral nerve injury: Sunderland 1 to 5. Mackinnon in 1989 added a 6th degree injury, a mixed nerve injury composed of fascicles of varying degrees of nerve injury.

To me, the Sunderland 3rd degree of injury is mysterious. It is related to the timing of nerve exploration. It might be getting better from Sunderland 3 up to Sunderland 2 or 1 by time, or getting worse from Sunderland 3 down to Sunderland 4 or 5 by time. Many researches were focused on the Sunderland 3. Starting from Sunderland 3rd degree injury, it has aberrant re-innervation, causing co-contracture, which is commonly found in the sequelae of OBPP with deformity of the shoulder and elbow, and post-paralysis facial synkinesis (PPFS). By highlighting the Sunderland 3rd degree injury, I intendedly shift the Sunderland 3 into one of the neurotmesis in 2006 [Figure 1] based on the infrastructural site of injury: Sunderland 3rd degree injury is disruption of nerve fiber called endoneurium neurotmesis; Sunderland 4th degree injury is disruption of nerve fascicle called perineurium neurotmesis and Sunderland 5th degree injury is disruption of nerve trunk called epineurium neurotmesis [Figure 1]. My conclusion here is that I still accept the Sunderland classification, but making a different explanation.

DEBATE 2: THE TIMING OF NERVE REPAIR

There are many controversies related to the timing of nerve exploration. There are no absolute rules but depends on the type of injury, patient condition, associated injury and others. The general principle is that, more delay will cause greater scars at the lesioned site and more central neuron death due to the absence of neurotrophic factors. This results in higher end organ

(skin and muscle) degeneration and poor recovery even after the nerve repair. However, the earlier repair sometimes causes poor results due to uncertain stump health. Because of these, I classify them into five types based on the timing of nerve repair.

Immediate nerve repair or repair within (2) days

Indicated in sharply transected nerve with open wound which results in sensation or motor loss. If it is associated with arterial rupture, the nerve repair can be performed along with immediate vascular repair or delayed repair within few days.

Early nerve repair within a month

Indicated in already closed wounds with known nerve severance , which was not repaired during primary wound exploration. The clinical history and evaluation alone is sufficient and further investigations are not needed. For example, radial nerve palsy associated with humeral fracture which has been openly reduced and radial nerve severance was noted, but without repair or infraclavicular brachial plexus injury (BPI) with vessel rupture which has been repaired.

Delayed early nerve repair within 5 months

Indicated in close traction wounds with unknown nerve injury, such as a closed type of BPI and radial nerve injury (RNI) and ,if there is a stagnation of Tinel's sign following 2–3 months of follow-up.

Late nerve repair more than 6 months after injury

Nerve repair is still possible, but additional procedure to enhance the result is often required. Longer rehabilitation is anticipated.

Degree of Nerve Injury (Chuang 2006)

Seddon (1943)	Sunderland (1951)	Chuang (2006)
Neurapraxia	1	1 (Segmental demyelination)
Axonotmesis	2	2 (Nerve axon disruption)
	3	
Neurotmesis	4	3 endoneurium neurotmesis : Nerve fiber disruption
		4 Perineurium neurotmesis : Nerve fascicle disruption
	5	5 Epineurium neurotmesis : Nerve trunk disruption

Figure 1: Degree of nerve injury with authors classification

Chronic nerve repair 1 year after injury

It is usually due to late referrals or presentation, failed recovery following nerve repair, and in complications of associated injury/ treatment such as head injury, electric burn, osteomyelitis, abdominal surgery complication, joint stiffness, pain, psychiatric disturbance, etc. In these cases, staged reconstruction is usually required. The examples include: nerve elongation first, followed by FFMT, or tendon ,or muscle transfer, or arthrodesis for palliative reconstruction. Long-standing facial paralysis and reconstruction is one of the good examples.

DEBATE 3: TECHNIQUE OF NERVE REPAIR

There are four types of nerve repairs: epineurial, perineurial, epi-perineurial and group fascicular repairs. There is still no consensus with regard to which one is superior to another. To me, it is always not a significant factor. The important point is the accuracy of the suture approximation. The following conditions are frequently encountered during the nerve repair:

1. One-to-one nerve suture with similar size or mild discrepancy of stumps [Figure 2]
2. One-to-one nerve suture with a significant discrepancy of stumps: using pencil-shaped trimming technique [Figure 3]
3. Multiple small nerves to one big nerve suture: using pencil-shaped trimming technique [Figure 4a and b].

Keep points for nerve suture:

1. Epineurotomy first: Resect (strip away) the external epineurium (several layers of loose connective tissues) circumferentially until the layer of normal epineurium (internal epineurium) with longitudinally oriented vessels on it; stop the bleeders with micro-bipolar forceps.

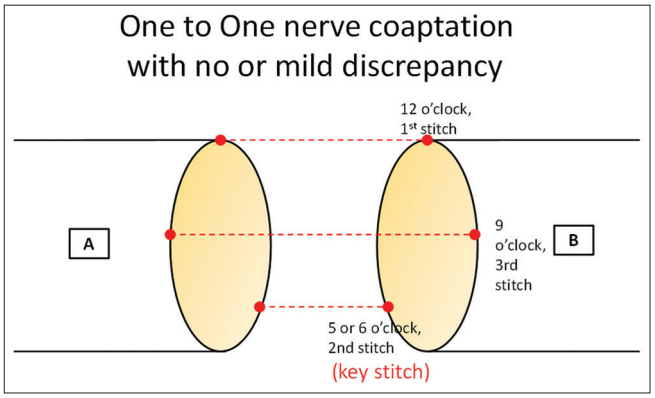

Figure 2: One-to-one nerve suture with no or mild discrepancy

2. Stitch always sutures at internal epineurium layer (or epi-perineurium layer) in acute stage because the perineurium is too thin to be separated from the internal epineurium.
3. The 2nd stitch is a key stitch following the 1st stitch, taken always at the posterior wall of both stumps to invert the stump surface; then apply the 3rd stitch for the opposed site.
4. Usually, 2–3 stitches are enough for stump approximation.
5. Always perform tension-free suturing. The nerve graft length is not a significant factor as we might think. Interposition grafting has been shown superior to suturing under tension.
6. Number of cable grafts for bridging nerve gap: for median nerve - 5–6; radial or ulnar nerve - 3–4; musculocutaneous nerve - 3; axillary nerve - 2; suprascapular nerve - 1 nerve graft.

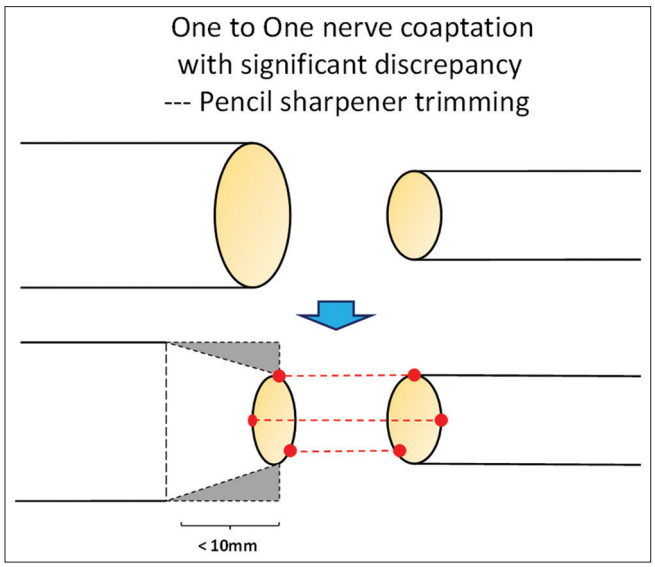

Figure 3: One-to-one nerve suture with significant discrepancy of stumps using pencil-shaped trimming technique

DEBATE 4: LEVEL OF BRACHIAL PLEXUS INJURY

Various classifications of the level of BPI have been proposed; for example, two levels as supraclavicular and infraclavicular; three levels as supra-, retro- and infraclavicular and four levels as pre-ganglionic root, post-ganglionic root, trunk and division, cord and terminal branches, etc. The most confusing aspect is the so-called post-ganglionic root. In fact, after the dorsal root ganglion, both ventral and dorsal roots continue for only a few millimetres (<5 mm) distance and unite to become a mixed nerve where it is no longer a root. Therefore, the components of the brachial plexus are roots, spinal nerves, trunks, divisions, cords and terminal branches. To avoid anatomical confusion, in Chang Gung Memorial Hospital, we have described brachial plexus lesions with 'number', Level I-IV, instead of word description[1] [Figure 5].

Level I injury

Inside the (vertebral) bone:, It is a pre-ganglionic root injury, including spinal cord, rootlet and root. It requires laminectomy to see the nerves.

Level II injury

Inside the (scalene) muscle: It is a post-ganglionic spinal nerve injury, located at the interscalene space proximal to the suprascapular nerve. It requires segmental resection of the scalene anterior muscle to see the nerves.

Level III injury

Pre- and retro-clavicular: It includes trunks and divisions. It requires an osteotomy of the clavicle to see the nerves.

Level IV injury

Infraclavicular: It includes cords and terminal branches injury proximal to the axillary fossa. It is usually an

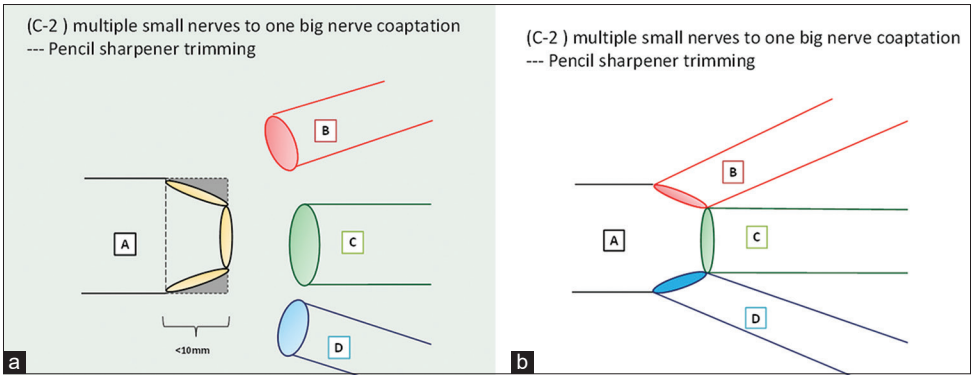

Figure 4: (a and b) Multiple small nerves to one big nerve suture: Using pencil-shaped trimming technique

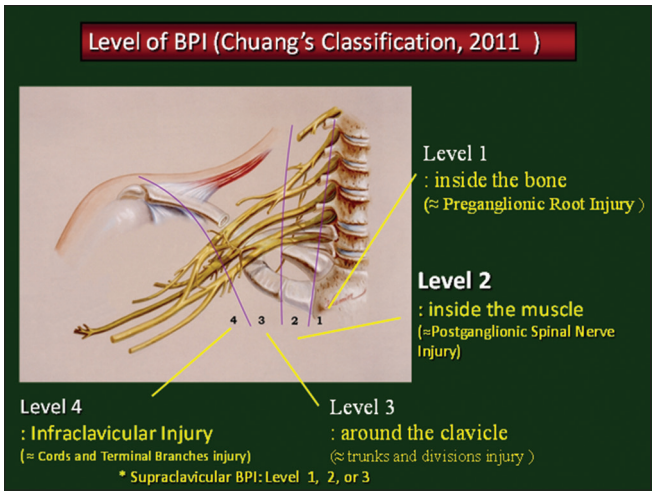

Figure 5: Brachial plexus lesions Level I-IV- authors classification

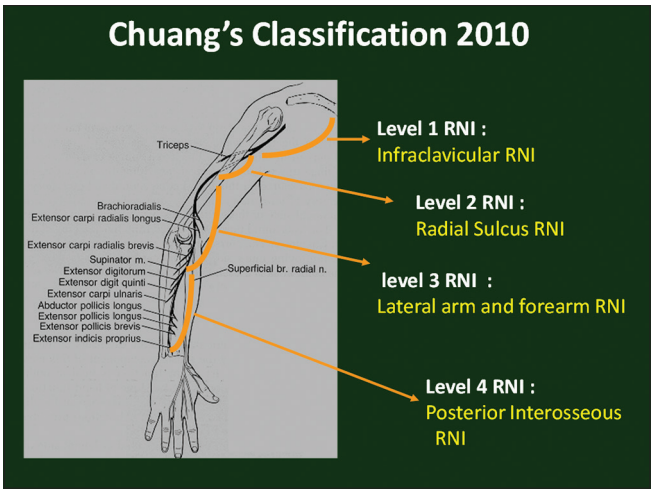

Figure 6: Four levels of radial nerve injury-authors classification

isolated level injury with a high incidence of scapular fracture, vascular injury and glenohumeral dislocation. Difficult dissection and long nerve grafts are frequently encountered.

The term 'supraclavicular BPI' will cover a large zone of injury, including Level I, II or III lesions.

DEBATE 5: LEVEL OF RADIAL NERVE INJURY

The radial nerve receives nerve fibres from C5 to C8 (and occasionally T1) nerve roots. The definition of the radial nerve should theoretically include the infraclavicular radial nerve as a whole. Based on this, four levels of RNI have been classified by their anatomical characteristics and related clinical pictures[2] [Figure 6].

Level I injury: Infraclavicular radial nerve injury (from emergence of the posterior cord infraclavicularly to the inlet of the humerus spiral groove)

Infraclavicular RNI may cause all palsies of elbow, wrist and digital extensions.

Level II injury: Humerus spiral groove radial nerve injury (from inlet to outlet of the spiral groove of the humerus)

Injuries here are very often accompanied with humerus bone fracture and cause palsies of the wrist, finger and thumb extension. However the elbow extension is usually spared, as nerves to the long and/or to the medial heads branches out before entering the groove.

Level III injury: Lateral arm and antebrachial fossa radial nerve injury (from outlet of the radial nerve to the humeroradial joint)

Fractures of the middle and middle-distal parts of the shaft have a significantly higher association with radial nerve palsy. RNI in this level may cause palsies of thumb and finger extension. The wrist extension may or may not be spared, and if spared, it will induce wrist extension.

Level IV injury: Posterior interosseous nerve radial nerve injury (terminal radial nerve branch distal to the branch to extensor carpi radialis brevis)

RNI in this level will cause palsies of thumb and finger extension, but the wrist extension is intact with radial deviation.

DEBATE 6: CLASSIFICATION OF TRACTION AVULSION AMPUTATION OF THE MAJOR LIMB

Traction avulsion amputation results from traction force and cause bone, muscle and skin avulsion amputations at different levels. Traction avulsion amputation of the major upper extremity is distinguished both clinically and prognosticatively from the guillotine or circular amputation, in which all tissues are sharply divided at the same level. With the advent of FFMT, the secondary reconstruction for the residual deformities of patients with traction avulsion amputation has been approached aggressively for better functional outcome. Through this, a new classification system was developed to

reflect the pattern of injury, management, which has a prognostic significance.[3] The classification is based on the disruption points of the muscles and their innervated nerves , but not on the level of bone fracture [Figure 7].

Type I amputation

Avulsion at or close to the musculotendinous aponeurosis with the muscle remaining intact and functional. Tendon to muscle suturing is feasible.

Type II amputation

Here, there is avulsion within muscle bellies, but distal to the neuromuscular junction with the proximal muscles still being innervated. The motor nerve refers to the anterior interosseous nerve in the forearm and the musculocutaneous nerve in the arm. Muscle-to-muscle sutures are mostly useless. Half of the patients need 2nd stage of functioning muscle transplantation.

Type III amputation

Here, there is avulsion within the muscles, but proximal to the neuromuscular junction with the entire muscles being denervated. In this type of amputation, remove the whole muscles and try to close the wound primarily. All these require 2nd stage functioning muscle transplantation.

Type IV amputation

Amputation through the elbow or shoulder joint (disarticulation amputation): external fixation, immediate flap (skin or myocutaneous flap) for wound coverage after vessel and nerve repairs are required.

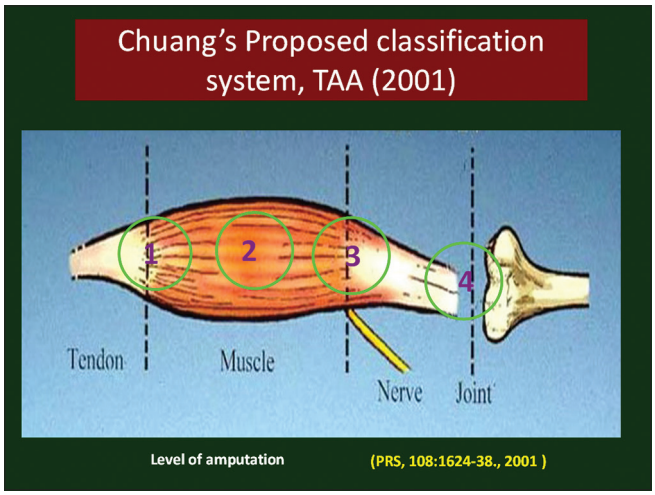

Figure 7: The classification of level of amputation is based on the disruption points of the muscles and their innervated nerves

DEBATE 7: PROXIMAL NERVE TRANSFER VERSUS DISTAL NERVE TRANSFER IN BRACHIAL PLEXUS INJURY

The proximal nerve transfer is the nerve transfer, when nerve co-aptation is performed within the brachial plexus zone (supraclavicular or infraclacivular fossa); distal nerve transfer is the nerve transfer, where the nerve co-aptation site is close to the neuromuscular junction (close-target nerve transfer) outside of the brachial plexus zone. The proximal nerve transfer, a traditional approach basically for both diagnosis and treatment, has the distinct advantage of the large amount of axons available for transfer if a healthy spinal nerve is found, and relies less on brain plasticity as native function is used for reconstruction. Proximal nerve transfers demand supraclavicular or infraclavicular scar dissection and identify healthy available spinal nerves for nerve grafting and nerve transfer. The distal nerve transfer is basically for surgical treatment. The distal nerve transfer avoids the use of a nerve graft, is technically much easier to perform and might prevent some of the target muscle atrophy due to shorter regenerative distance and time. However, the distal nerve transfer might sacrifice some of the donor functions and provide a smaller number of donor axons compared to proximal nerve transfer. The theoretical advantages and disadvantages of both treatments were debated extensively in the literature.

The conclusion here is, we advise combined approaches. If indicated, supraclavicular (or infraclavicular) approach to obtain accurate diagnosis, proximal nerve transfers to achieve shoulder function, and then distal nerve transfer in incomplete root avulsion of BPI to achieve a quick elbow function.

DEBATE 8: POST-PARALYSIS FACIAL SYNKINESIS

Related to the disputed terminology

There are many similar terms to describe the same defect such as post-paralytic facial nerve syndrome (PPFS), facial synkinesis, hemifacial synkinesis, aberrant facial nerve regeneration syndrome, secondary hemifacial spasm, post-facial palsy synkinesis, post-paralytic facial synkinesis, post-facial paralysis synkinesis, or post-paretic facial synkinesis, regenerated post-paralytic facial nerve syndrome, or contractures and synkinesis of the facial muscle. Since PPFS represents a wide spectrum

of unwanted facial movements after recovery of facial palsy from any aetiology, 'PPFS' is more accurate for its terminology.

Related to the treatment

PPFS is a healing process of facial nerve injury with various presentations. It is like scar formation, which is also an end result of a healing process. Not all scars are treated conservatively, some bad and ugly scars should be treated by a more aggressive procedure, such as wide excision and reconstruction with local or distant flap. Likewise, treatment of PPFS is the same. Some severe types require more aggressive approaches, such as radical excision of the synkinetic muscles (myectomy) and synkinetic nerves (neurectomy) and reconstruction by functioning free muscle for facial reanimation, like the treatment for chronic facial paralysis. I have started its observation and treatment since 1986. We classify the PPFS into four patterns (I-IV) based on the quality of the smile and the degree of synkinesis[4]:

- Pattern I: Good smile (at least 4 teeth visible) and mild synkinesis
- Pattern II: Acceptable smile (2–3 tooth visible) but moderate to severe synkinesis
- Pattern III: Unacceptable smile (0–1 teeth visible) and severe synkinesis
- Pattern IV: Poor smile (no tooth visible) but accompanying with mild synkinesis.

For the pattern I and some pattern II patients, I do treat them with Botox. These patients usually show a short-term follow-up (<2 years) after first-time injection. They keep seeking further treatment. They just do not want to have repeated injections for their follow-up period, even after explaining to them that, Botox injections are only for symptomatic relief, and not for the cure. Yet, some of them were treated also by the myectomy of orbicularis oculi, platysma, corrugators or zygomaticus major muscle to decrease the requirement of frequent Botox injections.

Only pattern III and some pattern II patients required more aggressive procedure. I have advocated taking more aggressive approaches for PPFS treatment if the patients are aiming at good and long-term results, in comparison to only Botox and rehabilitation treatment. They often require extensive myectomy and neurectomy and followed by gracilis FFMT transfer, with innervations from the cross face nerve graft, or spinal accessory, or masseter nerve. Removing the trigger muscle(s) or target muscles can effectively and significantly decrease the synkinesis. This is the principle of myectomy. Removing the innervated nerve(s) can paralyse the synkinetic muscles, and consequently decrease the degree of synkinesis. This is the principle of selective neurectomy. BT-A injection, rehabilitation and other additional aesthetic surgeries are only our adjuvant therapies to improve the result. But to the surgeons, correction of synkinesis should always be prior to the treatment of asymmetry.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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