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The cranial connection and the neuropathic process

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Narrative: This paper presents meticulous clinical instruction on the advanced-level diagnosis and treatment of presentations involving the cranial ventricles through highly targeted cranial adjusting.

The importance and clinical relevance of the Shimizu reflex is demonstrated along with impeccable approaches to relevant head and neck musculature.

The technique of choice is the NeuroImpulse Protocol,™ a low force and gentle technique which is specific in its application. Throughout the text, important clinical points are highlighted.

This paper is taken from Module 9 of the NeuroImpulse learning materials© and manual and provides the busy practitioner with a clean and tidy clinical approach to cranial adjusting in the young.

Indexing Terms: Chiropractic, Clinical management; Cranial; NeuroImpulse Protocol; Shimizu reflex.

The philosophy of cranial adjusting

Like all other adjustive procedures in chiropractic, the goal of cranial adjusting is to restore functional neurological balance. Neurological deficits, demonstrated by use of the standard, orthodox neurological examination, coupled with precise, detailed patterns of kinesiopathology throughout the body, now dictate the need to apply the adjustment to the bones of the cranium or any other structure which has the capacity to influence longitudinal tension in the dural membrane with consequent increase in the CSF pressure.

... A bilateral Shimizu reflex will be universally demonstrated in the presence of the cranial subluxation. NIP chiropractic is uniquely placed to interpret and address this clinical sign ...

A cranial adjustment is a potentially potent neurological stimulus and needs to be performed only when the confluence of examination findings indicate the need for such a procedure. The cranium is a very delicate structure. It should be handled with the greatest possible care, based on a thorough understanding of its mechanics, thus a technique that is gentle and with very limited force is used in normalising cranial function. (Wales, 1990)

The physiology of cranial adjusting

Cranial diagnosis and adjusting is based on the respiratory driven movements found throughout the entire body. A 'cranial subluxation' when defined as dysfunctional cranial movement coupled with specific neurological deficits may be positively influenced by the application of an adjustive impulse anywhere in the body.

Clinical experience suggests that apart from direct application of adjustive impulse to the skull to influence the function of the sphenobasilar mechanism in particular and the cranial bones in general, the cranial subluxation may be maximally influenced by applying adjustive impulse to the feet, the sacrum, the diaphragm or the pelvic floor.

To understand the physiological principles upon which cranial adjusting is predicated, it is essential to consider in depth the elements that constitute the primary respiratory mechanism.

Important clinical point

A cranial subluxation may be defined as dysfunctional cranial movement coupled with specific neurological deficits and may be affected by the application of an adjustive impulse anywhere in the body, but particularly at the feet, sacrum and diaphragm.

The primary respiratory mechanism (PRM)

Dr Sutherland observed 5 basic elements at work in the human body and he called these the '*five phenomena of the primary respiratory mechanism*'. The word '*primary*' was used as it indicated something that was basic or first. The word '*respiratory*' referred to metabolism or physiological respiration. He considered the human body to be a complex '*mechanism*' (a grouping of parts working together towards a definite action), hence the use of this word.

Though primary respiration has 2 phases, inhalation and exhalation, this is a separate concept from and not to be confused with secondary respiration. Secondary respiration refers to the process produced by movement of the primary and secondary muscles of respiration that results in the exchange of oxygen for carbon dioxide at the pulmonary alveoli. Primary respiration is a deeper, more basic process to life. The primary respiratory mechanism has two alternating phases, the inspiratory and the expiratory phases, also respectively known as the flexion and extension phases (Magoun, 1951).

For the sake of study and the ease of clinical application, the cranial bones are grouped into those that principally flex and extend (occiput and sphenoid) and those that externally and internally rotate as they flex and extend (temporal, parietal and frontal). During the inspiratory phase of primary respiration, as the midline bones flex, the peripheral bones externally rotate resulting in an increase in the transverse cranial diameter and a decrease in the anteroposterior diameter, (Di Giovanni & Schiowitz, 1997)

In addition, the basicranium and foramen magnum move superiorly which in turn draws the sacral base posterosuperiorly as a result of the changing tensions in the dural sac. During the expiratory phase of primary respiration, the midline bones extend, the peripheral bones internally rotate resulting in a decrease in the transverse cranial diameter and an increase in the anteroposterior diameter while the basicranium and foramen magnum move inferiorly thus drawing the sacral base anteroinferiorly.

The five phenomena of the PRM described by Sutherland (1988) are as follows:

- Fluctuation of the cerebrospinal fluid (CSF);
- Mobility of the intracranial and intraspinal membranes and the function of the reciprocal tension membrane (RTM);
- The inherent motility of the central nervous system (CNS;)

- Articular mobility of the cranial bones; and
- The involuntary mobility of the sacrum between the ilia.

The rhythmic fluctuation of the cerebrospinal fluid

CSF fluctuation is considered the first principle of the PRM. Movement of the CSF involves both circulation and fluctuation. Circulation occurs as a result of hydrostatic forces at the choroid plexus and arachnoid granulations (Di Giovanni & Schiowitz, 1997). However, forces generated by hydrostatic gradients are not sufficient in and of themselves to account for the exchange of CSF with the circulation of the body. Indeed, fluctuation within the CSF provides this force. The CSF fluctuates, or moves back and forth within a relatively closed container, the central nervous system.

As the brain and spinal cord change shape with the cycles of inhalation and exhalation, the CSF fluctuates back and forth in the spaces in the brain and spinal cord. As the brain is constantly producing CSF, a small excess travels out along the channels around the peripheral nerve trunks during the exhalation phase of primary respiration.

The CSF plays an important role in circulation and nourishment of body tissues. Fluctuation of the CSF has now been documented in MRI studies. (Schroth & Klose, 1992) This fluctuation provides a continuous mixing which, combined with the small circulatory forces, allows for adequate exchange of the CSF with the circulation of the body. The fluctuation of the CSF and cranial articular motion coincide under normal resting conditions. In addition, CSF fluctuation, changes in the contours of the central nervous system and motion of the craniosacral mechanism are synchronous at such times.

The motion of the Dural Membranes

The second principle of the PRM involves the reciprocal tension membranes (RTM), otherwise known as the meninges, which are made up of the duramater, arachnoidmater and piamater. Together they are the agencies for articular mobility of the cranial and craniosacral mechanisms, creating balance in all dimensions; aiding, controlling and limiting motion. Basically, the intracranial and intraspinal membranes allow a range of motion in the bones that are suspended within them in much the same way ligaments allow a range of motion of the joints of the spine and extremities.

All membranes change shape during the phases of the PRM. During the inspiratory phase, the anterior end of the falx cerebri moves slightly posteriorly and inferiorly, the tentorium shifts slightly anteriorly, and the craniosacral mechanism functions so that the spinal dura lifts the sacrum around its axis so that the base is superior and the apex anterior. The exact opposite occurs during the expiratory phase to complete the cycle.

Membranous structures in the body are all composed of connective tissue and are derived from the embryological mesenchymal layer. They are all continuous with one another with the intracranial membranes closely related to the rest of the body via fascial connections from the cranial base throughout the entire spine, the diaphragm, the extremities and viscera.

In the neonate and beyond into infancy, there are no interlocking sutures in the skull. The only functional joints in the neonatal skull are those of the occiput with the atlas vertebra (occipito-atlantal). The cranial bones of the newborn are suspended in space by the dural membrane and the pressure applied by the CSF. Developmentally, prior to completed bone formation, it is the membranes that house, protect, guide and limit motion.

Important clinical point

Dural membrane tone is maintained within critical parameters in the human body. Any variation outside these critical levels results in altered CSF pressure and therefore dysafferentation.

Inherent motility of the Central Nervous System (CNS)

The third principle of the PRM concerns the brain and spinal cord. The CNS has a 'jelly fish' type of mobility which has long been recognised by the scientific community. (Enzmann et al, 1992; Grietz et al, 1992; Poncelet et al, 1992). This mobility has a mechanical function in the operation of the PRM. The mobility of the bones of the skull is accommodative to that motility and therefore to the subsequent fluctuation of the cerebrospinal fluid.

This jelly fish mobility is evident as a very slight coiling (roughly mimicking its embryological state) during the inhalation phase of primary respiration with a shortening from top to bottom (decreased cephalad to caudad length) of the spinal cord. The transverse diameter of the cranium becomes slightly wider (increased) and shorter from front to back (decreased AP diameter). The exhalation phase of primary respiration produces the exact opposite (Di Giovanni & Schiowitz, 1997; Magoun, 1951). This expansion and contraction of the cranium is not large, but it is physiologically significant. Estimates place the change at hundredths of an inch, but it varies according to where it is measured. There are cavities and spaces in and around the CNS, and as the brain and spinal cord change shape with the inherent rhythmic motion, the volume of these spaces and hence the amount of fluid that they hold will change. This type of motion is not limited to man, but is a basic and vital property of any living organism with a nervous system.

Articular mobility of the Cranial Bones

The fourth principle of the PRM involves the articular mobility of the cranial bones. The cranium is made up of 26 bones in total and they are all in slight rhythmic motion along with the CNS, CSF, membranes, and sacrum. These bones all fit together like the gears of a watch and influence each other. The cranial sutures contain connective tissue, nerves and blood vessels. They are like any other joint in the body and as such they are designed to facilitate motion.

The cranium of the newborn is predominantly cartilage and membrane without sutures between the bones as in the adult. By 13 years of age there is a moderate degree of suture formation and this is complete by approximately 18 years of age. As the skull ossifies, sutures are formed in response to forces exerted by adjacent bones on each other, the shape of the suture being consistent with the direction of inherent mobility. Hence, the motion of a bone may be deduced by the shape of its suture with adjacent bones. When an axis of motion crosses a suture line, that suture will form in the shape of a bevel (i.e. the squamosal suture) since motions on the opposite sides of an axis are different. These points are called pivots.

Cranial bone motion will also exert a developmental effect on the shape of the facial bones (Magoun, 1951) but for the purposes of this module, only the occiput, sphenoid, temporal, parietal and frontal bones will be discussed in detail.

The Sphenoid

The sphenoid bone consists of the body, the greater and lesser wings laterally, and the pterygoid plates inferiorly. The sphenoid articulates with 12 other cranial bones being the occiput, temporal bones (2), parietal bones (2), frontal bone, ethmoid, palatine bones (2), vomer and zygomae (2).

The palatine bones, the vomer and the zygomae are intermediary between the sphenoid and the maxillae, making the sphenoid influential in the motion of the frontal and facial bones.

Although all the cranial bones move together during respiration, the sphenoid and occiput principally work together to induce flexion and extension about the midline at the articular junction of the sphenoid and the basilar process of the occiput (Fig 1) referred to in its functional capacity as the '*sphenobasilar mechanism*'. On inspiration, this mechanism moves into flexion and on expiration it moves into extension.

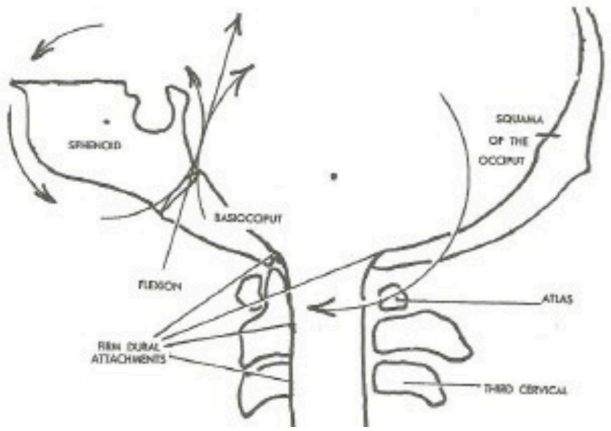


Fig 1: The physiological movements involved in flexion of the sphenobasilar mechanism. These movement occur during inspiration. Extension, which occurs during expiration, involves movements in exactly the opposite directions to those shown above. (Adapted from Magoun HI, Ed,1966 Osteopathy in the Cranial Field. The Journal Printing Company, Kirksville, Missouri p. 39).

The Occiput

The occiput is in four parts at birth, namely the squamous, basilar, and two lateral condylar parts. The base of the occiput articulates anteriorly with the base of the sphenoid. The occiput articulates with 6 other bones, namely the sphenoid, the parietal bones (2), the temporal bones (2) and the atlas vertebra. The occiput, like the sphenoid, moves primarily into flexion during inspiration and extension during expiration.

The Temporal

At birth the temporal bone consists of the petromastoid portion and the squamous portion. The petromastoid part is developed in cartilage that projects obliquely between the occiput and the greater wing of the sphenoid to articulate at its apex with the body of the sphenoid. This portion contains the auditory and vestibular apparatus. The squamous part is developed in membrane and forms the greater part of the lower lateral wall of the skull (Fryman, 1976). The squamous and tympanic parts unite just prior to birth.

The temporal bone forms articulation with the occiput, parietal, sphenoid, mandible and zygomatic bones on each side of the cranium. The temporal bones primarily externally rotate during inspiration, and internally rotate during expiration.

The Parietal

The parietal bone only ever has one part. The parietal bones articulate at the sagittal suture with each other along with the frontal, sphenoid, temporal and occiput. Like the temporal it moves into external rotation during inspiration and internal rotation during expiration.

The Frontal

The frontal bone consists of the squama, orbital and nasal parts. The external or frontal surface of the squama is divided by the metopic suture, running from the nasion through the glabella to the bregma. The frontal bone articulates with the sphenoid, parietal bones, ethmoid, lacrimal bones, maxillae, nasal bones and zygomae.

Like all peripheral plates, the frontal bone primarily externally rotates during inspiration and internally rotates during expiration. It should be noted that as the peripheral bones externally and internally rotate it is within the context of flexion and extension.

So therefore, they primarily externally rotate while flexing (inspiration) and internally rotate while extending (expiration).

The articular mobility of the Sacrum between the Ilia

The fifth and final principle of the PRM involves the articular mobility between the sacrum and the ilia. The dural membrane is firmly attached to the base of the skull, the sacrum and coccyx resulting in the motion of the cranial mechanism being transmitted to the sacrum. The end result of this is that the cranium and the sacrum work together as a functional unit.

The involuntary motion of the sacrum is about a transverse axis anterior to the sacral canal, through the body of S2 at the junction of the short and long arms of the L-shaped sacroiliac joints. (Di Giovanni & Schiowitz, 1997) The anterosuperior movement of the foramen magnum during the inspiratory phase of the PRM lifts the spinal dural membrane, a function referred to as the 'core link' between the cranial and pelvic bowls (Magoun, 1951). The effect of the lift is to rotate the sacrum about the transverse axis at S2 whereby the base of the sacrum moves posterosuperiorly and the apex moves anteroinferiorly towards the pubic symphysis. The opposite occurs in the expiratory phase to complete the cycle.

Principles of diagnosis

The key diagnostic decision that needs to be made is whether or not a given patient has need of a cranial adjustment as the primary approach to their pattern of dysafferentation. This decision is based on the demonstration of a confluence of kinesiopathology and neurological deficits. There are four key patterns of kinesiopathology.

In its original concept, the decision to make a cranial adjustment depended upon the ability of the individual clinician to feel the various rhythms of movement inherent within the PRM. Recent research has seriously called into question the validity of a palpatory cranial diagnostic method based on the ability of the clinician to appreciate the rhythm of movement inherent within the PRM (Hanten et al, 1998; Moran & Gibbons, 2001; Sommerfield et al, 2004; Wirth-Pattullo & Hayes, 1994). As a result of this research, the notion of making a diagnostic decision based on palpating 'cranial rhythm' has been abandoned in favour of a radically different approach that relies on the demonstration of a pattern of kinesiopathology that correlates with a series of predictable neurological deficits. There are four key patterns of kinesiopathology.

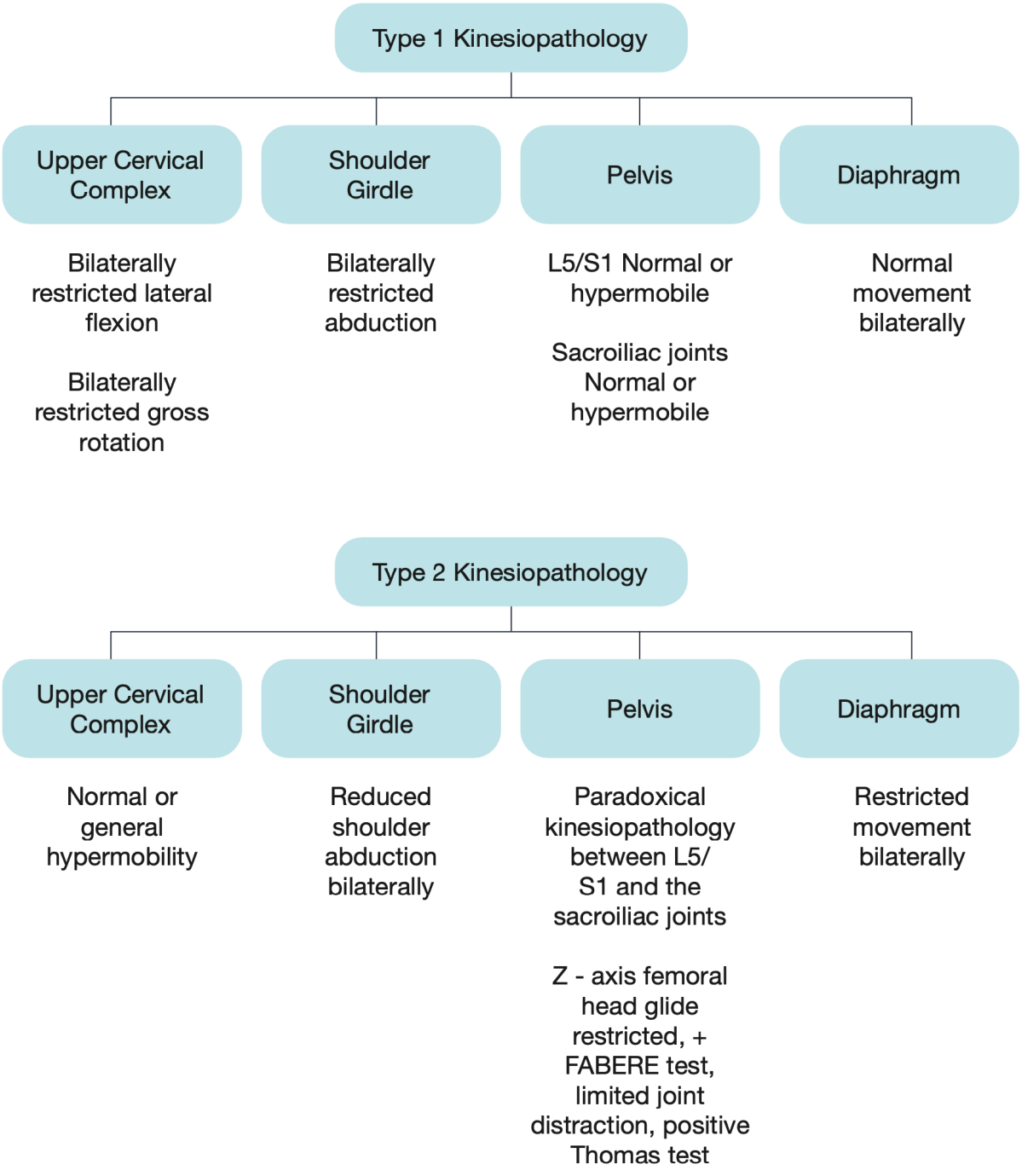
Important clinical point

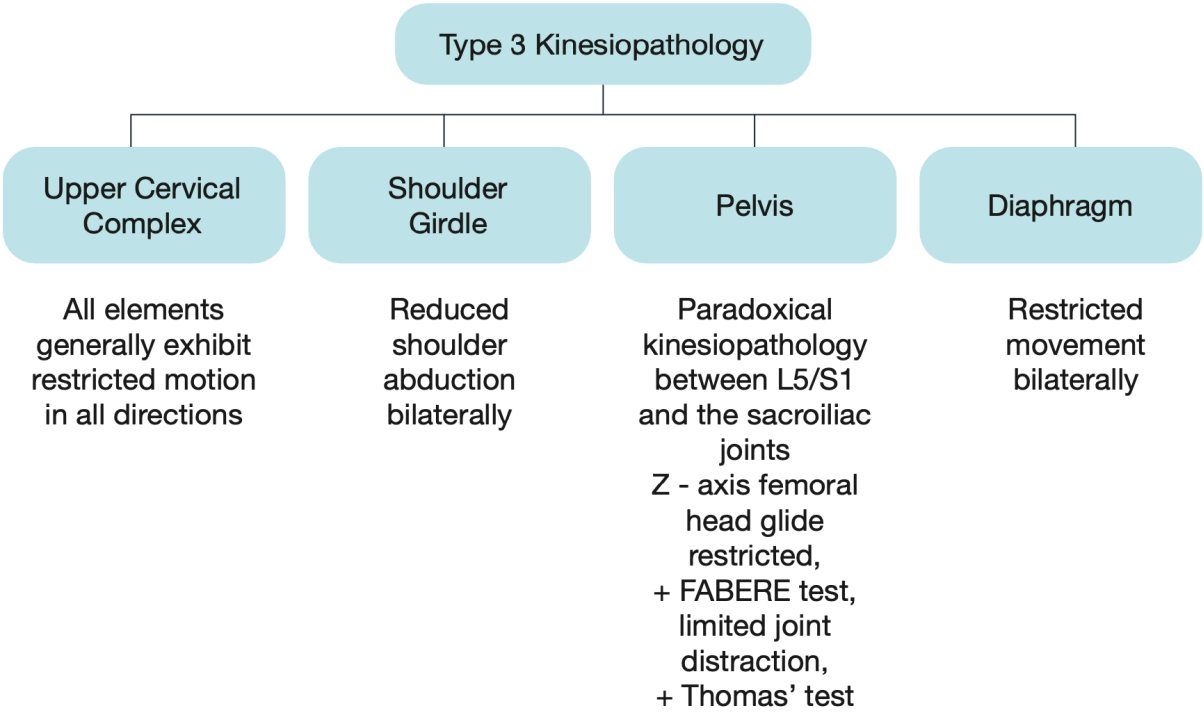
The diagnostic methodology utilised in today's world of neurological chiropractic is based on a sophisticated protocol which involves the identification of specific patterns of confluence of kinesiopathology and neurological deficits.

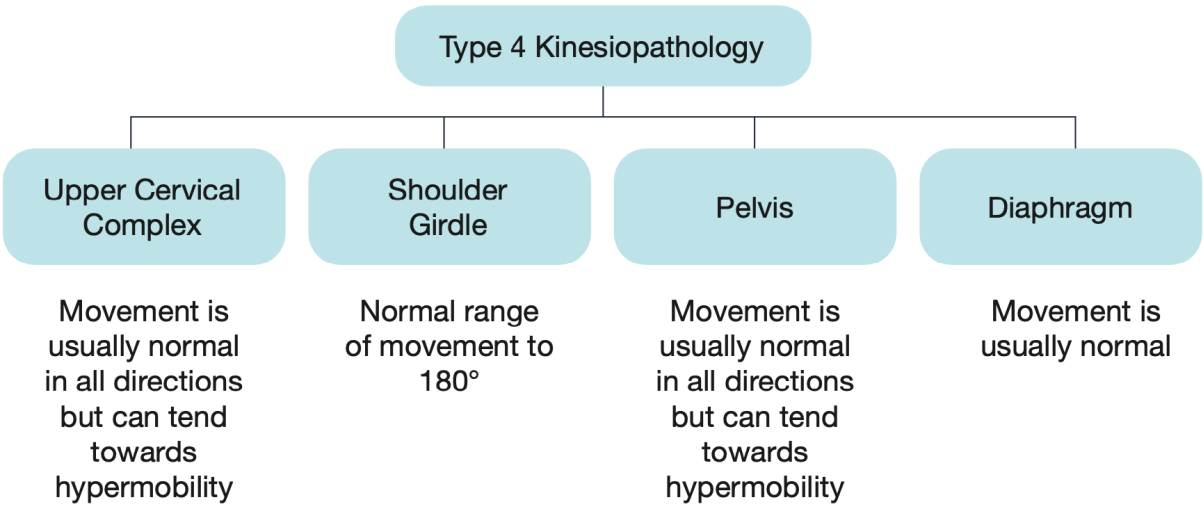
Key patterns of kinesiopathology

The method of biomechanical assessment that leads to the identification of patterns of kinesiopathology has been exhaustively covered in Modules 3 and 4. From that examination, the four patterns of kinesiopathology detailed in the flow plans shown as Table 1 emerge as being highly suggestive of a cranial problem. Bear in mind that once identified, specific patterns of neurological deficits also need to be demonstrated before a definitive diagnosis is arrived at.

Table 1: The four patterns of kinesiopathology and neurological deficits suggestive of a cranial subluxation in the NIP™ model.







Neuropathology

When a cranial subluxation is the cause of one of the four patterns of kinesiopathology described in Table 1, various neurological deficits will be seen.

Neurological deficits associated with the Cranial Subluxation

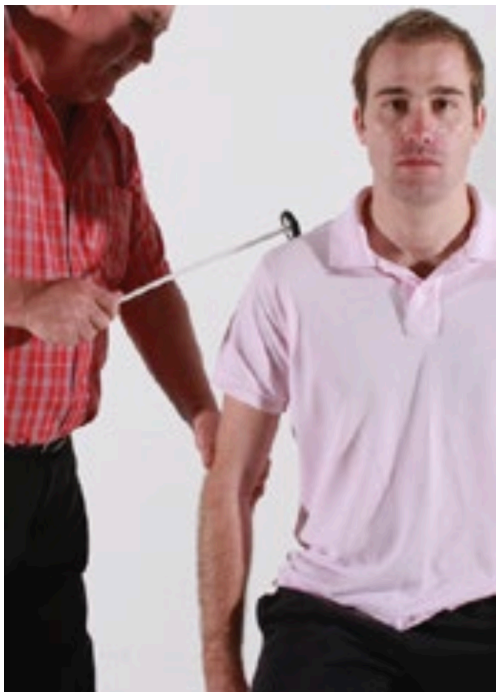
Shimizu Reflex

A bilateral Shimizu reflex will be universally demonstrated (normally not able to be elicited) in the presence of the cranial subluxation. This reflex (otherwise known as the scapulohumeral reflex of Shimizu) response is elicited by striking the lateral third of the spine of the scapula in an anteroinferior direction. The reflex can also be elicited by striking the acromion process in a caudal direction.

The reflex should not be considered absent unless both the spine of the scapula and the acromion process have been struck to no effect either in the clear or following examination on

full, sustained inspiration and expiration. The Shimizu reflex is considered to be clinically significant when elevation of the scapula, abduction of the humerus or both appear following the strike of the reflex hammer. (Shimizu et al 1993) The methodology of performing the Shimizu reflex test is shown in Fig 2.

Fig 2: The method of eliciting the Shimizu reflex

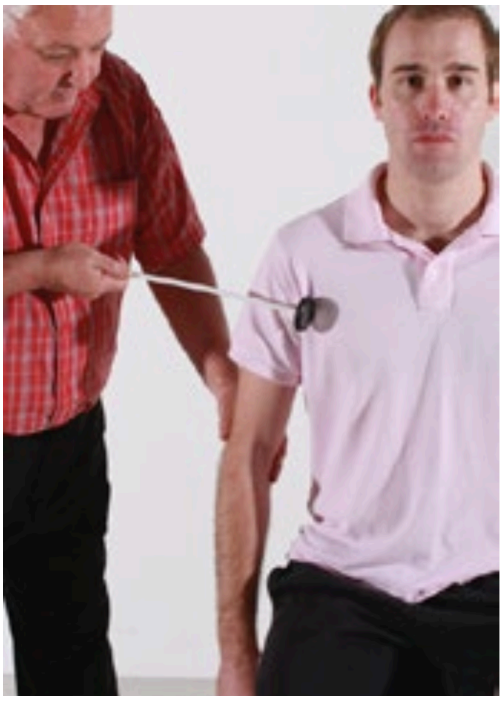


Pectoral Reflex

This reflex is elicited by striking the tendon of the *pectoralis major* muscle at the anterior margin of the axilla as shown in Fig 3. A hyperactive pectoral reflex, like the Shimizu reflex, is suggestive of upper cervical cord compression. (Watson et al 1997) In the presence of the cranial subluxation, a bilateral hyperactive pectoral reflex response is obligatory.

The notable and clinically important exception to this rule is the dropped sphenoid, either unilaterally or bilaterally.

Fig 3: The method of eliciting the Pectoral Reflex



Cranial Nerve XI Deficit

In children who are of schoolage and older along with adult patients, the XIth cranial nerve may be tested and will be found to be universally deficient bilaterally in the presence of the cranial subluxation.

The preferred method for testing the XIth cranial nerve is by direct evaluation of the upper trapezius muscle as shown in Fig 4.

Fig 4: Technique for testing the strength of the *upper trapezius* muscle. This muscle derives its nerve supply exclusively from the XIth cranial nerve and weakness implies a mechanical lesion at the level of the nerve roots in the brainstem.

The Ocular Function Tests

The assessment of ocular movement, intraocular pressure, conjugate response to circular tracking and most importantly, past pointing under direct proprioceptive stress are critical to our understanding and measurement of the cranial subluxation in which an increase in CSF pressure and shunting from side to side has occurred.

These tests will not only assist us in determining the need for a basic level Type I, II, III or IV intrusion, but form the basis of the neurological assessment in beyond the basic four types advanced cranial adjusting in dark and fragmented neurological patterns.

Asymmetry in the Ocular Follow Response

The examiner holds their finger 50cm in front of the patient's face and asks them to focus on it (Fig 5). The examiner then carefully observes the width between the medial edge of the iris and the medial epicanthus. Asymmetry is universal in the presence of the cranial subluxation. In addition to initial asymmetry, as the examiner moves their finger slowly towards the patient's face, the eyes will converge at different rates.

Fig 5: Technique of examination for ocular follow.

Intraocular pressure changes

It is virtually universal to see asymmetric intra-ocular pressure when there is a cranial subluxation.

The technique for assessing intra-ocular pressure is shown in Fig 6.

Fig 6: Technique for the palpatory assessment of intra-ocular pressure.

Circular Ocular Tracking

The patient is asked by the examiner to follow the index finger as a circle is traced out in the air (Fig 7). Poor performance on this test and the observation of reproducible circular nystagmus is very common in patients with cranial subluxations.

Fig 7: Technique for the assessment of circular ocular tracking.

Once all the kinesiopathology and neurological tests have been carried out, the examiner is now in a position to determine the following;

- Does the evidence point to a cranial diagnosis?
- Which cranial pattern Type (I, II, III or IV) is in evidence?

The point of intrusion and the phase of respiration on which the intrusion is made will be determined by diagnostic neurological pretesting.

Cranial adjusting protocols

The majority of cranial adjusting in NIP™ is driven by the demonstration of one of the four pattern types and the presence of neurological deficits followed by precise neurological pretesting designed to determine the anatomical point of intrusion and the appropriate phase of respiration.

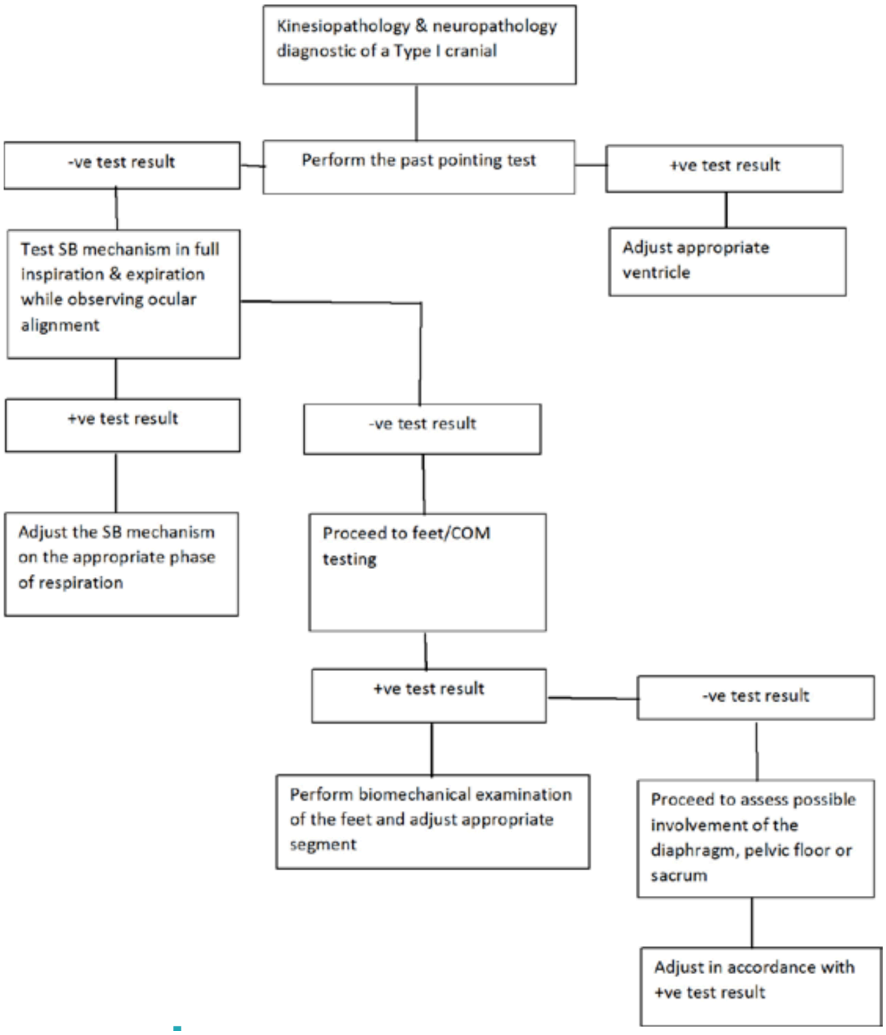
In Type I adjusting, the great majority of intrusions involve flexion and/or extension of the sphenobasilar mechanism the feet or decompression of the fourth ventricle. Less frequently the third and lateral ventricles may be involved. In a few instances, pretesting can only be positively demonstrated by intrusion at the pelvic floor, sacrum or diaphragm.

Type II and III adjusting is much the same as Type I adjusting minus the ventricular decompression techniques with a greater preponderance of positive pretests occurring outside the cranial vault, particularly at the thoracolumbar junction or the (Type II) feet.

Finally, Type IV adjusting is either at the sphenobasilar mechanism or the feet.

The Type I Cranial

Neurological pretesting



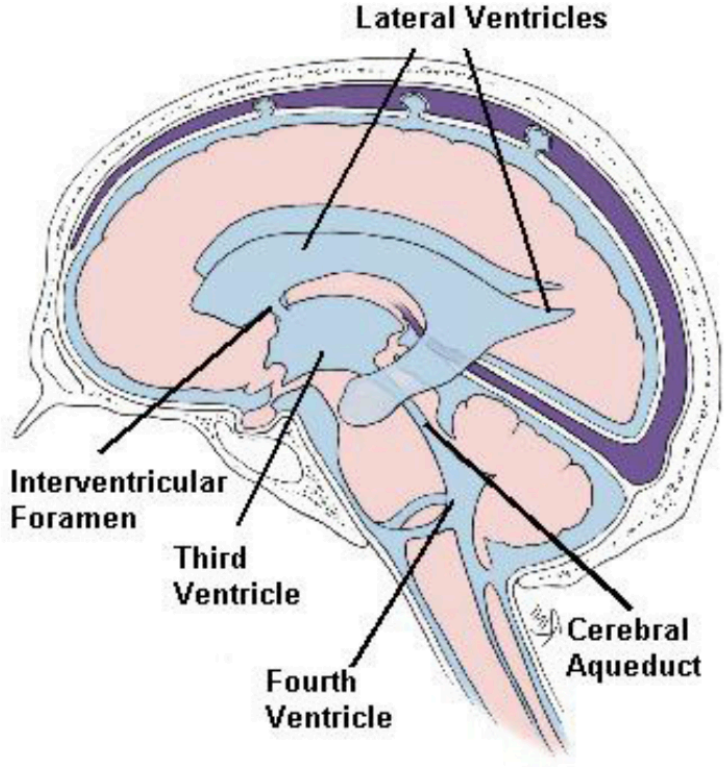
Ventricular Compression

Relevant anatomy & physiology

The fourth ventricle is one of the four interconnected fluid-filled cavities found within the human brain (Fig 8). Collectively, these cavities are known as the 'ventricular system' of the brain.

The fourth ventricle is the most inferiorly located, the other ventricles being the left and right lateral ventricles and the third ventricle. The fourth ventricle extends from the Sylvian aqueduct, otherwise known as the cerebral aqueduct, to the obex. The obex is the marker that lies at the level of the foramen magnum and is therefore theoretically the junction between the caudal end of the medulla oblongata and the beginning of the spinal cord. The fourth ventricle and is filled with CSF.

Fig 8. Anatomy of the ventricles of the brain



The roof of the fourth ventricle is formed by the superior and inferior medullary vela, the floor by the rhomboid fossa which is named because of its shape. It is located on the posterior surface of the pons and medulla oblongata. The side walls of the fourth ventricle are formed by the cerebellar peduncles. CSF enters the fourth ventricle through the Sylvian aqueduct and can exit though the two lateral foramina of Luschka and a single midline foramen of Magendie.

The region of the brain referred to as the posterior fossa is a small area cradled on all sides by bone and limited above by the tentorium cerebelli. The brain stem, cerebellum, and fourth ventricle occupy this region of brain. Expansion of a mass in this infratentorial area occurs at the expense of the normal structures in the region and may result in brain stem or cerebellar dysfunction, often associated with blockage of the fourth ventricle and hydrocephalus.

While the presence of a tumour is a gross example of a space occupying lesion that creates pressure on the affected structures, the work of Shimizu and subsequently that done in the development of NIP™ has demonstrated that mechanical pressure at the upper cervical region and the craniocervical junction is adequate to produce measurable neurological deficit that appears to be related to an increase in the CSF pressure in the ventricles.

The ventricular decompression techniques are based on this observed and testable phenomenon. Given the intimate relationship of the cerebellum and the fourth ventricle, cerebellar testing using the past pointing method is used to identify when a technique designed to decompress the ventricles is indicated. The need to use one of these techniques can be demonstrated neurologically to be actively present or latent, the latter being the more usual case.

Important clinical point

A positive test for IVth ventricle compression may be seen with any of the cranial types. However, it should only be adjusted when it co-exists with a Type I cranial subluxation.

Diagnosis

The ventricular system cranial problem most commonly involves the IVth ventricle, but may also involve the third ventricle. The lateral ventricles will, by extension, be involved to a greater or lesser extent when the third ventricle is involved. They may, however, be involved individually as a unilateral problem.

The presumptive diagnosis of a ventricular compression problem is based on the clear kinesiopathological and neurological evidence for a Type I cranial subluxation. Once this has been established, the definitive test is the past pointing and proprioceptive stressed past pointing tests, The direct past pointing test is represented in Figures 9 and 10. The patient is asked to repeatedly touch their nose and then the examiners finger held at eye level and approximately 30cm (1 foot) away. Once the co-ordinate in space has been visually established by the patient's brain and they are comfortable doing the test, ask them to close their eyes and continue to touch their nose and then the examiners finger. In otherwise normal individuals, the cerebellum should automatically take over the function from the visual axis system and the patient should be able to continue to comfortably perform the test. This is the expected normal outcome and indicates that there is no direct neurodysfunction from the ventricular system.

Important clinical point

The first stage (eyes open) of the past pointing test allows the brain to establish the co-ordinate in space of the examiners finger. During the second phase (eyes closed), the cerebellar system should allow the brain to "see" the point in space previously established and comfortably touch it.

The past pointing test done under proprioceptive stress to expose latency is the second phase of the examination. In this case, to begin with, the patient is asked to do exactly the same thing they did on the direct past pointing test. Once it can be demonstrated that the patient can perform this competently, they are then asked to put their hand down to their side, turn the head fully to one side with their eyes open, close their eyes and return to the midline position where they will be asked once again, with their eyes still closed, to touch their nose and the then the examiner's finger.

Fig 9: Past pointing with yes open. Note how the patient is able to easily identify the point in space at which the examiner is holding their finger.

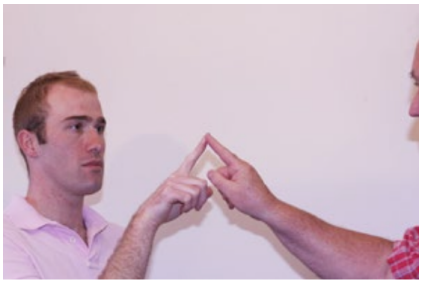


Fig 10: Incompetent past pointing with eyes closed. Note how the patient is unable to identify the point in space at which the examiner is holding their finger after doing so comfortably with their eyes open.

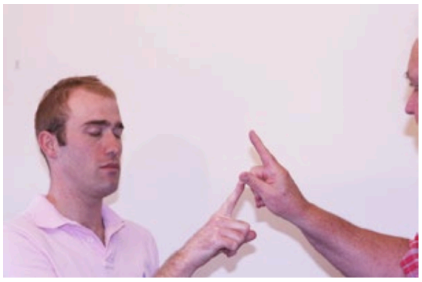
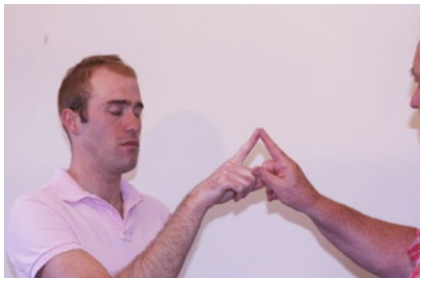


Fig 11: Competent past pointing with eyes closed. Note how the patient is able to identify the point in space at which the examiner is holding their finger after first doing so with their eyes open.



Important clinical point

The diagnostic testing for ventricular compression should always be done in the standing position since in a small number of cases, proprioceptive stress testing involving the upper cervical complex will only cause incompetence in performing past pointing when the clinical problem is in the feet. In these cases, when gravitational stress is removed from the feet, post pointing following proprioceptive stress of the upper cervical complex will be normal.

The same procedure is repeated with the patient turning their head to the other side. The examiner closely observes where the patient's nose comes to rest in relation to the true Z-axis midline position and where they point in relation to the examiners finger. A normal test result, seen illustrated in Figure 11, would be twofold;

- The patients nose will settle in the Z-axis midline
- The patient points to the examiners fingertip with an acceptable error of 5mm (half a finger's width) in any direction at the correct distance, again within 5mm, from their own nose.

The past pointing tests are definitive of the neurological level at which to aim the decompression as follows:

On direct examination

When the patient misses the examiners fingertip ('designated co-ordinate') with eyes closed on direct past pointing examination (Fig 10), it does not matter whether they miss low, level, high or to the side, that result indicates serious compression of the ventricular system and therefore the need to decompress the IVth ventricle.

Important clinical point

Decompression of the IVth ventricle when the patient tests positive to direct past pointing assessment will normally have a significant effect on kinesiopathology and symptoms in the very short term, but equally, it will only be the first in a string of patterns that will usually emerge over the next few days.

On proprioceptive stress testing

The first thing to note is the position of the patient's nose when returning to the midline. They may come right back to the midline and settle in the Z-axis (Fig 12), stop short of the midline on the side to which they turned their head (Fig 13), or go past the midline (Fig 14). In each of these figures the patient first turned their head to their left, then returned.

Fig 12: This patient has returned their nose to the Z-axis midline.

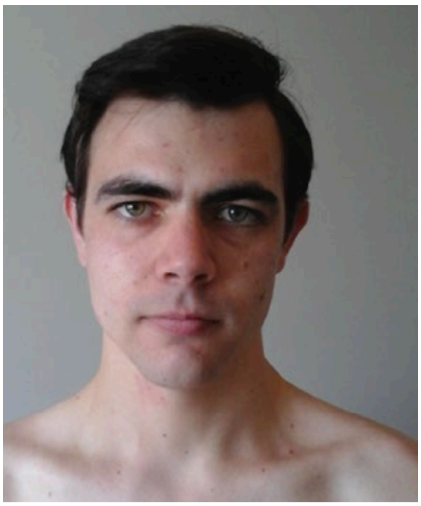


Fig 13: This patient has returned their nose to a position just short of the Z-axis midline.

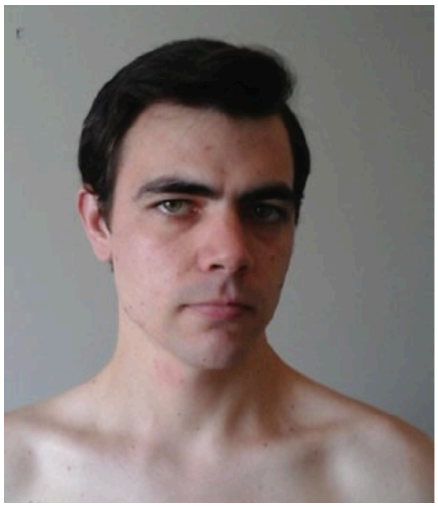
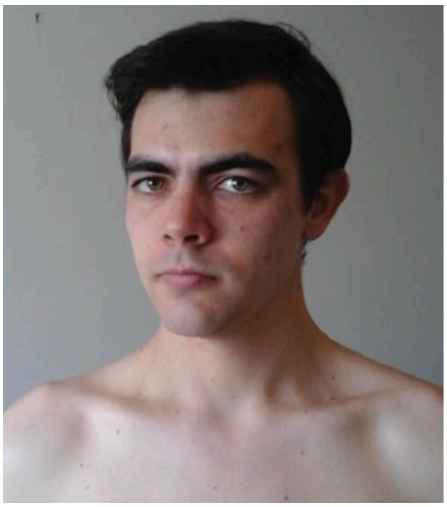


Fig 14: This patient has returned their nose to a position just past the Z-axis midline.



Second, the examiner notes exactly where the patient points in relation to the designated coordinate in space recorded by the brain on successful direct testing. The possible outcomes and their clinical/neurological meaning are shown in Figures 15 - 20.

Fig 15: NORMAL

In this case, the patient's head has returned to the midline and they are pointing directly to the examiners fingertip.

This is considered to be normal physiological function and therefore this represents a negative test result.

Clinically this implies that there is no significant compression in the ventricular system and therefore the reason for the Type I cranial will be found at the sphenobasilar mechanism, in the feet

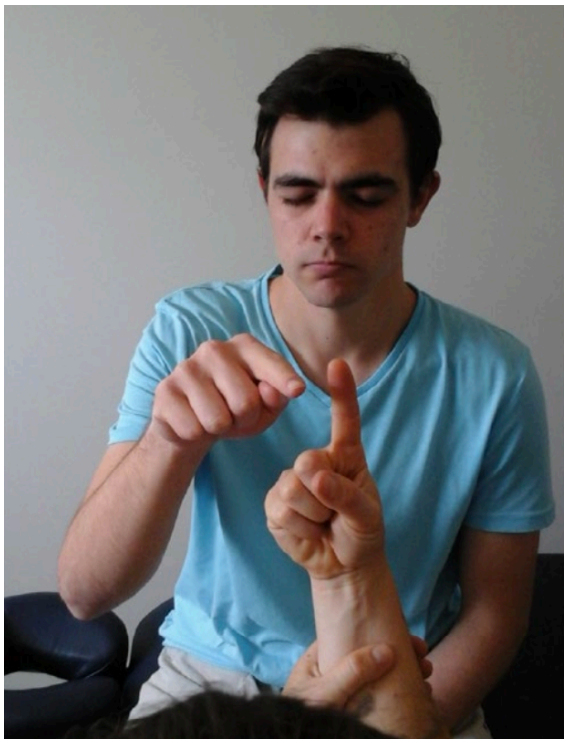


Fig 16: MODERATE 3rd VENTRICLE or SINGLE LATERAL VENTRICLE PROBLEM

In this case, the patient's head has returned to a point beyond the midline and they are pointing:

- laterally to the designated co-ordinate to the same side the nose is pointing
- normal in distance from the patients nose
- at a height level with the designated co-ordinate

Clinically this is the classic moderate third ventricle compression problem when the response is identical to both sides.

In the event this response is unilateral and when tested on the other side the nose returns to the Z-axis midline and the patient can touch the designated co-ordinate, what is implied is a

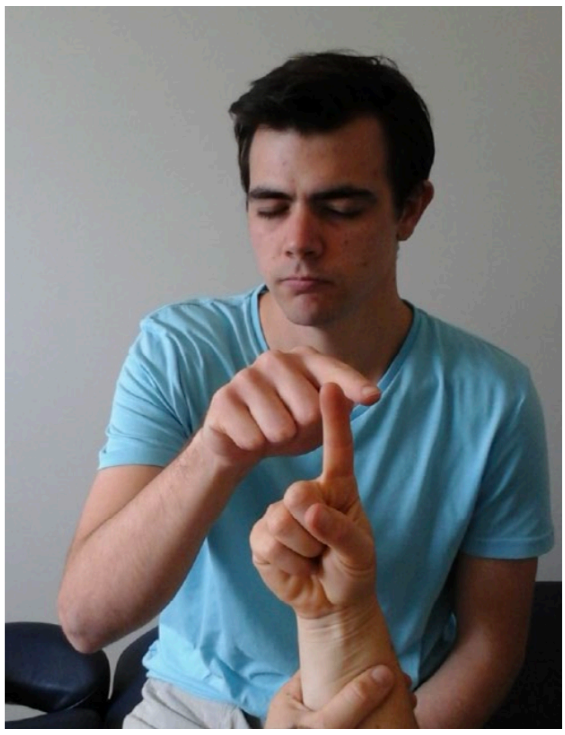


Fig 17: MORE SEVERE 3rd VENTRICLE PROBLEM

In this case, the patient's head has returned to a point beyond the midline and they are pointing:

- laterally to the designated co-ordinate to the opposite side the nose is pointing
- normal in distance from the patients nose or over-reaching
- at a height level with the designated co-ordinate

Clinically this represents a more severe third ventricle compression problem when the response is identical to both sides.

This test result is highly unlikely to be unilateral. The more severe the CSF compression the more likely it is to be exerting effect across the lateral and including the third ventricle, making it a

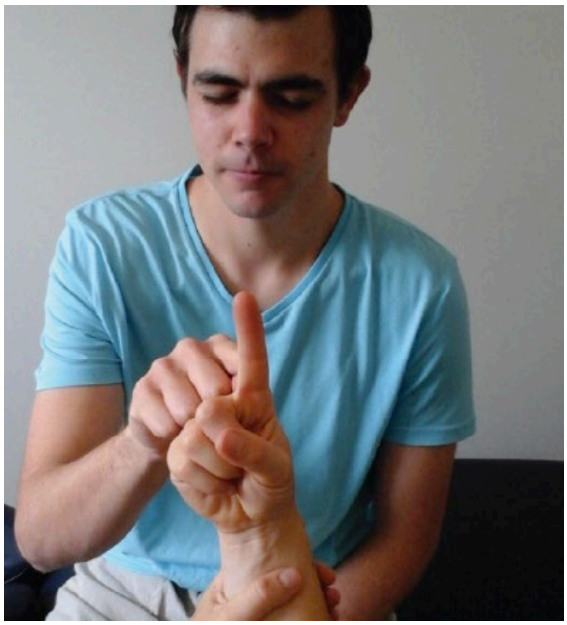


Fig 18: MILD to MODERATE 4th VENTRICLE PROBLEM

In this case, the patient's head has returned to the midline and they are pointing:

- directly toward the examiners finger
- normal in distance from the patients nose
- at a height well below the designated co-ordinate

Clinically this is the classic moderate fourth ventricle compression problem when the response is identical to both sides.

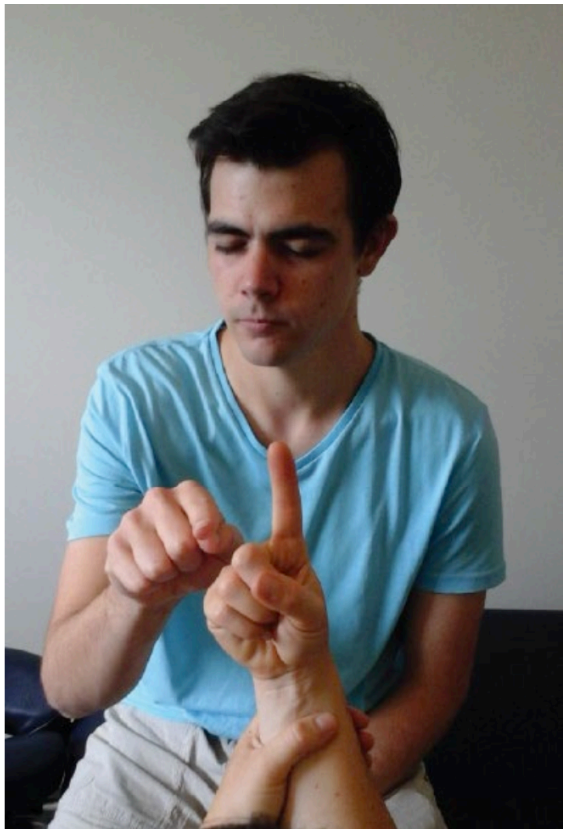


Fig 19: MODERATE 4th VENTRICLE or POSSIBLE FRAGMENTED or SECOND LAYER SINGLE LATERAL VENTRICLE PROBLEM

In this case, the patient's head has returned to a point beyond the midline and they are pointing:

- laterally to the designated co-ordinate to the same side the nose is pointing
- normal in distance from the patients nose
- at a height below the designated co-ordinate

Clinically this is the classic moderate and indeed very common fourth ventricle compression problem when the response is identical to both sides.

In the event this response is unilateral and when tested on the other side the nose returns to the Z-axis midline and the patient can touch the designated co-ordinate, the diagnosis remains a fourth ventricle problem, but the examiner would do well to keep in mind that it might test as a third or even a lateral ventricle immediately following the fourth ventricle decompression as a fragmented or even completely separate pattern.

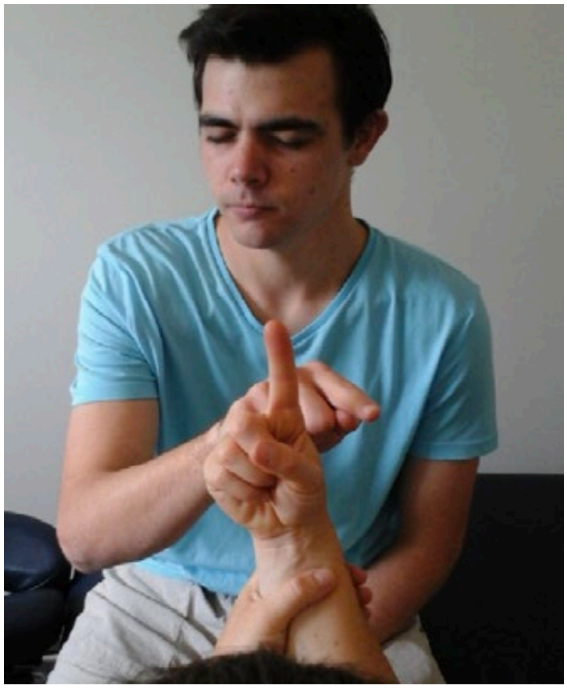


Fig 20: MORE SEVERE 4th VENTRICLE PROBLEM

In this case, the patient's head has returned to a point beyond the midline and they are pointing:

- laterally to the designated co-ordinate to the opposite side the nose is pointing
- normal in distance from the patients nose or over-reaching
- at a height below the designated co-ordinate.

Clinically this represents a more severe fourth ventricle compression problem. This response to testing is universally bilateral.

This test result is highly unlikely to be unilateral. The more severe the CSF compression the more likely it is to be exerting effect across the lateral and including the third ventricle, making it a midline problem.

Test variables to consider

While not the most common of findings, some patients may fall short of the designated target and some may over-reach. This usually occurs alongside a lateral and/or a low miss. It may, however, be a false positive when it appears as a single entity with the patient pointing at or close to the designated co-ordinate and the examiner needs to consider the possibility of performance anxiety on the part of the patient. Repeated attempts and quiet reassurance are the best way to determine if you have a true positive or true negative.

The Third Ventricle

Third ventricle decompression is indicated in a patient who has a Type I cranial and tests positive on past pointing as shown in Figures 16 & 17. Decompression implies that the hydrodynamics of the cerebrospinal fluid is negatively affected and a relative stasis has occurred.

The purpose of all decompressive techniques is to restore normal fluid dynamics in the ventricular system.

The technique utilised to decompress the third ventricle is to use the movement of the adjacent temporal bones. The technique is relatively simple. It involves compressing the lower parts of the temporal bone on inspiration while rocking the bone posteriorly (Fig 21) to assist in external rotation. On expiration, the upper parts of the bone are compressed and rocked anteriorly (Fig 9.22) to assist with internal rotation. The compression on expiration, assisted by the natural diminishing effect on the total cranial volume which occurs concurrently with expiration is the mechanism that decompresses the third and lateral ventricles by pushing the CSF inferiorly.

Important clinical point

The anatomical impact of using the temporal bone is not isolated solely to the hydrodynamics of the third and lateral ventricles. The temporal bone articulates with the zygoma, sphenoid, parietal, occiput in addition to forming the mandibular fossa. It is also a key attachment area for the dura as well as the fascia which extends down into the cervical area. The effect, therefore, of making an intrusion at the temporal area has widespread anatomical and clinical implications.

The Lateral Ventricles

The lateral ventricles are only ever involved unilaterally. In the event both ventricles are in need of decompression, it is a given that the third ventricle is the problem. The technique for decompression of a lateral ventricle is identical to that used for the third ventricle only it is applied to the affected side only as the opposite, non-affected side of the head is gently stabilised using a flat hand contact (Fig 23 & 24).

Fig 23: The lateral ventricle being adjusted during inspiration.

Fig 24: The lateral ventricle being adjusted during expiration.

The Fourth Ventricle

A fourth ventricle decompression is indicated in a patient who tests positive for a Type I cranial and demonstrates incompetent past pointing following proprioceptive stress testing at the upper cervical complex.

Important clinical point

It is only ever appropriate to employ the fourth ventricle decompression technique when stress testing is positive in the context of a Type I cranial.

The application of the forth ventricle decompression technique is performed in two distinct actions, each one being dictated by the respiratory cycle (Fig 25). On inspiration, the

craniocervical junction is taken into hyperextension (Fig 26) and on expiration, counter-pressure is applied simultaneously at the glabella and external occipital protuberance (Fig 27).

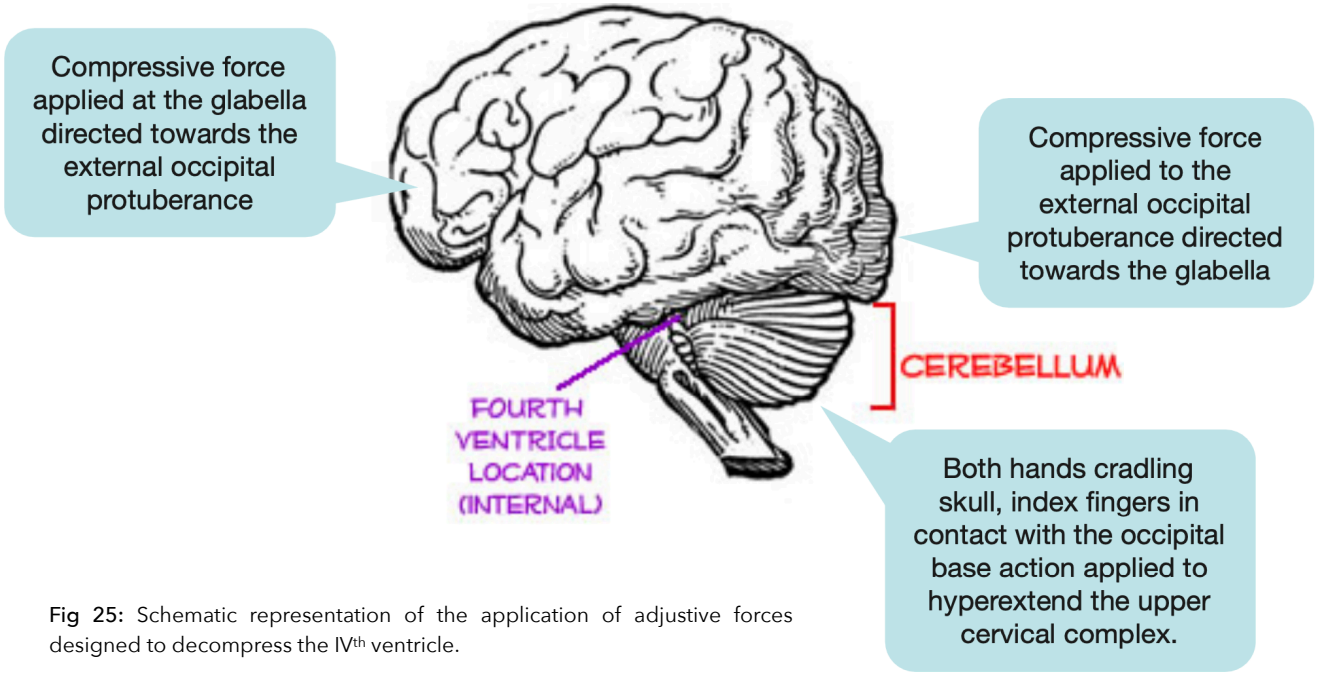
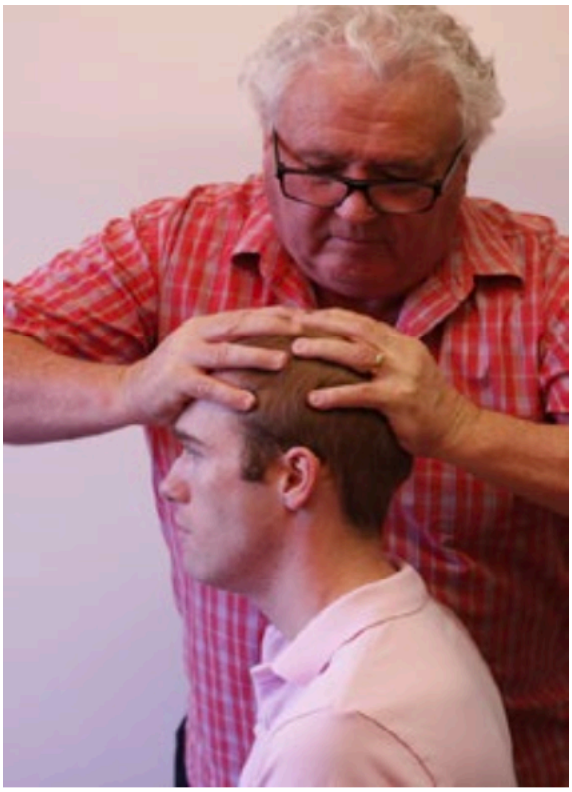


Fig 25: Schematic representation of the application of adjustive forces designed to decompress the IVth ventricle.

Fig 26: During inspiration, the upper cervical complex is taken into hyperextension or negative X-axis rotation. This is achieved by the application of counteracting forces being applied simultaneously to the frontal area and the upper cervical complex.

Fig 27: During expiration, counteracting compressive forces are simultaneously applied to the external occipital protuberance and glabella.



The Sphenobasilar mechanism

Pretesting procedures

The sphenobasilar mechanism (SBM) is a common point of intrusion in cranial adjusting across the four types and can be directly pretested in two separate steps to account for flexion and extension. As the patient breathes to maximal inspiration and holds it the examiner exerts a gentle pressure to the midline of the hard palate in a cephalad direction while testing ocular follow (Fig 28). Normalisation of ocular follow implies that the sphenobasilar mechanism is fixed in extension.

In the event the above action does not change the ocular follow function, the patient breathes to maximal expiration and holds it as the examiner exerts a gentle pressure to the posterior surface of the upper middle incisor teeth in a P-A direction while testing ocular follow (Fig 29). Normalisation of ocular follow implies that the sphenobasilar mechanism is fixed in flexion.

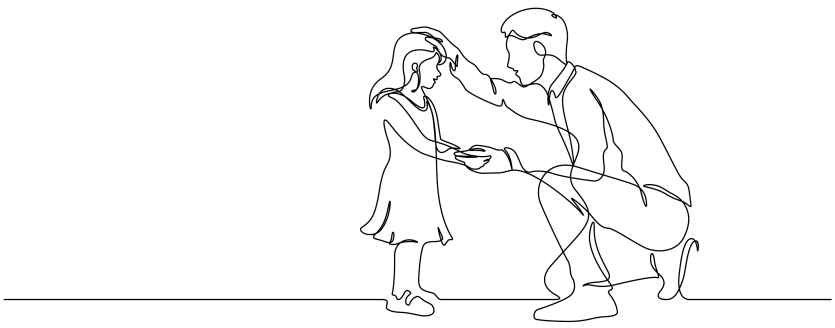
Fig 28: Pre-testing the sphenobasilar mechanism during flexion.

Fig 29: Pre-testing the sphenobasilar mechanism during extension.

Corrective procedures

Correction of the extension fixation is achieved by the maintenance of sustained, constant impulse to the hard palate during the inspiratory phase of respiration as the occiput is simultaneously taken into extension. This action has the effect of raising the SBM into flexion.

Correction of the flexion fixation will be achieved by the maintenance of sustained, constant impulse to the upper incisors in a P-A direction during the expiratory phase of respiration as the occiput is simultaneously taken into flexion. This action has the effect of pulling the SBM into extension.



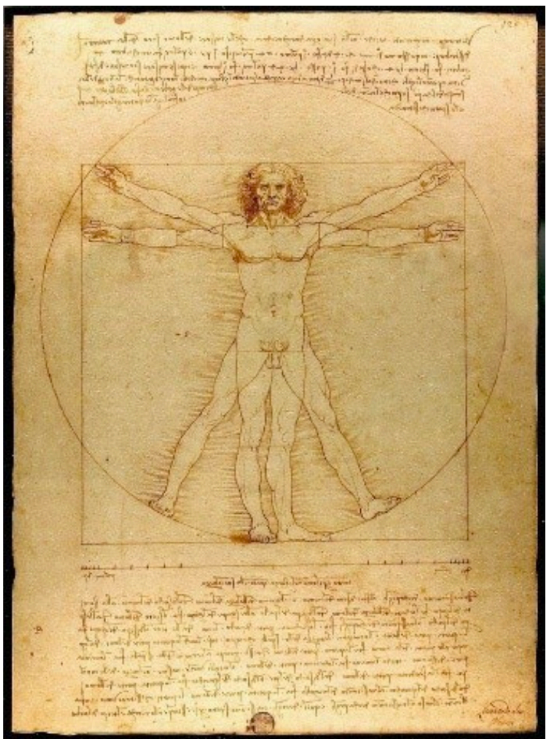
The Feet

Subluxation at the feet plays a major and important role in cranial subluxation and often represents the most efficient place to make the adjustive intrusion. The principal diagnostic reason to be looking to the feet for the point of intrusion is the failure of the type I cranial pattern to pre-test as a ventricular compression syndrome or for any of the four cranial types to pre-test as a sphenobasilar problem.

To screen for foot involvement, one needs to understand the concept of centre of mass (COM) in the human body and the effect dysafferentation has on its location. In the perfect homeostatic state, the centre of mass (COM) is the unique point at the centre of the distribution of the body's mass in space that has the property that the weighted position vectors relative to this point sum to zero. (Boundless 2015) In other words, a point representing the mean position of the body's mass, or 'balance point'. Any movement of the body that changes the location of the COM sets up torque forces (Morasso & Sanguineti 2002) that may negatively influence the reciprocal tension membranes.

In the dysafferentated patient, the reciprocal tension membranes are asymmetrically affected and therefore the COM will be in a relocated position relative to the X and Z axes. This concept of COM has been an important consideration in understanding the human body throughout history. The Vitruvian man drawing by Leonardo da Vinci (Fig 30) demonstrates these observations.

Fig 30 Vitruvian Man: A drawing created by Leonardo da Vinci. The drawing is based on the correlations of ideal human proportions with geometry described by the ancient Roman architect Vitruvius in Book III of his treatise *De Architectura*.



When screening feet for involvement in changes in balance in the reciprocal tension membranes, the patient needs to be standing in an XYZ neutral position in order to appreciate the full effect of gravity through the tarsal arch and hind foot. This places the COM within the pelvis at

approximately the level of the second sacral segment and immediately anterior to it. While maintaining the head in the fixed co-ordinate in space dictated by the XYZ neutral position, the COM location is changed by asking the patient to move their pelvis first positively and then negatively along the Z-axis, followed by positively and then negatively along the X-axis, the effect of which is to change the tension balance in the tarsal arch which in turn changes the torque forces acting on the reciprocal tension membranes. The biophysics of this movement is demonstrated in principle in Figure 31.

The magnitude of torque forces are related in direct proportion to weight and the magnitude of the shift in the COM. (Morasso & Sanguineti 2002) If this change of COM position, with the resultant change in the tarsal arch tension balance brings the torque forces to net zero, the reciprocal tension membranes will react to adopt a more balanced and symmetrical condition, freeing up the bones of the cranium to move more normally with respiration and the neurological elements that were measured in the initial assessment will disappear. This change in the neurological elements should be considered prima faciae evidence of biomechanical foot dysfunction, which will always be bilateral and identical.

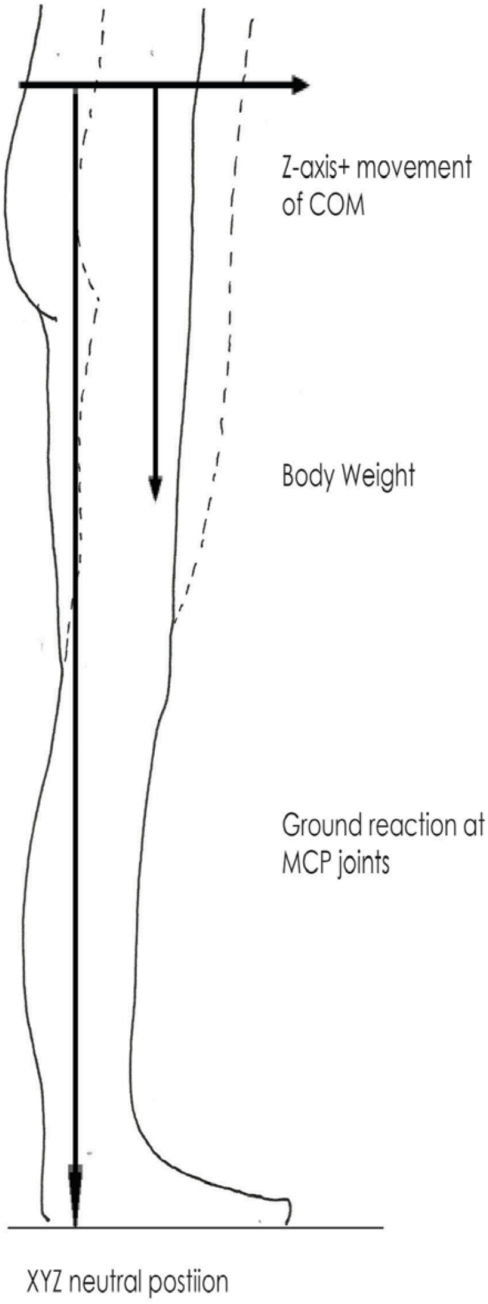


Fig 31: The patient begins in the relaxed XYZ neutral position, then keeping the head and the feet still, the centre of mass (COM) is moved along the Z-axis, translating the weight towards the MCP joints and thus creating a ground reaction as shown above.

The complete examination will involve assessment of the neurological elements of the cranial subluxation after the patient has moved to change the location of the COM positively (Fig 32) and then negatively (Fig 9.33) along the Z-axis, positively along the X-axis (Fig 34) and finally, negatively along the X-axis (Fig 35).

Corrective procedures

Once it has been determined that COM movement with its torque effects on the dura has normalised the cranial kinesiopathology and neuropathology, it is now appropriate to carefully examine the feet beginning with the interosseous articulation then progressing through the hindfoot and midfoot to the forefoot structures as taught in an accompanying Module, # 7. Loss of movement with concomitant weakness of a muscle that crosses the affected joint, identical on both sides of the body will be the bedrock of diagnosis of foot problems causing cranial subluxation. Appropriate intrusive thrust is given, simultaneously to both feet if possible and the cranial signs are then re-evaluated.

Fig 32: Performance of the ocular follow test as the patient moves their COM positively along the Z-axis.

Fig 33: Performance of the ocular follow test as the patient moves their COM negatively along the Z-axis.

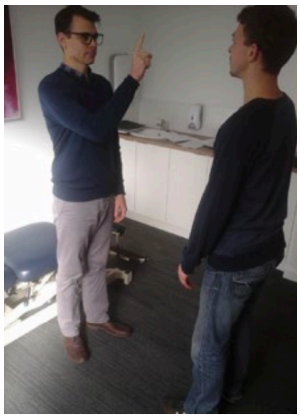


Fig 34: Performance of the ocular follow test as the patient moves their COM positively along the X-axis.

Fig 35: Performance of the ocular follow test as the patient moves their COM negatively along the Z-axis.

The Sacrum

The sacrum is less commonly found to be the key point of intrusion for adjusting the cranial subluxation despite its intimate structural and functional relationship with the occiput and sphenoid. When indicated, this relationship can be made use of for pretesting by applying pressure in such a way as to influence normal movement during respiration and therefore increase or decrease the pull of the dura on the primary respiratory mechanism accordingly.

Pretesting procedures

To pretest the sacrum, the patient lies supine with the hips and knees comfortably flexed (Fig 36). The examiner places the right hand along the posterior surface of the sacrum so the tips of the fingers are in contact with the sacral base and the thenar/hypothenar areas are in contact with the sacral apex and coccyx (Fig 37).

As the patient breathes to maximal inspiration and holds it the examiner exerts a gentle pressure to the sacral apex/coccyx in an anterior direction while testing ocular follow. Normalisation of ocular follow implies that the sphenobasilar mechanism is fixed in extension and correction will be achieved by the application of impulse to the sacrum in that direction during the expiratory phase of respiration.

Holding pressure in this direction over the sacral apex/coccyx will have the effect of assisting the normal movement of the sacral base posterosuperiorly, thus reducing the pressure on the dural membrane which in turn will allow the sphenobasilar mechanism to move out of extension into flexion.

Second, as the patient breathes to maximal expiration and holds it as the examiner exerts a gentle pressure to the sacral base in an anterior/inferior direction while testing ocular follow. Normalisation of ocular follow implies that the sphenobasilar mechanism is fixed in flexion and correction will be achieved by the application of impulse to the sacrum in that direction during the inspiratory phase of respiration. Holding pressure in this direction over the sacral base will have the effect of assisting the normal movement of the sacral base anteroinferiorly, thus increasing the pressure on the dural membrane which in turn will draw the sphenobasilar mechanism out of flexion and in to extension.

Corrective procedures

For an extension fixation of the SBM, the patient breathes to maximal inspiration and holds it as gentle pressure is applied to the sacral apex in an anterior direction.

For a flexion fixation of the SBM, the patient breathes to maximal expiration and holds it as gentle pressure is applied to the sacral base in an anterior/inferior direction.

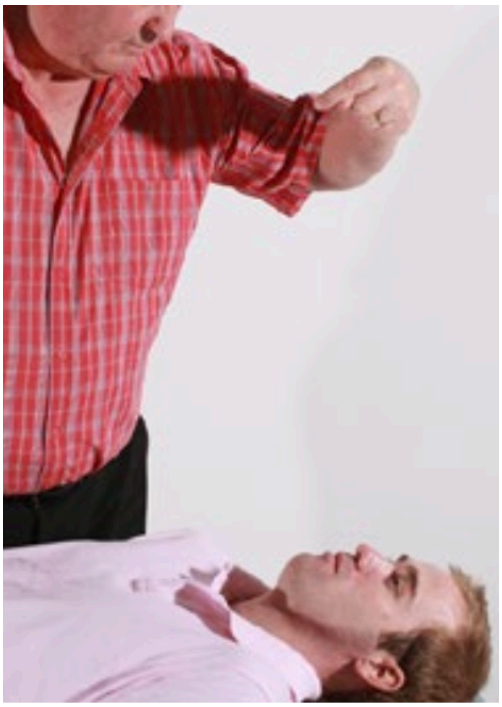


Fig 36 (left): Patient position for sacral pre-testing.
Fig 37 (below): Doctor's contact for sacral pre-testing.

Important clinical point

The purpose of applying respiratory phase related pressure to the sacrum is to change the tension in the dural membrane in a way which optimises normal function in the sphenobasilar mechanism.

The Diaphragm

The function of the diaphragm is intimately linked to the pelvic structures and through them via the dural membrane to the upper cervical complex and cranial vault. Dysfunction of the diaphragm can therefore be reflected in the function of the primary respiratory mechanism.

Pretesting procedures

Diaphragmatic pretesting is performed with the patient supine. Contact is taken by the examiner under the costal arch in its medial one-third and cephalad pressure is applied, first during inspiration and secondly during expiration.

As the pressure is applied at the peak of each respiratory cycle, ocular follow is assessed (Fig 38).

Corrective procedures

Normal adjustive impulse should be applied at the peak of the phase of respiration in which ocular follow was shown to normalise during pretesting.

Fig 38: Technique for pre-testing the diaphragm.

Important clinical point

While the majority of diaphragm related cranial faults are found to be due to dysfunction at the medial one-third of the diaphragm, occasionally pretesting will be positive in the middle one-third and rarely in the lateral one-third.

The Pelvic Floor

The function of the pelvic floor, also known as the pelvic diaphragm is directly linked to the pelvic structures through the muscular attachments. Dysfunction in the pelvic floor can therefore be reflected in the function of the primary respiratory mechanism.

Pretesting procedures

Pelvic floor pretesting is accomplished by asking the seated patient to take a full inspiration and then bear down gently in the same manner as one would do during defecation. The neurological and kinesiological deficits that were previously demonstrated will normalise, indicating that the intrusion should be made on inspiration.

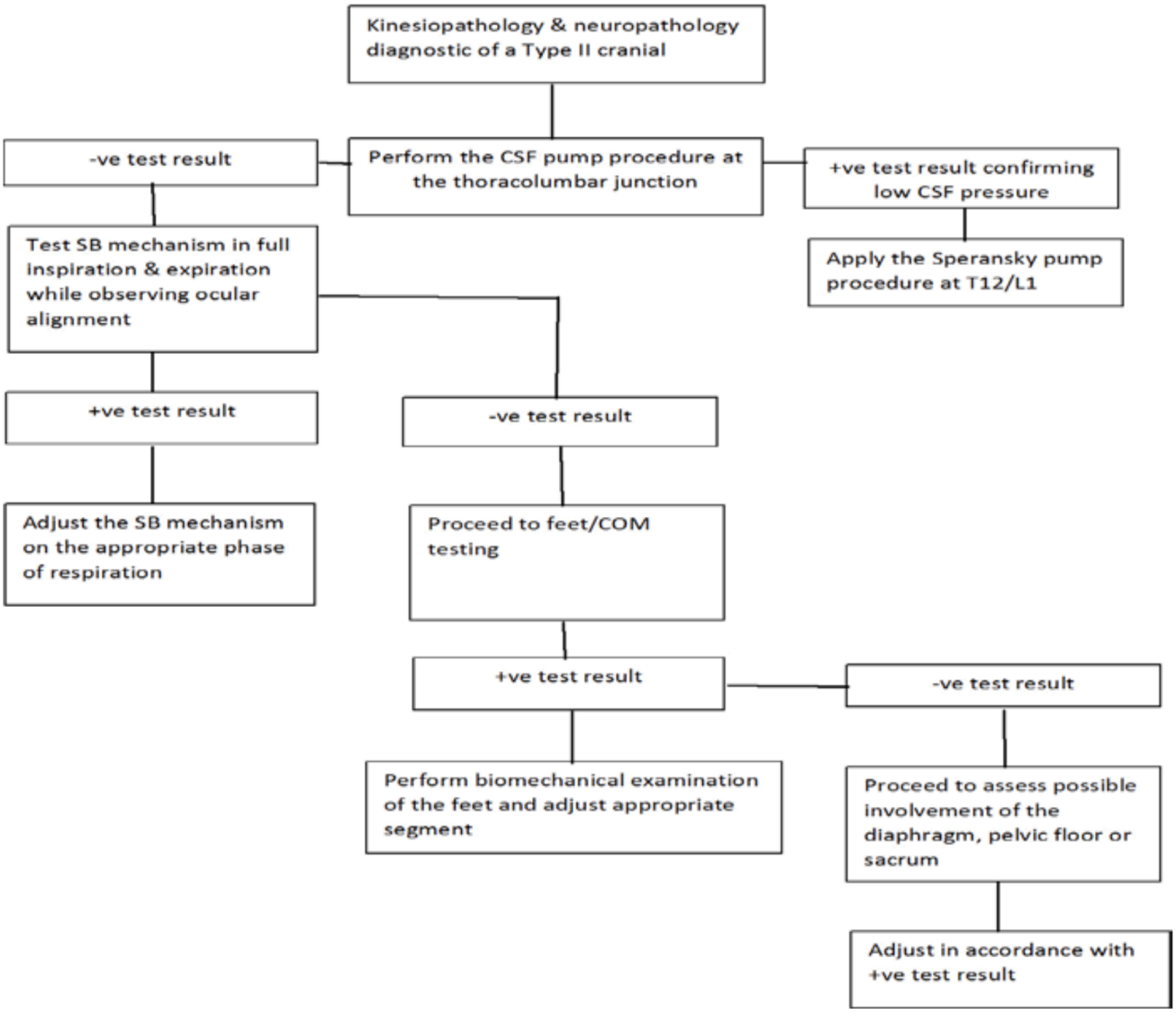
The same procedure is repeated during a full expiration.

In the event the problem at the pelvic floor is latent, testing using the S-reflex is required (see related NIP™ manual 10 p39). When this is the case, performance of the S-reflex test will normalise the cranial findings.

It is, of course, necessary to perform the S-reflex test in both full inspiration and full expiration.

Neurological pretesting

The following order of examination will apply when kinesiopathology and neuropathology identifies a Type II cranial subluxation:



In his text '*A Basis for the Theory of Medicine*' the Russian physician/medical researcher AD Speransky (1943) describes a procedure that was central to his theory where he injected normal saline into the filum terminale to increase the total CSF fluid volume and therefore the upward pressure on inspiration and then removed it again, effectively creating a pumping mechanism.

In NIP™, that experimental strategy is emulated by the application of physical pressure on inspiration which is released on expiration thus emulating the Speransky experiment.

The pumping action is effected by contacting the thoracolumbar junction and applying a force directed from P-A and I-S as the patient breathes in. The pressure is released on expiration. The purpose of this procedure is to increase the CSF pressure in the ventricles. This pumping action is used as a neurological pretest and also as a corrective procedure. In the Type II cranial, where the CSF pressure in the ventricles is low due to impaired sacral mechanics, the neurological pretest is carried out by applying the pumping action 2-3 times, then immediately reassessing the diagnostic elements of the Type II cranial which will disappear if low CSF pressure is the problem.

Corrective procedures

Contact is taken over the *coccygeus* muscles bilaterally. The *coccygeus* is a triangular plane of muscular and tendinous fibres, which arises from both the spine of the ischium and the sacrospinous ligament. It inserts into the lateral margin of the coccyx and the fifth sacral segment, making it the most readily accessible pelvic floor muscle.

The intrusion is made by applying pressure in a medialward and cephalad direction during the appropriate phase of respiration for 10-15 seconds. The usual NIP™ thrust is made to complete the correction. The patient is then reassessed.

Adjusting individual sutures

On rare occasions, a patient will test positive for cranial subluxation with the usual full complement of kinesiopathological deficits, neurological deficits that may be unilateral or bilateral but which do not respond to dural membrane pretesting. When this is the case, individual sutures need to be assessed for separation or congestion. Sutural subluxations tend to be point specific within a suture and are usually due to separation strain made worse on inspiration and better on expiration.

Conversely, when the suture is congested, it is made worse on expiration and better on inspiration.

Pretesting procedures

When there is a specific sutural subluxation in a midline suture such as the sagittal suture, the effect neurologically will be midline in much the same way as a dural tension subluxation is midline. The assessment protocol involves examining each midline suture at intervals approximately one fingers width apart. The suture is first separated on inspiration as ocular follow is tested (Fig 9.39) and then closed on expiration as ocular follow is tested (Fig 40).

When there is a specific sutural subluxation in a lateral suture such as the coronal suture, the effect neurologically will usually be seen in the Shimizu reflex on the opposite side of the body. The assessment protocol involves examining each suture at intervals approximately one fingers width apart. The suture is first separated on inspiration as the contralateral Shimizu reflex is tested (Fig 41) and then closed on expiration again as the contralateral Shimizu reflex is tested (Fig 42).

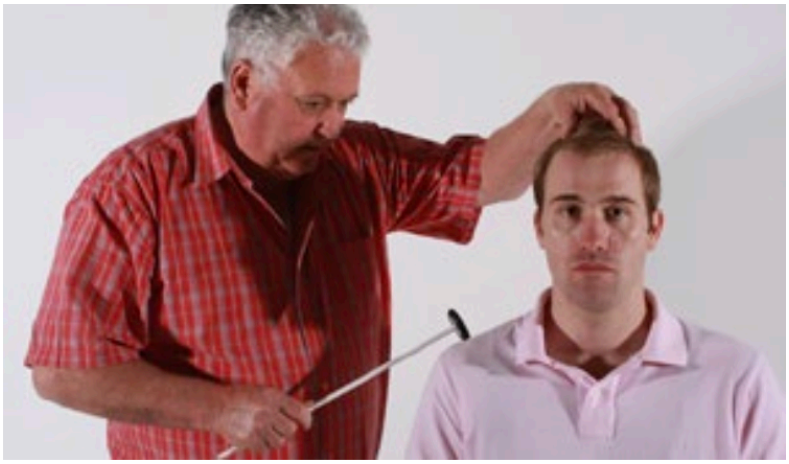


Fig 39: Pre-testing the sagittal suture on inspiration.

The examiner separates the suture at peak inspiration as ocular follow is assessed.

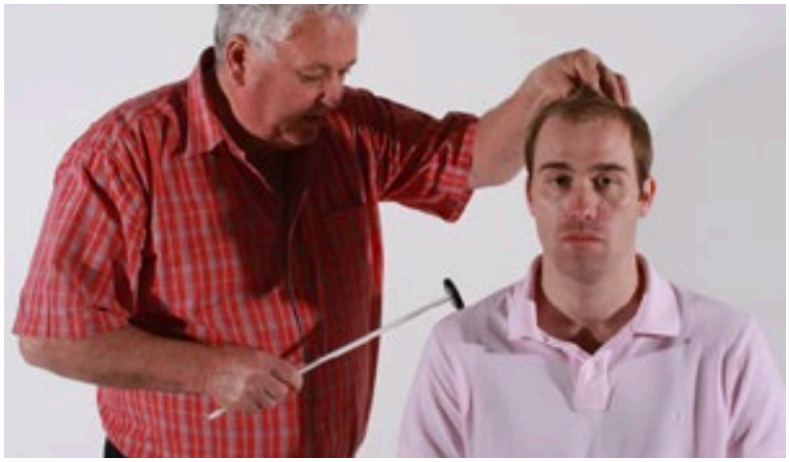


Fig 40: Pre-testing the sagittal suture on expiration.

The examiner closes the suture at peak expiration as ocular follow is assessed.

Fig 41: Pre-testing the coronal suture on inspiration.

The examiner separates the suture at peak inspiration as the contralateral Shimizu reflex is assessed.

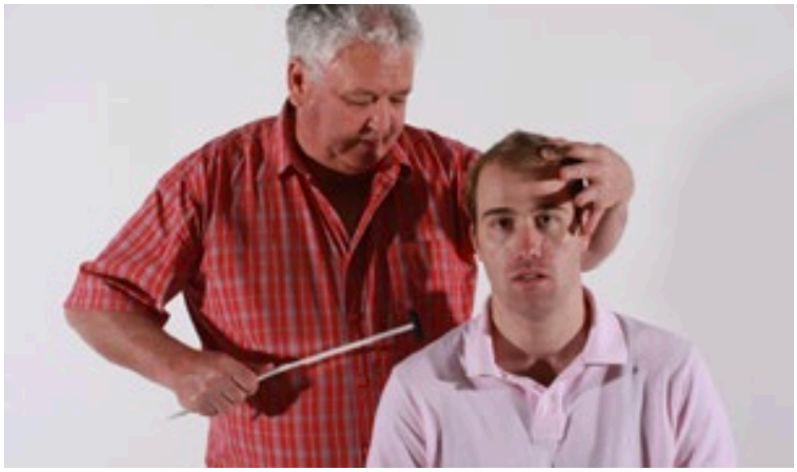


Fig 42: Pre-testing the coronal suture on expiration.

The examiner closes the suture at peak expiration as the contralateral Shimizu reflex is assessed.

Corrective procedures

The direction which changes ocular follow or Shimizu reflex positively is determined by the pretesting procedure previously described. The pretest will determine the direction of intrusion which is then sustained as a constant gentle pressure for several respiratory cycles. Following correction, the suture is retested. Ocular follow will normalise and the Shimizu reflex will disappear.

The 'Dropped' Sphenoid

The dropped sphenoid is a unique cranial problem in that it is both very common and exists outside of the four cranial patterns described previously. Neurologically, its only association is with a positive Shimizu and a weak upper trapezius, both contra-laterally.

Diagnostic criteria

There will be a contralateral loss of lateral flexion at the upper cervical complex with decreased glenohumeral abduction and a positive Shimizu reflex. Gross rotation is preserved and the pectoral reflex is normal. The contralateral upper trapezius will be weak.

Pretesting procedures

The pretest is simple. The greater wing of the sphenoid is gently lifted while the Shimizu reflex is tested on the opposite side of the body. In the event the sphenoid is dropped, lifting it will eliminate the Shimizu reflex (Fig 9.43) and normalize both shoulder abduction and upper cervical lateral flexion.

It is common for the dropped sphenoid to present as a latent finding, meaning the magnitude

of the neurological deficit is inadequate to create a Shimizu reflex. When the other diagnostic criteria suggest a dropped sphenoid, but the Shimizu reflex is absent, it is appropriate to depress the greater wing of the sphenoid and check the Shimizu again. Whether the dropped sphenoid is directly measurable or latent, the corrective procedure remains the same.

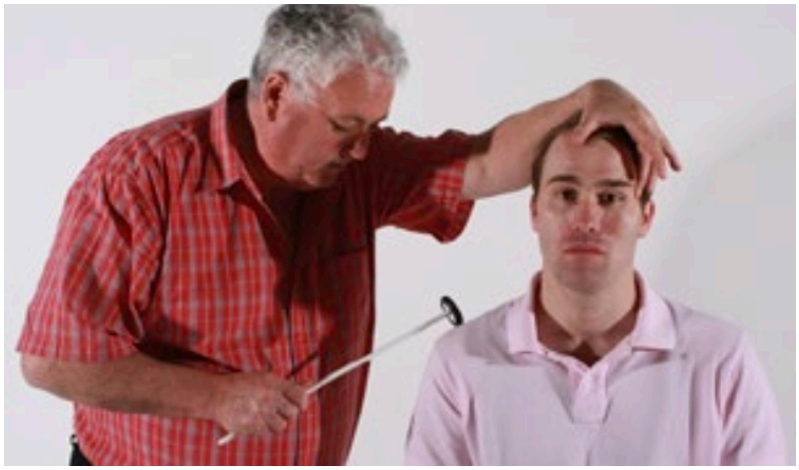


Fig 43: Pre-testing the dropped sphenoid.

The greater wing of the sphenoid is gently distracted superiorly on full inspiration as the contralateral Shimizu reflex is assessed.

Corrective procedures

The corrective procedure is simple. The greater wing of the sphenoid on the affected side is lifted and held during several cycles of inspiration. The Shimizu reflex is then retested and will be normal if the correction has been successful.

Important clinical point

While not common, on occasion a bone other than the sphenoid will drop. These most commonly include the frontal, temporal, parietal and occiput, but may also include, albeit less commonly, the facial bones. When the sphenoid fails to positively pretest, each of these bones should be lifted and the Shimizu reflex tested. If an individual bone will not pretest, combinations of these bones need to be tested.

The Pseudocranial muscle compartment syndromes

Muscles which have attachment to the cranium and mandible, or exert influence over the upper cervical biomechanics are capable, when dysfunctional (i.e. muscle compartment syndrome), of creating kinesiopathological and neuropathological findings, often very confusing or conflicting, which mimic upper cervical and/or cranial subluxation. It is often initially thought the patient may be switched until this is demonstrated not to be the case.

The critical points of understanding are these:

- In relation to the Type I cranial, there will always be kinesiopathology and neuropathology that looks quite solid and stock standard until a pretest is attempted. When the pretest fails, a muscle compartment syndrome may be the cause of it all. Applying impulse to the muscle according to its state of tonicity will alleviate the neuropathology despite the fact it is bilateral and the muscle compartment syndrome is almost always unilateral.
- In relation to the upper cervical subluxation, the kinesiopathology will suggest a particular problem and the neuropathology may concur, it may be on the opposite side to that expected leading to a conclusion the patient is switched or it may be bilateral indicating an overlying cranial. The point is, none of these will pretest positively and this fact leads you to consider a muscle compartment syndrome.

The affected muscle will usually demonstrate weakness on direct challenge and if not, will definitely demonstrate weakness when stressed by inspiration and expiration. Application of impulse in accordance with the tonic state of the muscle will result in a positive outcome, indicating the muscle compartment syndrome to be the cause of the problem.

It is critical to understand that these confusing findings are very uncommon and do not follow the normal rules of unilaterality, bilaterality and pretest. The following discussion identifies these muscles and the effects of their dysfunctional action.

The Levator Anguli Scapulae Syndrome

Origin

The posterior tubercles of the transverse processes, typically of C1-C4 but at times also C5.

Insertion

The superior angle of the scapula as far inferior along its medial margin as the insertion of the *rhomboid minor*.

Action

It both lifts the scapula and laterally flexes and hyperextends the cervical spine.

Clinical significance

When the muscle is hypertonic, its action creates the impression of a contralateral AS occiput and its effect on the upper cervical cord will give neuropathology consistent with a cranial subluxation complex. This would include Shimizu reflex, hyperactive pectoral reflex and cranial specific findings such as increased intraocular pressure and asymmetric ocular follow. When the muscle is hypotonic, the action of its antagonists gives the impression of an ipsilateral PS occiput.

Pretesting procedures

When the muscle is hypertonic, pretesting is performed by asking the patient to depress their shoulder and flex their head in such a way that the origin and insertion of the muscle are separated (Fig 44). When the muscle is hypotonic, pretesting is performed by asking the patient to shrug their shoulder and extend their head in such a way that the origin and insertion of the muscle are approximated (Fig 45).

A positive pretest is seen as previous ocular follow asymmetry will normalising into parallel action, confirming the diagnosis. It is important to perform the ocular follow pre-test not in the Z-axis of the body, but in a line at right angles to a line taken through the pupils in the cranial midline as seen in Figs 44 and 45.

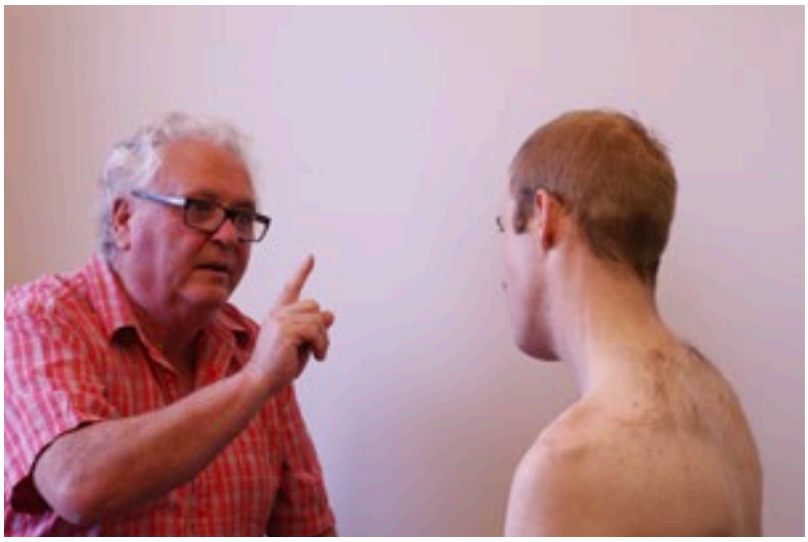


Fig 44: The hypertonic muscle is pre-tested in the stretch position.

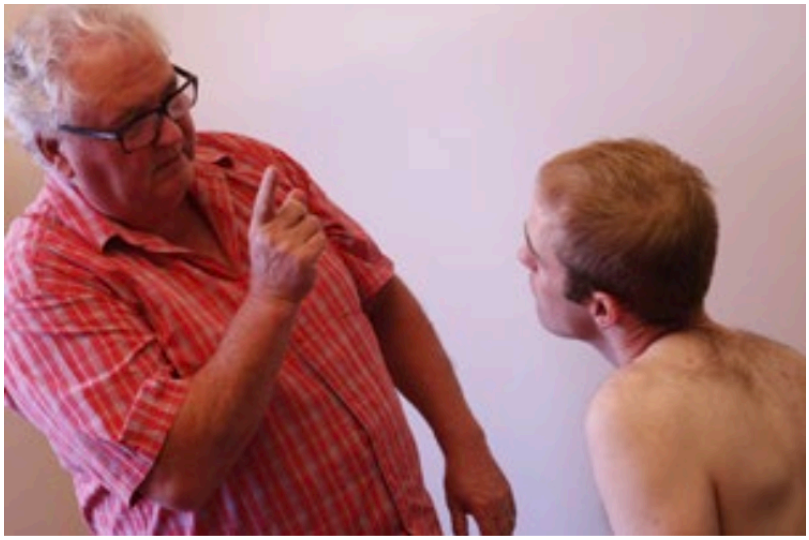


Fig 45: The hypotonic muscle is pre-tested in the shortened position.

Treatment Protocol

When the muscle is hypertonic

With the patient prone, take contact over both the origin and insertion of the muscle. Impulse is initiated by applying pressure in opposite directions to stretch the muscle and a typical NIP™ thrust is made after 10-15 seconds (Fig 46). As a modification, the thoracic drop piece may be utilised in larger, endomorphic patients.

When the muscle is hypotonic

With the patient prone, take contact over both the origin and insertion of the muscle. Impulse is initiated by applying pressure towards the mid-belly from the origin and insertion. A typical NIP™ thrust is made after 10-15 seconds (Fig 9.47). As a modification, the thoracic drop piece may be utilised in larger, endomorphic patients.

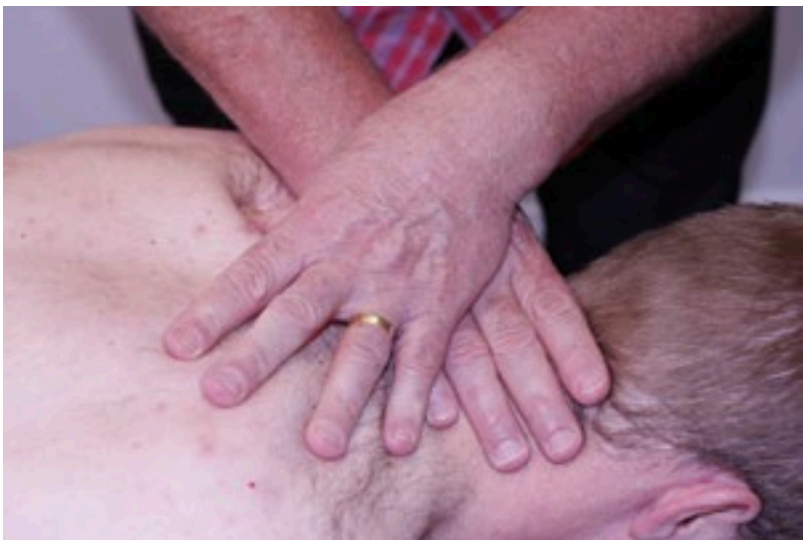


Fig 46: Adjustive intrusion is applied to the hypertonic muscle in a manner which separates the origin and insertion.

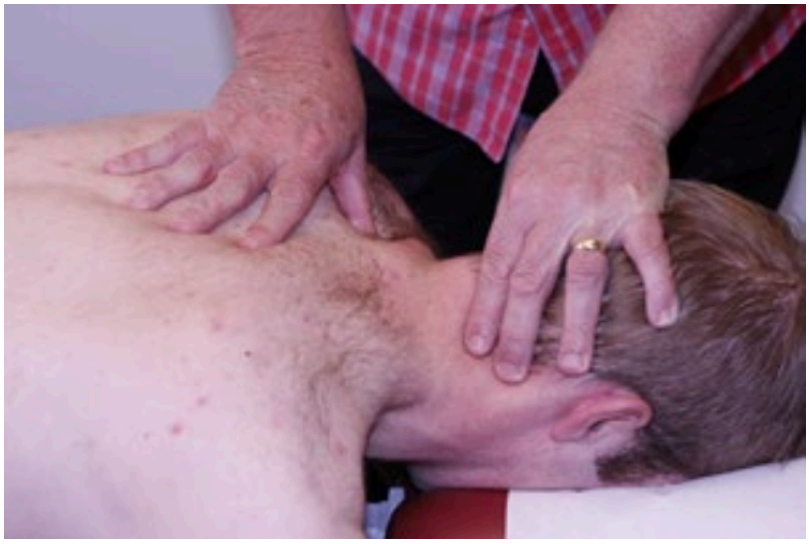


Fig 47: Adjustive intrusion is applied to the hypotonic muscle in a manner which approximates the origin insertion.

The Upper Trapezius Syndrome

Origin

Medial superior nuchal line, external occipital protuberance and ligamentum flavum.

Insertion

Lateral clavicle, acromion and spine of scapula.

Action

Elevates the scapula and extends the head/neck.

Clinical significance

When the muscle is hypertonic, its action, in pulling the head and neck into extension creates the impression of an ipsilateral or even bilateral AS occiput and its effect on the upper cervical cord will give neuropathology consistent with a cranial subluxation complex. This would include Shimizu reflex, hyperactive pectoral reflex and cranial specific findings such as increased intraocular pressure and asymmetric ocular follow. When the muscle is hypotonic, the action of its antagonists gives the impression of an ipsilateral PS occiput.

Pretesting procedures

Pretesting the hypertonic muscle is performed by asking the patient to depress their shoulder and flex their head in such a way that the origin and insertion of the muscle are separated (Fig 48). Pretesting the hypotonic muscle is performed by asking the patient to shrug their shoulder and extend their head in such a way that the origin and insertion of the muscle are approximated (Fig 49). In both cases, previous ocular follow asymmetry will normalise into parallel action, confirming the diagnosis. It is important to perform the ocular follow pre-test not in the Z-axis of the body, but in a line at right angles to a line taken through the pupils in the cranial midline.

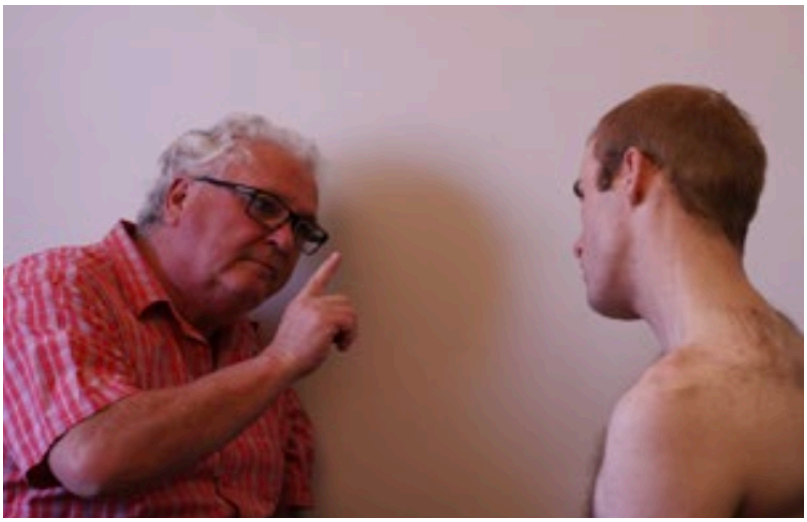


Fig 48: Pretesting the hypertonic muscle.

Fig 49: Pretesting the hypotonic muscle.

Treatment protocol

When the muscle is hypertonic

With the patient prone, take contact over both the medial aspect of the upper nuchal line and just superior to the spine of the scapula, those landmarks being the origin and insertion of the upper trapezius fibres. Impulse is initiated by applying pressure in opposite directions to stretch the muscle and a typical NIP™ thrust is made after 10-15 seconds (Fig 50). As a modification, the thoracic drop piece may be utilised in larger, endomorphic patients.

When the muscle is hypotonic

With the patient prone, take the same contact described for the hypertonic muscle. Impulse is initiated by applying pressure from each end of the muscle towards the mid-belly in order to shorten the muscle and a typical NIP™ thrust is made after 10-15 seconds (Fig 9.51). As a modification, the thoracic drop piece may be utilised in larger, endomorphic patients.

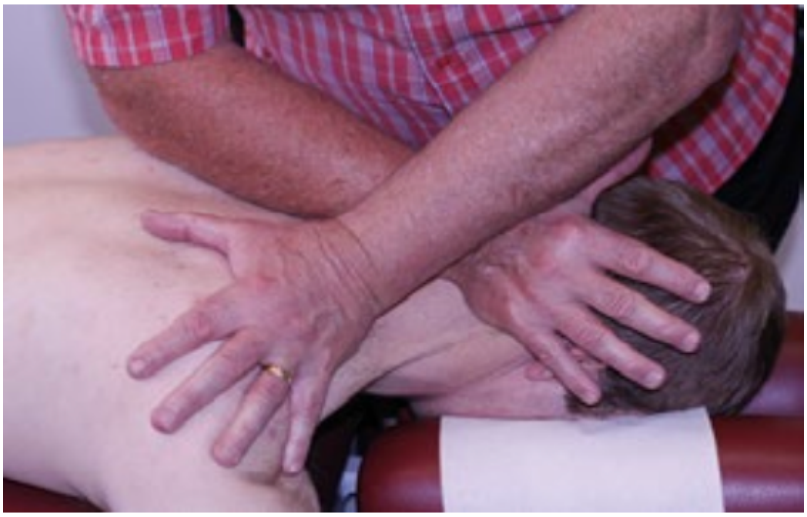


Fig 50: Generation of impulse when the muscle is hypertonic.

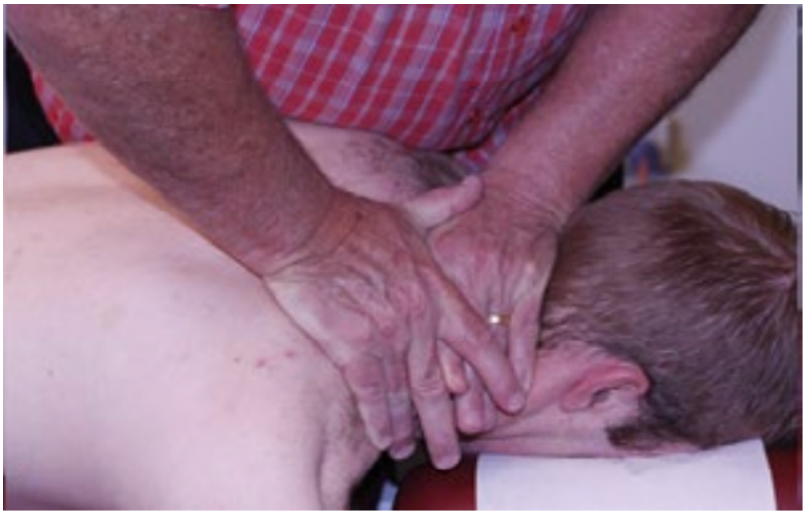


Fig 51: Generation of impulse when the muscle is hypotonic.

The SCM Syndrome

Origin

The medial or sternal head arises from the upper part of the anterior surface of the manubrium sterni and is directed superiorly, laterally, and posteriorly. The lateral or clavicular head, arises from the superior border and anterior surface of the medial third of the clavicle and is directed almost vertically upward.

Insertion

The lateral surface of the mastoid process, from its apex to its superior border, and by a thin aponeurosis into the lateral half of the superior or upper nuchal line of the occiput.

Action

The function of this muscle is to rotate the head contralaterally and in doing so it elevates the chin on that side. It also produces neck flexion ipsilaterally.

Clinical significance

The action of this muscle tends to produce a PS occiput contralaterally and its effect on the upper cervical cord will give neuropathology consistent with a cranial subluxation complex. This would include Shimizu reflex, hyperactive pectoral reflex and cranial specific findings such as increased intraocular pressure and asymmetric ocular follow.

Pretesting procedures

When the muscle is hypertonic, pretesting will be positive when the patient rotates their head to the contralateral side while at the same time hyperextending the cervical spine in order to maximally stretch the muscle (Fig 52). Previous ocular follow asymmetry will normalise into parallel action, confirming the diagnosis. When the muscle is hypotonic, pretesting will be positive when the patient rotates their head to the contralateral side while flexing their head to the ipsilateral side in such a way that the origin and insertion of the muscle are approximated (Fig 53). Previous ocular follow asymmetry will normalise into parallel action, confirming the diagnosis.

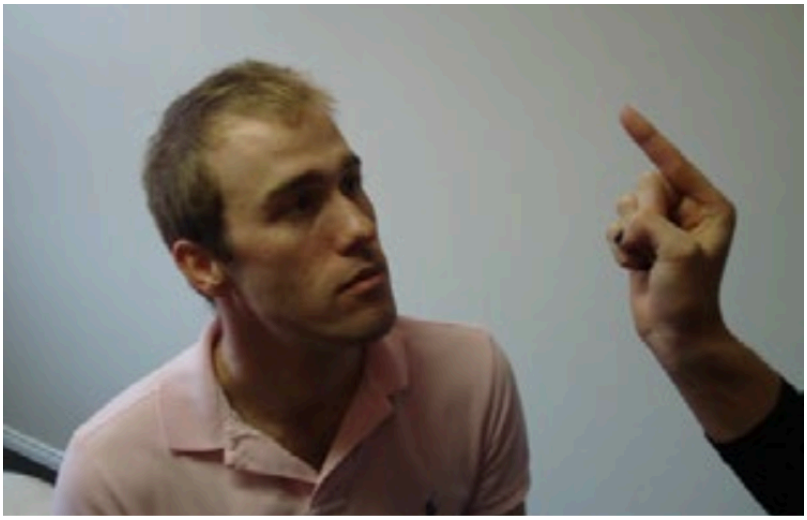


Fig 52: Pretesting the hypertonic muscle.

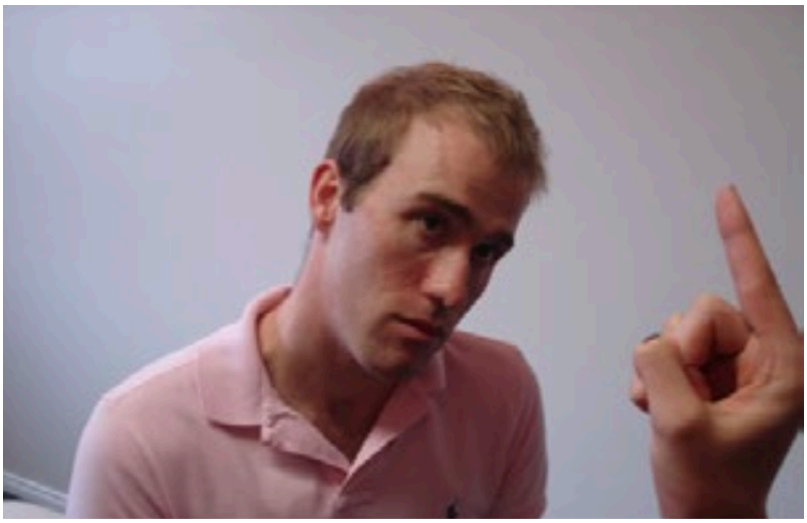


Fig 53: Pretesting the hypotonic muscle.

Treatment protocol

When the muscle is hypertonic

With the patient supine, take a thumb contact over both the medial aspect of the clavicle (origin) and the lateral aspect of the mastoid and upper nuchal line (insertion). Impulse is initiated by applying pressure in opposite directions to stretch the muscle and a typical NIP™ thrust is made after 10-15 seconds (Figure 54). It is never necessary to utilise the drop piece when adjusting the SCM.

In the event there is a trigger point in the mid-belly of the muscle, it is appropriate to pre-test the muscle belly by applying pressure from P-A and then A-P. If this area of the muscle pre-tests for ocular follow, the treatment protocol is simply to hold pressure constant for 10-15 seconds followed by the usual NIP™ thrust.

When the muscle is hypotonic

With the patient supine, take a thumb contact over the origin and insertion of the muscle. Ask the patient to turn their head about 30˚ away from the side of contact. Impulse is initiated by applying pressure simultaneously from both ends of the muscle towards the mid-belly and a typical NIP™ thrust is made after 10-15 seconds (Figure 55). It is never necessary to utilise the drop piece when adjusting the SCM.

The hypotonic muscle will not be associated with a trigger point in the mid-belly of the muscle.

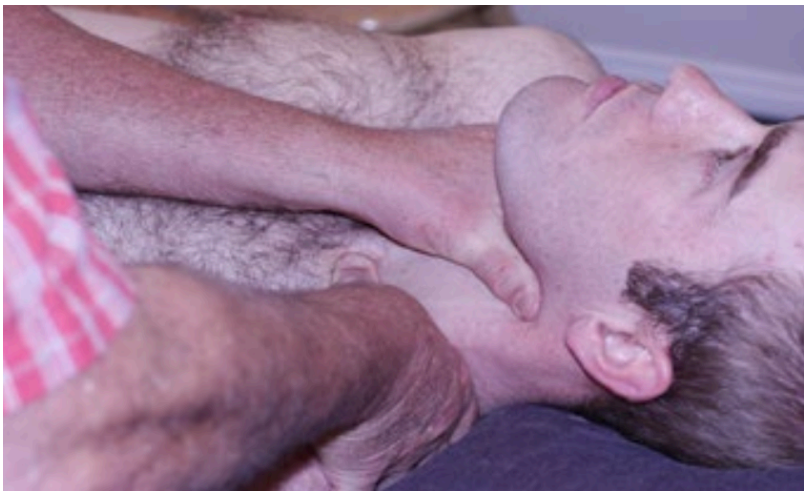


Fig 54: Impulse generation when the muscle is hypertonic.

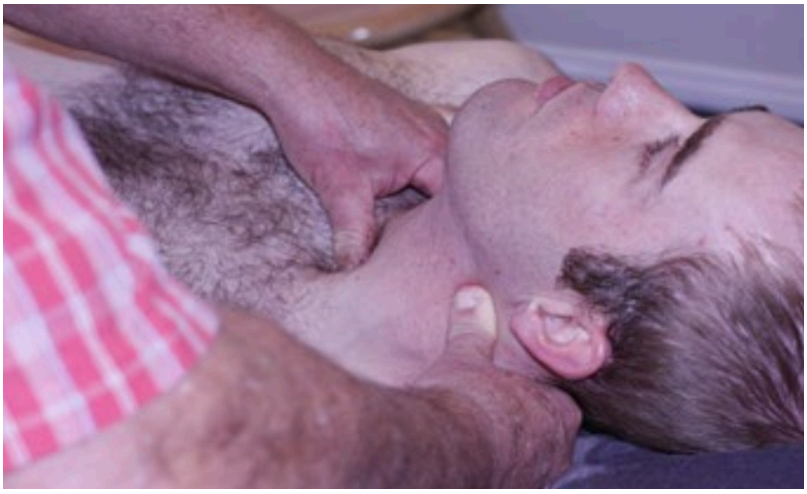


Fig 55: Impulse generation when the muscle is hypotonic.

The Scalene Syndrome

The scalene group is made up of the *scalenus anticus*, *scalenus medius* and *scalenus posticus*. In addition, there is a small sliver of muscle named the *scalenus minimus* which arises from the seventh cervical transverse process and inserts into the inner border of the first rib as well as the fascia supporting the dome of the pleura.

The scalene muscles may exert influence on the osseous structures acting as individual muscles or acting as a group. When they act as a group and are fixated superiorly, they elevate the first and second ribs and are therefore considered muscles of respiration. When they are fixated inferiorly they flex and slightly rotate the cervical spine.

Origin

Scalenus Anticus

Arises from the anterior aspect of the third, fourth, fifth and sixth cervical vertebrae. It lies deep to the *sternocleidomastoideus*.

Scalenus Medius

Arises from the posterior aspect of the second, third, fourth, fifth, sixth and seventh cervical vertebrae.

Scalenus Posticus

Arises from the posterior aspect of the fifth, sixth and seventh cervical vertebrae.

Insertion

Scalenus Anticus

It inserts by a narrow flat tendon into the scalene tubercle on the posterior surface of the first rib.

Scalenus Medius

It inserts as a broad attachment into the cranial surface of the first rib between the costal tuberosity medially and the subclavian groove laterally.

Scalenus Posticus

It inserts into the outer or anterior surface of the second rib lateral to the mid-clavicular line. Its insertion lies deep to the *serratus anterior.*

Action

Scalenus Anticus & Medius

According to the *American Academy of Manual Medicine*, the action of the *scalenus anticus* and *medius* is described as follows:

> '*When it is acting superiorly, it elevates the 1st rib and when it is acting inferiorly, it flexes and rotates the cervical column*'.

Scalenus Posticus

According to Gray's Anatomy, the action of the *scalenus posticus* is as follows;

> '*Raises the second rib; bends and slightly rotates the neck*'.

Clinical Significance

Scalenus Anticus & Medius

The importance of this muscle group is the effect that is borne on the function of the first rib and lateral flexion of the cervical spine. Both will exert a negative influence over the brainstem and carry the capacity to mimic a cranial subluxation, in particular the IVth ventricle compression

syndrome in Type I cranial. The measured neurological effects include the Shimizu reflex, hyperactive pectoral reflex and cranial specific findings such as increased intraocular pressure and asymmetric ocular follow.

Scalenus posticus

The importance of this muscle, so often overlooked or ignored completely, lies in the fact that it affects lateral flexion the same way that the anterior and medial muscles do, but its correction requires a contact on the second rib as opposed to deep inside the anterior cervical triangle.

On occasion the posterior muscle, with its affect of elevating the second rib, may be the cause of a anterior/medial scalene that is not pretesting in the expected way. When this is the case, correction of the posterior muscle will clear the whole clinical picture.

Pretesting procedures

Pretesting is performed by asking the patient to flex their neck in order to move their head into one anterior quadrant at 45° which has the effect of approximating the origin and insertion of the muscle. Previous ocular follow asymmetry will normalise into parallel action, confirming the diagnosis. It is important to perform the ocular follow pre-test not in the Z-axis of the body, but in a line at right angles to a line taken through the pupils in the cranial midline.

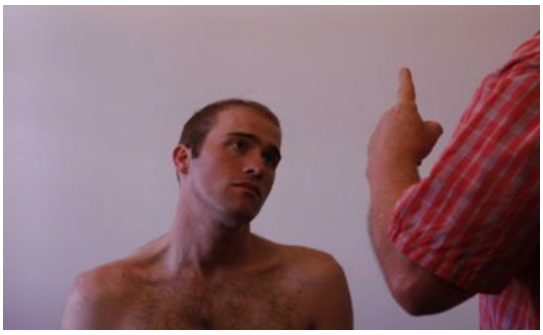


Fig 56: Pretesting the hypertonic *scalenus anticus* and *medius*.

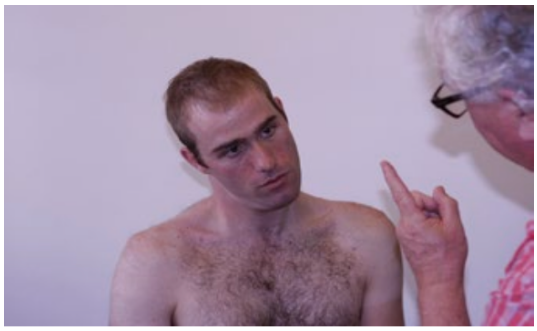


Fig 57: Pretesting the hypotonic *scalenus anticus* and *medius*.

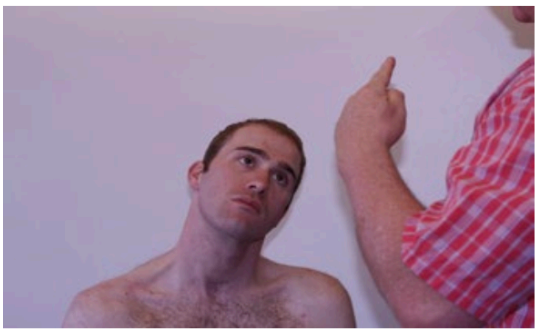


Fig 58: Pretesting the hypertonic *scalenus posticus*.

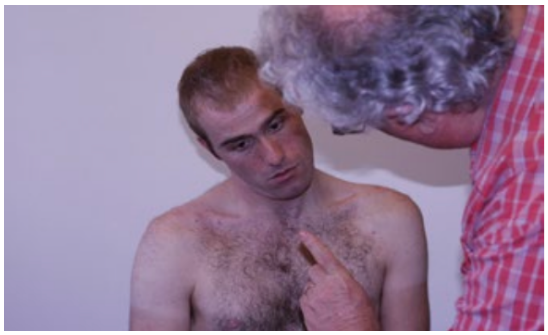


Fig 59: Pretesting the hypotonic *scalenus posticus*.

Treatment Protocol

When the muscle is hypertonic

Scalenus Anticus & Medius

With the patient supine, take a thumb contact over the superior border of the first rib. Impulse is initiated by applying pressure in a downward direction to stretch the muscle and a typical NIP™ thrust is made after 10-15 seconds in much the same way as one would do with a first rib subluxation (Figure 60). It is never necessary to utilise the drop piece when adjusting the anterior neck flexor.

Scalenus Posticus

With the patient supine, take a thumb contact over the superior border of the second rib and ask the patient to laterally flex their cervical spine away from the side of contact. Impulse is initiated by applying pressure in a downward direction to stretch the muscle and a typical NIP™ thrust is made after 10-15 seconds in much the same way as one would do with a first rib subluxation (Figure 62). It is never necessary to utilise the drop piece when adjusting this anterior neck flexor.

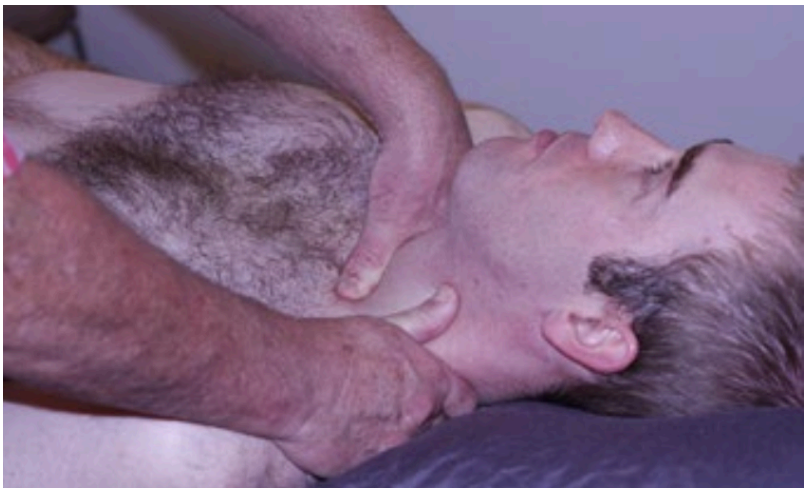


Fig 60: Impulse generation for the hypertonic *scalenus anticus* and *medius*.

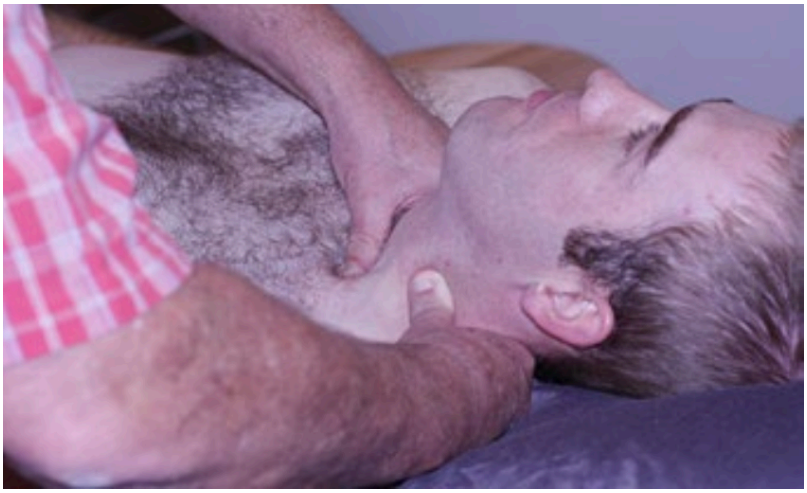


Fig 61: Impulse generation for the hypotonic *scalenus anticus* and *medius*.

When the muscle is hypotonic

Scalenus Anticus & Medius

With the patient supine, take a thumb contact as deeply into the anterior cervical triangle as possible so the thumb lies just above the superior border of the first rib. Impulse is initiated by applying pressure in a cephalad direction and a typical NIP™ thrust is made after 10-15 seconds (Fig 61). It is never necessary to utilise the drop piece when adjusting the anterior neck flexor.

Scalenus Posticus

With the patient supine, take a thumb contact over the anterior surface of the second rib and ask the patient to laterally flex their cervical spine towards the side of contact. Impulse is initiated by applying pressure in a cephalad direction and a typical NIP™ thrust is made after 10-15 seconds (Fig 63). It is never necessary to utilise the drop piece when adjusting the anterior neck flexor.

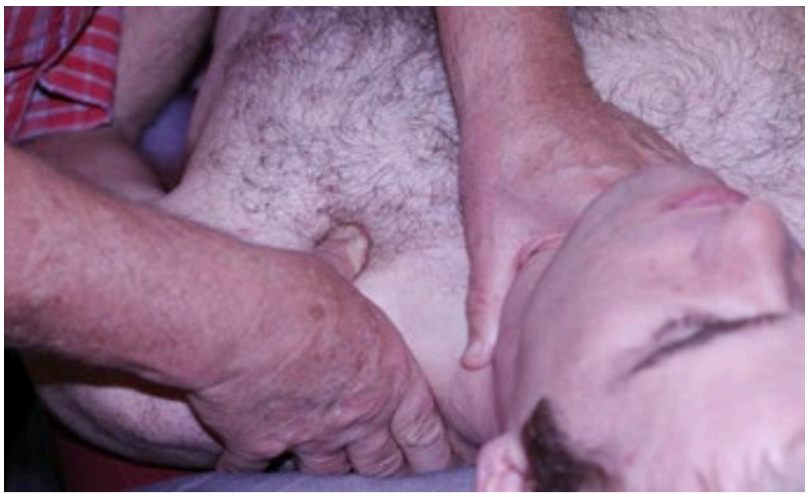


Fig 62: Impulse generation for the hypertonic *scalenus posticus*.

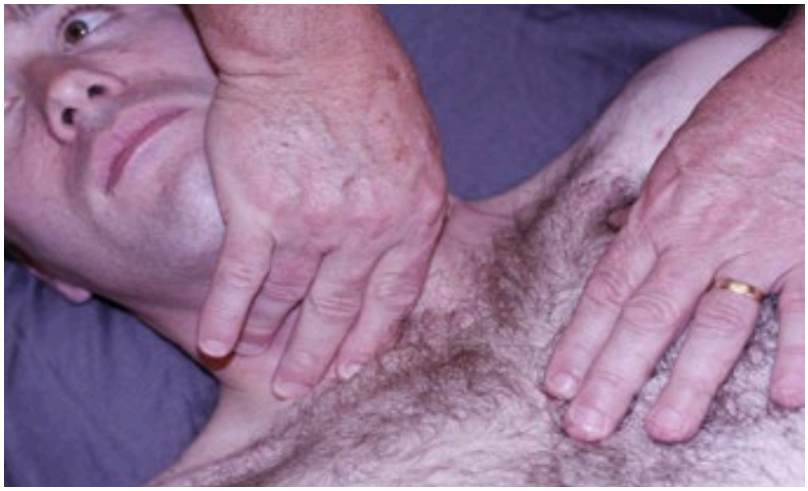


Fig 63: Impulse generation for the hypotonic *scalenus posticus*.

The Temporalis Syndrome

Origin

Temporal fossa between inferior temporal line (of parietal bone) and infratemporal crest.

Insertion

Coronoid process and anterior ramus of the mandible.

Action

Elevates mandible and retracts the mandible.

Clinical significance

The nerve supply to all the muscles of mastication, including the *temporalis*, is the mandibular branch of the trigeminal nerve. The significance of this, of course, is the fact that the Vth nerve nucleus is located in the middle pons and extends inferiorly as far as the second cervical level making it subject to negative effect from dural tension and changes in the CSF pressure dynamics caused by upper cervical kinesiopathology.

In addition, the motor root which becomes the mandibular nerve, like the two sensory roots pass through the Gasserian (also known as the trigeminal or semilunar) ganglion which lies between the layers of the duramater in a hollow in the floor of the middle cranial fossa known as Meckle's cave. This anatomical intimacy of the motor fibres of the trigeminal nerve with the duramater in the middle cranial fossa and the location of the nucleus in the lower reaches of the brainstem make the connection of upper cervical dysfunction and the temporalis syndrome obvious.

This explains why the typical upper cervical subluxation, when corrected, has a huge effect on TMJ function and superficial cranial pain seen so commonly over the location of the temporalis muscle.

In addition to the neurology, the origin and insertion of the temporalis afford it the opportunity to cause dysfunction of the parietal, temporal, sphenoid and frontal. Assessment of the timing of the mandibular movement at the TMJ is critical in making the right diagnosis.

Pretesting procedures

When the muscle is hypotonic, the TMJ on the involved side will open before its opposite and vice versa when the muscle is hypertonic. In terms of the cranial faults, the hypotonic muscle tends to lead to dropped sphenoid, frontal and temporal with the parietal unaffected. The hypertonic *temporalis* tends to lead to sutural closure/jamming.

Before looking for a sutural subluxation one should see if indeed the temporalis is hypertonic. Adjusting the dropped sphenoid/frontal/ temporal will usually restore normal tone to the temporalis muscle. It is reasonable, however, to pre-test the temporalis when a diagnosis of a dropped sphenoid, temporal or frontal has been made. It is one of the more obscure reasons for a persisting Shimizu reflex. The pre-test for a hypotonic temporalis is achieved by taking a contact over the origin and insertion and approximating them gently as the neurology is retested (Fig 64).

The pre-test for a hypertonic temporalis is achieved by taking a contact over the origin and insertion and stretching them gently apart as the neurology is retested (Fig 65). An alternative pre-test is to locate the expected trigger point which will normally be in the belly of the muscle located just anterior to the ear at the level of the top of the pinna. Tissue depth pressure is applied to the trigger point and the neurology is pretested.

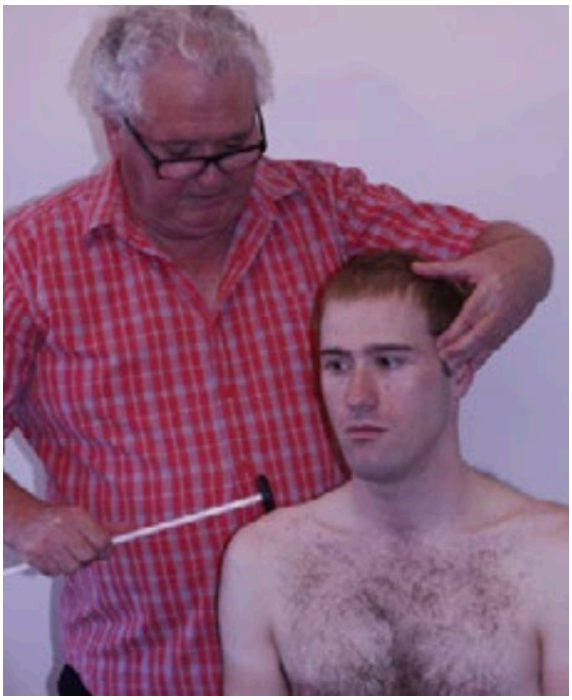


Fig 64: Pretest for the hypotonic *temporalis*.

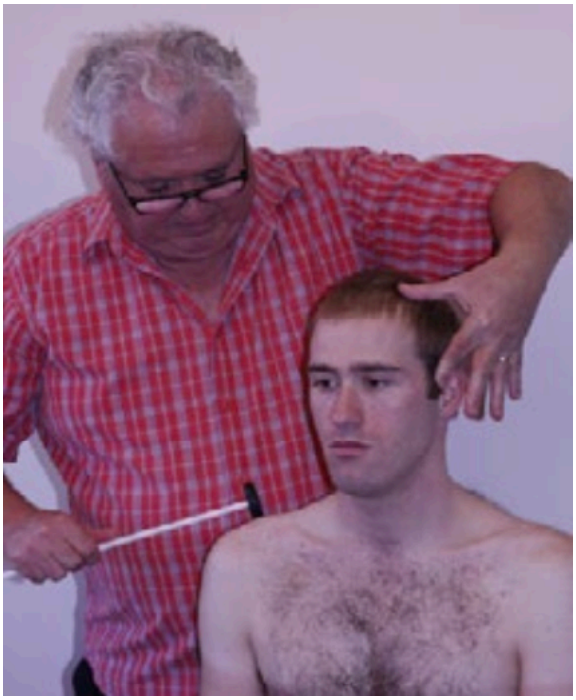


Fig 65: Pretest for the hypertonic *temporalis*.

Treatment protocol

The treatment protocol for the temporalis muscle depends entirely upon whether it is hypotonic or hypertonic. This will be determined by the presence of a dropped sphenoid, temporal or frontal (hypotonic) or cranial indications that don't pretest which would suggest a sutural subluxation (hypertonic). In the latter instance, a trigger point will be present as described above.

When the muscle is hypertonic

For the hypertonic muscle, the patient lies supine and a firm contact is taken over the trigger point affected area of the muscle. The muscle is then lifted superiorly to tissue tension and held through several respiratory cycles.

When the muscle is hypotonic

Treatment of the hypotonic muscle is achieved by taking simultaneous contacts over the origin and insertion of the muscle and gently approximating them for several respiratory cycles. No thrust is ever made. In either case, correction can be considered achieved if the neurology is normalised and the jaw opens simultaneously on both sides.

The t-zones

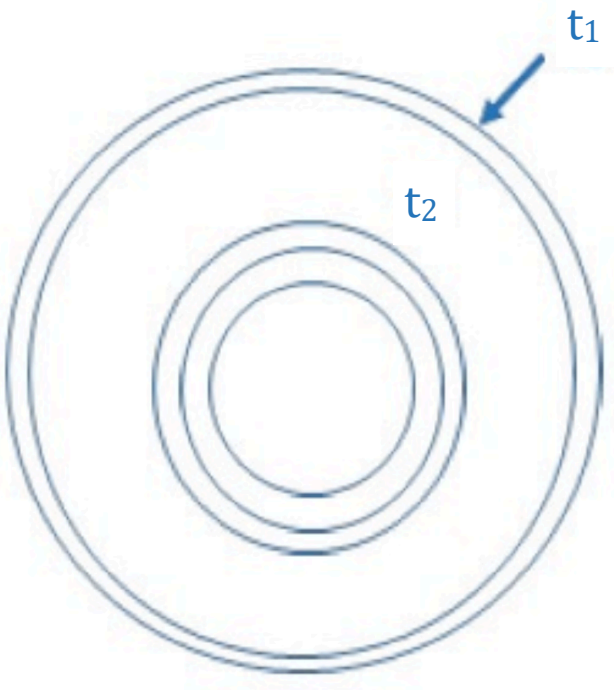


Fig 66: A conceptual representation of the t-zone demonstrating variability in magnitude according to the density of patterns of dysafferentation in relation to their depth in the cortical structure.

In its original concept, the t-factor, or t-zone as it is now referred to, is a period of time in which the patterns of dysafferentation of a given patient is acquiescent and quiet, a period of inactivity so to speak. This concept served the explanation of the protocol well enough until it became clear that the t-zone is anything but a quiet, acquiescent period of neurodysfunctional activity.

What determines the presence of a t-zone in the first place is a condition in which a patient fails to demonstrate any of the elements of the NeuroImpulse Protocol™ that are characteristic of an active pattern that can be measured and quarantined by precise intrusion. It was originally thought that this lack of measurable activity was a result of patterns located deeper in the cortical substance that were yet to make their way to the surface where they could indeed be measured and addressed.

At least three types of neurodysfunctional activity, namely dark patterns, fragmented patterns and pattern neogenesis may be active in any given patient during an extended t-zone.

Dark patterns

For me, astronomy and its near relative astrophysics, is the most awe-inspiring of the sciences. To look up on a truly dark night, away from the light pollution of civilisation, is to be struck by the sheer enormity and raw beauty of creation. But modern astronomy teaches that the celestial bodies that can be seen with the naked eye are not even half of what is out there. The matter that can be seen spread across the sky is in the form of planets, comets, stars, nebulae and galaxies, etc, though they be numbered in the billions, make up just fewer than 15% of the total mass in space.

Astronomers have dubbed the remaining 85% 'dark matter' because it neither absorbs nor gives off light and therefore cannot be seen with the tools currently available to scientists today. Although they are confident it is real, they know nothing about it directly. How can scientists be so sure that something that is invisible is nevertheless there? The simplest answer is that there seems to be too much gravity in the universe. The amount of matter that scientists can see through their telescopes is far too small to explain things like the structure of galaxies and the way in which stars within them move. In other words, while the dark matter cannot be seen directly, its presence can be inferred by observing its gravitational effects on that which can be seen.

One very close analogy is the discovery of the planet Neptune in the 1800s. Neptune's existence was predicted before it was observed, when scientists noticed that the orbit of Uranus, Neptune's nearest neighbour, wasn't quite what Isaac Newton's theory of gravity said it should be. Assuming the existence of an eighth planet in the solar system solved that problem nicely.

Utilising the same deductive logic, assuming the existence of a large quantity of extra mass neatly explains the behaviour of the universe on very large scales. A similar situation exists with the electromagnetic spectrum.

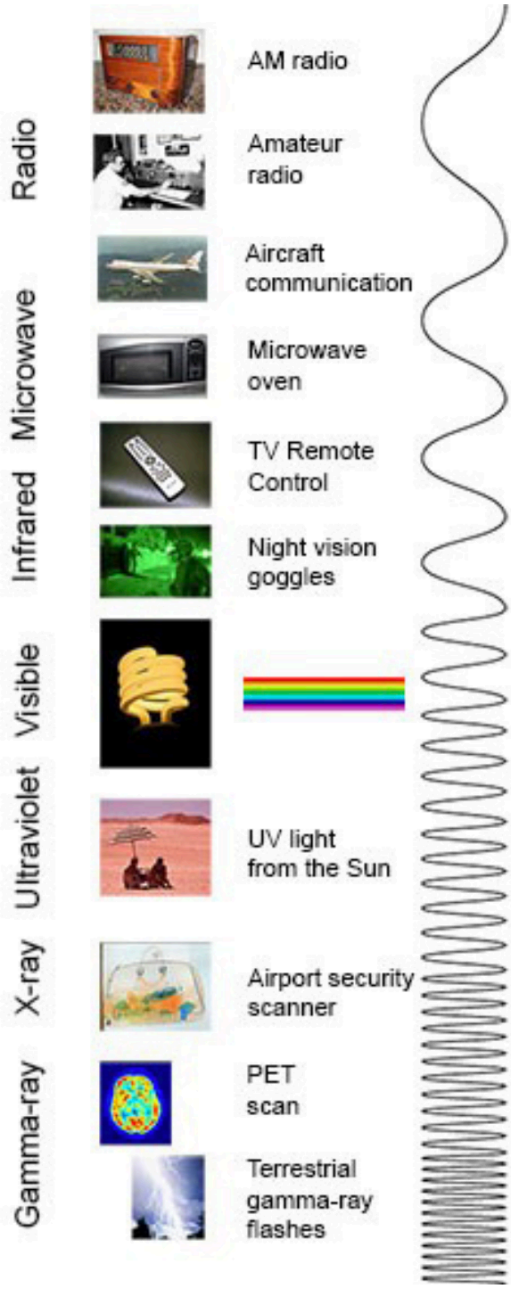
Consider this chart

The visible spectrum is very narrow compared to the non-visible long wave length radiation and the non-visible short wavelength radiation, but both of these can be measured and are evidenced by the effects they exert in nature. Step out in the sun for too long and you will soon become convinced of the presence of ultraviolet radiation.

The reason I have included this discussion on dark matter in space and the electromagnetic spectrum is because the concept in both cases exactly parallels a phenomenon in functional neuroscience that we have become aware of in NIP™, which of course is the topic of this discussion; dark patterns, the next frontier in NIP™ and functional neurology.

I think it is now beyond question that 'dark' patterns exist as we are able to demonstrate their presence by the effects they have on what is generally referred to as the 'soft' and 'semi-soft' neurological signs.

These dark patterns can be shown to exist in the acquiescent space between measurable patterns referred to as the t-zone. Here there is no information derived from the usual application of biomechanical assessment, elicitation of reflexes, muscle strength testing and other hard, focal neurological signs. This raises the uncomfortable notion of patterns of dysafferentation, active but not within the visible spectrum in the t-zone, exerting a negative influence over the function of the nervous system throughout the brain; supratentorial, subcortical, brainstem, reticular formation, cerebellum and the sympathetic structures.



It seems apparent that 'dark' patterns reside in the deeper regions of the brain and are related to areas of primordial structure and early development. While more detailed discussion will follow, a patient with manifest 'dark' patterns may present as generally hyper-reflexive yet have no evidence of upper motor neuron disease, a typically paediatric phenomenon. In addition, the deficits which appear are signs which are normally seen in neurodevelopmental delay such as synkinesis, dysdiadokokinesia, motor impersistence and laterality. Short–term auditory memory is yet to be assessed.

What also seems apparent, but warrants more detailed study and analysis, is that performance of these neurological tests, or 'soft' and 'semi-soft' neurological signs, forces these 'dark' patterns out into the visible or measurable spectrum where they can be identified and quarantined. All well and good, but what is not known at this point and needs to be identified is:

- How long do they remain measurable and therefore vulnerable to quarantine after the stimulation of the neurological testing ceases?
- Does their vulnerability to quarantine by the application of impulse follow the inverse Square law as one might expect? The divergence of a vector field which is the resultant of radial inverse-square law fields with respect to one or more sources is everywhere in the physical universe proportional to the strength of the local sources, and hence zero outside sources. Newton's law of universal gravitation follows an inverse-square law, as do the effects of electric, magnetic, light, sound, and radiation phenomena. It seems reasonable to assume then that neurological patterns, being essentially electrical phenomenon, would follow the same laws of physics, the only difference being that distance is measured by the time that elapses from the stimulation that brought the 'dark' pattern into the measurable spectrum.

Application of the Principle

Dark patterns can only be stimulated to the place where they become measurable when all active cortical surface patterns have been quarantined and the patient is situated neurologically in a t-zone. The genesis of dark patterns can be any kind of stimuli that has exerted an adverse effect on the nervous system anywhere along life's journey from birth to the present time. It is now beyond question that negative, stressful in-utero experience may also create neurological dysfunction that becomes dark patterns as the child grows into maturity and adulthood.

Barbara Holden Nixon of the *Urban Child Institute* has said:

> *'The brain is the primary stress organ: It is responsible for activating, monitoring and shutting down the body's reactions to stress. Infants' developing brains are particularly vulnerable; babies are affected by stress even in the protective environment of the womb. Since maternal cortisol levels affect the developing foetus, a mother's level of stress is directly related to the well-being of her baby. Positive and tolerable stress levels are safe, but toxic stress increases the risk of preterm delivery, low birth weight and other complications. It is also associated with impaired mental, behavioural and motor development in infancy'*

There is now no question that that which is neurotoxic to the pregnant mother is also neurotoxic to the developing foetus (Nixon 2012).

In principle, the stimuli that will be needed to discover a dark pattern will fall into one of two clear categories; specific neurological and age related stimuli that 'awakens the brain' to an insult which has caused a dysfunctional pattern to arise at some point in the patients past or a general stimuli that exerts a mild toxic effect on the nervous system and thus exposes a dark pattern. A

detailed description of each of the specific tests that relate to the stimulation of dark patterns is given in the booklet entitled 'NIP™ Dark Pattern Testing Manual' [email Dr Davies for your copy]

Quarantining a dark pattern

Similar to latency, one cannot drive a dark pattern into quarantine unless it has first been activated and is exerting measurable effect on the cortical surface. Once this has been achieved, however, the intrusion one needs to make must be done so in the posture or under the influence of the stimulus that exposed it. For the most part, a stimulus is applied to the patient who is seated or standing XYZ neutral.

The stimulus exposes the pattern which is now able to be measured, the pattern is determined and the intrusion is made. While that particular dark pattern is unlikely to recur, there may be many dark patterns that will evolve with time as the patient is managed. There are, of course, dark patterns whose exposure is dependent upon sustained stimulus. A typical example is the patient whose pattern becomes exposed due to sustained sphygmomanometer pressure. The pressure should be maintained during the process of making the intrusion due to the fact that releasing the pressure will see the pattern disappear very quickly.

Exposure to white noise is the same and needs to be sustained while the intrusion is executed.

Fragmented patterns

Consider the conceptual representation in Fig 67.

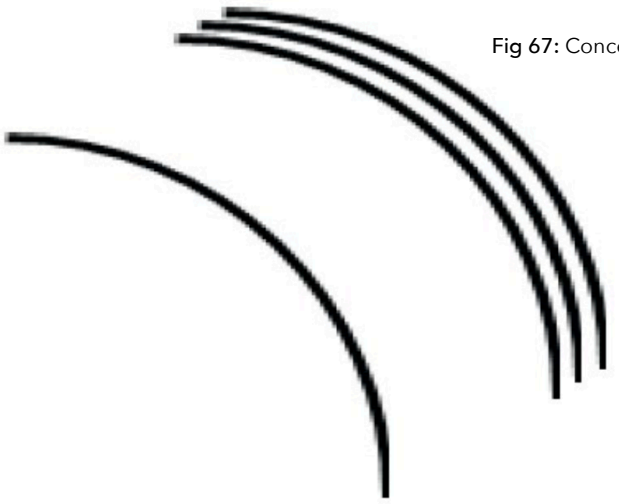


Fig 67: Conceptual diagram of multiple patterns.

In this particular case, there are three patterns with a very, very short t-zone between them, then a long t-zone to the next pattern. Our original conceptualisation of this situation, very common in general practice, is that there are 4 distinct patterns with variable periods of quiet acquiescence between them. In order to quarantine all four patterns, the NIP™ chiropractor needs to wait out each t-zone until the next pattern becomes measurable and therefore susceptible to being quarantined.

This concept and process is indeed a reality and will suffice as the management strategy for many patients, but by no means all of them. The problem we must face with brutal honesty and academic integrity is related to the change that occurs when the intrusion is made. In many cases, like the example in Fig 67, the patterns are seemingly independent of one another and all three will need to be sequentially quarantined.

In other cases, all the elements of all three 'patterns' are eliminated when the intrusion is made, raising the very reasonable question of whether that was a single pattern with multiple facets, a question I believe we have now resolved in the affirmative and which in fact has no meaningful clinical bearing on patient management. In point of fact, these patients seem to be the happy recipients of very rapid and sustainable symptomatic improvement.

There is a third scenario, however, and a very troublesome clinical scenario it is. The multi-faceted pattern is identified and an intrusion is made. The kinesiopathological features of that pattern for the most part are eliminated, but maybe one element is not quite resolved. A typical case is the patient with a sacrum who when adjusted demonstrates all elements resolved except for full head rotation to one side. More troublesome than this mechanical element, however, is the persistence of abnormal neurology, in particular the Shimizu reflex. This pattern is understood to have fragmented and needs to be quarantined as a fragment rather than be allowed to remain to produce a full blown pattern, a process referred to as 'pattern neogenesis'.

The following flow chart identifies the step by step process required to identify a fragmented pattern and how to quarantine it.

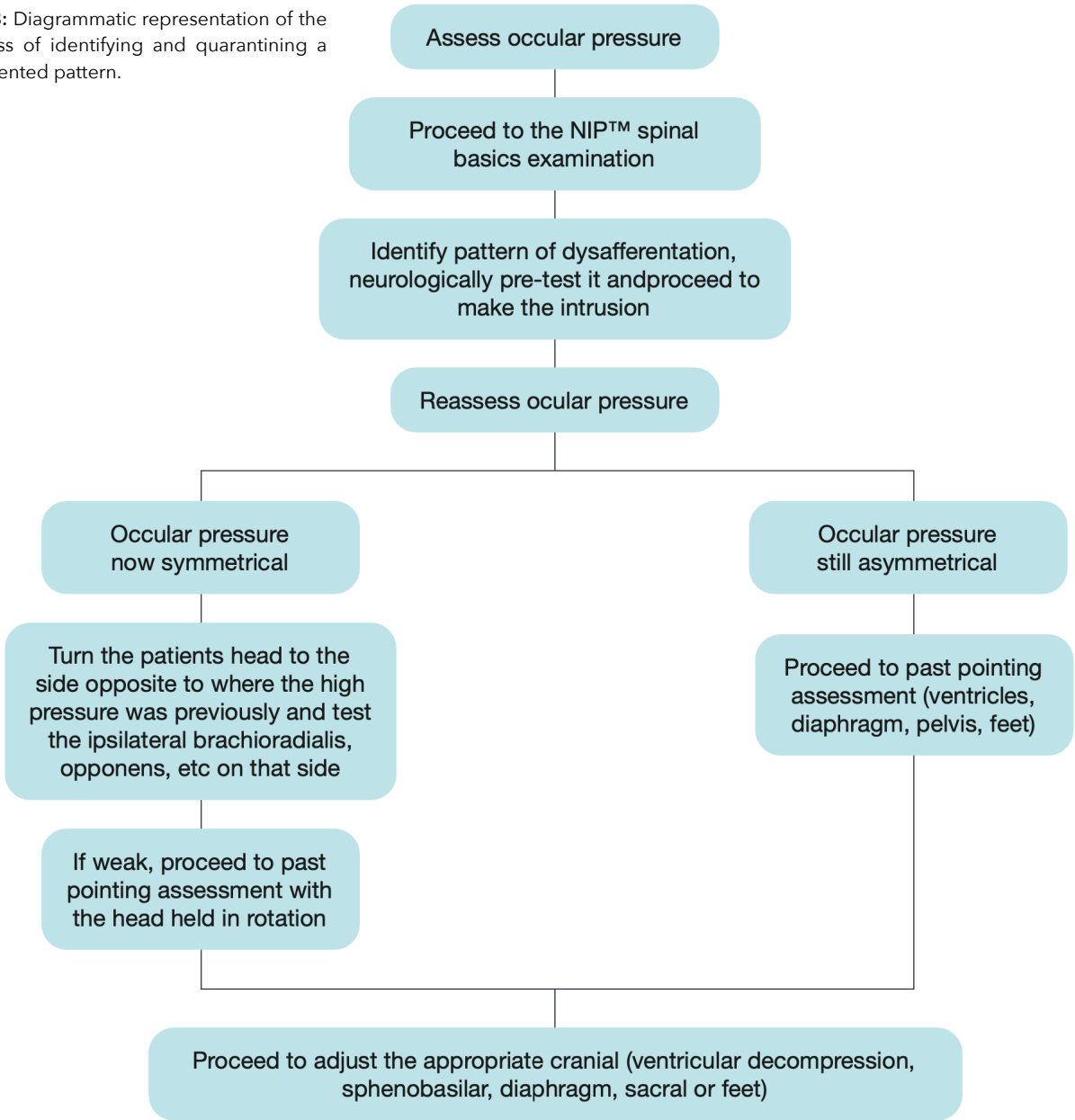


Fig 68: Diagrammatic representation of the process of identifying and quarantining a fragmented pattern.

Pattern neogenesis

The problem with pattern fragmentation if not identified and quarantined is the process of pattern neogenesis, a process all NIP™ practitioners are familiar with, to their endless frustration. Once a pattern has fragmented, if the fragment is not identified and quarantined quickly, it will, in the true sense of the word, metamorphose into a full blown pattern and be there waiting for you at the patients next visit. In particular, this is the patient who leaves your clinic with a definite Shimizu reflex still active. In some cases, the pattern that results from this neogenesis process will be the same as that which was there originally, but in other cases it will be quite different, reasonably leading one to conclude that the next deeper pattern is emerging and therefore progress is being made when it is not.

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Also by Dr Neil Davies

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About the NeuroImpulse Protocol (NIP)

The NIP technique came about during the 1990's. It is the brainchild of Dr Neil J Davies, the author of the popular text "*Chiropractic Pediatrics A Clinical Handbook*" published by Churchill Livingstone. During this time Dr Davies was striving to evolve a technique approach to be used with babies that was absolutely precise neurologically, but extremely gentle in its application.

The NIP technique is used exclusively throughout Dr Davies' Chiropractic Clinics in Victoria, Australia and the result has been nothing short of astonishing in terms of new patient numbers. Patients drive long distances to receive NIP technique because the results they are getting are quick and sustainable and they don't get hurt by hard manipulative style techniques

Contact

https://neuroimpulse.net/

Note for readers

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