Brain stem infarcts are less common than hemispheric infarcts but it is often difficult to localise them exactly to a particular vascular territory. With knowledge of their clinical features and some neuroophthalmological signs that they may have, the physician’s ability to localise them to an anatomical site and vascular territory may be improved. When one is faced with a patient with varied clinical signs and symptoms and where a stroke is suspected, one of the first important things to be done, is to find out whether he has a hemispheric infarct or a brain stem infarct. There are some pointers such as some special clinical features associated with strokes of individual varieties and the knowledge of brain stem anatomy which would help physicians distinguish one set from the other. The second important thing, in the diagnosis of brain stem strokes, is to determine the site of the occlusion and the likely pathogenetic mechanism underlying it. The third thing one may be required to do, is to determine whether infarcts have occurred at multiple levels in the brain stem or not, because multiple level infarcts are not easy to localise.

In this text the approach to the localisation of brain stem infarct is discussed in 3 main steps which the physicians will find very helpful. First of all, one has to distinguish between a brain stem infarct and a hemispheric infarct, and there are some clinical features that would give clues to one or the other. The second step will be recognising brain stem infarction syndromes in single brain stem vascular territories and the third step will be identifying the difficult type multiple level brain stem infarctions which have overlapping features.

How to differentiate between a brain stem and a hemispheric infarct?

Here, one starts by picking on some special symptoms that point to the brain stem disease. One of the first such cardinal symptoms is vertigo, which one must look for in the history, and if the patient has vertigo, it should immediately point towards a brain stem disorder. The second important feature, is the presence of cranial nerve symptoms or signs in the history or on physical examination. The third important pointer is the presence of crossed signs, i.e., presence of ipsilateral motor and sensory cranial nerve signs or symptoms and contralateral hemiplegia, hemianaesthesia, or both. The fourth important pointer is the presence of oculomotor signs. These features are some of the primary and common ones that the physician would need to pickup immediately and which suggest a brain stem disease. The fifth important pointer is bilateral simultaneous involvement of long tracts, either sensory or motor, either symmetric or asymmetric, either simultaneous or sequential. For example, if there is a patient of stroke who has simultaneous bilateral hemiparesis or sequential hemiparesis, first on one side of the body and then after a while or the next day, on the other side, one should think of brain stem. One may occasionally see a patient where one day, he finds difficulty using the right hand and holding something with it and next day he may develop a contralateral hemiplegia. Such confusing signs may mislead the clinician to make an incorrect diagnosis, but in fact they suggest bilateral involvement of long tracts and are a pointer to a brain stem problem. Simultaneous or sequential bilateral sensory alterations are equally important in suggesting that both sides have been involved and they must be picked up when present,
to help the clinicians to distinguish brainstem infarcts from hemispheric infarcts. The next important point to note is the presence of unsteadiness of gait or ataxia, and dysarthria-clumsy hand syndrome. Dysarthria-clumsy hand syndrome means slurring of speech plus clumsiness of the hand. If all the criteria defining this syndrome are present in the patient, they usually point to a brain stem lesion, but the same may also occur with lesions of internal capsule.

There are some rare features of brain stem diseases which are not encountered frequently, but once one sees them they should immediately point to the physician that he is dealing with a brain stem problem. One of them is called “salt and pepper” feeling in the face. It simply means a feeling of stinging “needles” in and around the eyes, as if “salt and pepper” have been thrown in the eyes. It suggests involvement of the quintothalamic tract in the pons and the medulla. Another uncommon pointer may be presence of blepharospasm, which has been reported with infarcts involving the brain stem. Hiccough is another symptom that may be present although not very prominently and it can be missed or not given enough attention as a brain stem symptom. Hiccough might mislead one to think of some diaphragmatic or stomach problem rather than a brain stem disease. Trismus, clenching of teeth and bruxism, i.e., grinding of teeth can also be present in basilar artery occlusion. A roaring sound in the ear may be perceived quite often by patients at the beginning of a medullary infarct. Similarly, decreased hearing is often a sign of anterior inferior cerebellar artery occlusion. Palatal myoclonus may also occur rarely with brain stem infarcts. Respiratory dysfunction may be present and when seen in a patient of acute onset vertigo, it should point not always to a pulmonary or a cardiac cause, but also to an acute brain stem dysfunction. One needs to be careful of patients who present with acute vertigo and then suddenly develop respiratory failure, because medullary infarction is a well described cause for brain stem strokes. Then there is a special type of hallucinosis called “peduncular hallucinosis” where there are visual hallucinations manifesting as repetitive seeing of small objects in the visual field. This symptom can arise from a mid-brain infarct.

In pure hemispheric cortical infarcts, the clinical picture is often straightforward. For example, presence of specific features like seizures, aphasia, hemianopsia, or confusion immediately point to a hemispheric cortical disease, but in some cases there could be great difficulty in differentiating one from the other. An area where a physician’s clinical acumen becomes most important is where the syndrome produced in the brain stem and the hemisphere are similar looking. The confusion here, is mostly with subcortical lacunar syndromes such as the pure motor strokes, ataxic hemiparesis, pure sensory strokes and mixed sensory motor strokes. These lacunar syndromes can be due to small lacunar infarcts both in the internal capsule and brain stem locations. So the physician has to use his clinical acumen and try to pick up some subtle signs accompanying these syndromes that tell him the difference between a brain stem and a capsular infarct.

Decline or loss of consciousness can be present in both brain stem and hemispheric strokes, but usually when it is a brain stem problem it comes on pretty early more or less at the onset of the stroke, whereas when it is in the hemisphere it might come a little slowly. But this is not a great difference because large hemispheric infarcts can be associated with decline in consciousness. There is another feature which is important but also confusing and that is appearance of repeated clonic jerks or shaking of a limb. These jerks look like seizures and if the physician sees them or is told about them at the first sight, he begins to think of epileptiform convulsions. They are often mistaken for seizures and even treated with phenytoin and other drugs as such, but it is now well described that one can get these rapid clonic jerky movements in brain stem problems particularly with occlusion of the basilar artery. It has been known that basilar artery occlusion can give small amplitude jerky
movements of the arms or the legs. There are a few other important differentiating points as well, that can indicate that these are simple clonic movements and not real seizures. The first point is, that in brain stem clonic movements, the amplitude is not as wide as it is in a seizure. The second point is, that one may see them in an alert state whereas seizure patients are usually unconscious or have some disorder of consciousness.

Conjugate eye deviation can be another point for differentiating one from the other. It can be present in both hemispheric or brain stem strokes, but there are important distinguishing features to point the diagnosis to one or the other. If the eyes look towards the lesion, the lesion is hemispheric; on the other hand, if the eyes look towards hemiparesis, the cause is in the brain stem. Pseudobulbar symptoms may be seen in both hemispheric as well as brain stem lesions. Also, there could be orthostatic symptoms in brain stem disease which have not received adequate recognition by physicians. Dizziness or loss of consciousness on standing, walking or appearance of focal signs on standing or walking, can be due to brain stem disease, especially bilateral vertebral artery disease.

The syndrome of pure motor hemiparesis

Pure motor hemiparesis can be an important brainstem sign and one sees this type of syndrome commonly in clinical practice. It occurs in lesions at 3 brain stem sites: i) cerebral peduncle; ii) basis pontis; and iii) medullary pyramids. The pons is the most frequent brain stem site for pure motor hemiplegia. Any hemiparesis which has a cause located in the brain stem, means affection of the pyramidal tracts and since the pyramidal tracts in the brain stem are located anteriorly, the cause of hemiparesis must be located anteriorly as well. The pyramidal tracts run right down the front part of the brain stem. This is one of the first clues for localisation of such lesions.

Features that point to midbrain lesions

One may be able to tell whether a lesion is in the cerebral peduncle of the midbrain because there may be ipsilateral oculomotor paralysis with contralateral hemiplegia, the so called Weber syndrome. The presence of a cranial nerve deficit will tell the physician what the rostro-caudal site of the lesion is, whereas the presence of the pyramidal tract involvement will tell him the ventro dorsal location of the lesion. For example if the 3rd nerve is involved, one knows that the lesion is in the midbrain and not in the pons or the medulla. This is a very important anatomical principle, on which the diagnosis of brain stem strokes is based, i.e., the localisation on the basis of a ventrodorsal and rostro-caudal level of the lesion through the cranial nerve deficit and pyramidal tract or other long tract involvement.

In the history given by the patient of stroke with pure motor hemiparesis, there are several symptoms that point to a brain stem disease for example the presence of dizziness or vertigo. Vertigo is usually described by patients as dizziness, because they cannot always differentiate between the two. This means, that if the patient complains of dizziness, the physician needs to check whether he really means vertigo.

A transient gait imbalance is usually due to ataxia and if ataxia is present it is an important brain stem sign as well.

Features that point to a pontine lesion

Complaint of double vision in the history or a sensation of ear canal blockage are important signs of pontine involvement. However, there are other signs that one may find in the neurological examination that will help the physicians to localise the lesion to the pons including dysarthria which will distinguish a pontine lesion from other levels. If the patient has a combination of severe dysarthria and a history of vertigo or transient gait abnormality, it suggests involvement of pons rather than a hemispheric lesion, particularly if the patient...
has right hemiparesis, but clinical findings do not definitely distinguish between capsular and pontine levels. For some reason, in right hemiparesis of brain stem origin, patients have more dysarthria than the hemiparesis on the left and this can easily be confused with motor dysphasia. Another sign is the presence of extensor posturing in the arms or the legs or both, and one can see bilateral Babinski sign in some patients who have only unilateral manifest hemiparesis. The ratio of pontine to capsular location of infarct is about 3 to 7.

Another important sign is the presence of horizontal nystagmus and one should look carefully for it and should make sure that it is a definite, sustained nystagmus and not the end point nystagmoid jerks that are seen frequently in normal individuals. These nystagmoid looking jerks are of small amplitude; they do not last long and tend to disappear. Another important sign of pontine involvement is the ease of conjugate eye movements which seem to occur more easily when the gaze is turned towards hemiparesis than away from the hemiparesis. The reason for this phenomenon is that the pontine lesions here extend, sometimes backward to the PPRF (paramedian pontine reticular formation) and therefore the patient gets a subclinical gaze palsy. This is not a real gaze palsy, it is just that the conjugate eye movements are easier on one side than the other. But one has to look for it carefully. Contralateral limb dysmetria may also be seen and this is due to extension of the lesion laterally to involve the middle cerebellar peduncle. One may also notice gait imbalance. These patients have a tendency to slump when they are sitting more than when they are standing. One may also note loss of sensation of the face, if one tests carefully, which suggests involvement of the root of the trigeminal nerve or the tract.

The cause of these lacunar infarcts, which are often due to occlusive vascular disease, is located either in the paramedian perforating arteries, which are branches of the basilar artery or in the basilar artery itself which is occluded. It has now been know that if the infarct extends to the surface of the pons, it is more likely to be due to occlusion of the basilar artery, in the background of atherosclerosis on its intimal lining, and if the infarct does not extend to the surface of the pons, it is more likely to represent a perforating artery disease. So syndromes developing from these occlusions are correspondingly more severe because the resulting infarcts extend to the basal surface. Pure motor hemiparesis is not always a straight-forward condition caused by a lacunar infarct resulting from occlusion of a perforating branch of the basilar artery. It can also be due to a serious basilar artery narrowing and one must not forget this fact. They can also present as a temporary hemiplegia which improves and then recurs again in a few hours to a few days and rarely may recur many times. This early temporary hemiparesis has been called a “herald hemiparesis" by Miller Fisher and it signifies basilar artery occlusion. It is important to note that such hemiplegias do not always occur once but several times in the course of the illness.

Pure motor hemiparesis can also occur as a result of lesion in the medulla, caused by infarction of the medullary pyramid where the corticospinal fibres are concentrated. In the medulla, if the infarct extends backward to involve the medial lemniscus, a sensory motor hemiplegia may also result. It quite often involves the exiting fibres of the hypoglossal nerve to give rise to what is called the Dejerine syndrome where the ipsilateral half of the tongue is paralysed together with a contralateral hemiplegia. Ataxic hemiparesis

This is an interesting condition that can occur from a lesion either in the internal capsule or in the pons. The typical location of the lesion is in the rostral pons, close to the midline. In a lesion located close to the midline, one would expect bilateral ataxia and the reason for this is that the corticopontine fibres descending from the cortex to the pons cross over to go to the opposite cerebellum. So a lesion located near the midline
tends to affect fibres crossing over to both right as well as the left and so they give rise to bilateral ataxia. The usual thing however, is to see a unilateral ataxia. Other features that may be associated with ataxic hemiparesis as also in pure motor hemiparesis, are dysarthria, nystagmus, and trigeminal sensory loss. One may see ataxia either contralateral to hemiparesis or ipsilateral to hemiparesis. This is due to the critical location and extension of the lesion. If the lesion does not involve the crossing fibres from the other side because the crossing fibres may have an oblique course or if it is a little more caudal or lateral, the ataxia may be unilateral. So ataxic hemiparesis whether bilateral ataxia, ipsilateral ataxia or contralateral ataxia can be explained by the way the pyramidal fibres cross over14.

Pure sensory strokes

This is also seen frequently in a brain stem lesion15. An important clinical observation is that, it may vary from level to level based on the anatomy of the brain stem and its arterial supply. In the medulla, the medial lemniscus and the spinothalamic tract are well separated from each other, so usually with a small lacunar infarct in the medulla, either one sees posterior column loss, i.e., loss of joint sensation and vibration or loss of pain and temperature sensation, because here the tracts are separate and their arterial supplies are different. As these two sensory tracts ascend up the pons, the medial lemniscus and the spinothalamic tracts come much closer, so in a lesion in the pons, one is more likely to see a combined sensory loss of both pain and posterior column type. However, one can still get lacunar infarcts involving one or the other of these. Very frequently these patients present with burning dysaesthesia. The superior cerebellar artery territory sometimes separates the two tracts and in the occlusion of the superior cerebellar artery one may get a pure pain and temperature loss. As these tracts ascend further up to the inferior midbrain, the tracts have come even closer and they are likely to be affected together. As described earlier, as anywhere else in the brain stem, in midbrain also, stroke patients have more sensory involvement, if the lesion is more posterior and have more motor involvement if the lesions are located more anterior. Whether the symptoms are more sensory or more motor gives a clue to the ventro dorsal localisation of the lesion that caused them.

Brain stem artery syndromes

The next important clue in recognizing brain stem infarcts is to localise single brain stem artery syndromes. These are the syndromes resulting from occlusion of paramedian mesencephalic arteries, the basilar penetrating arteries in the pons, the superior cerebellar artery, the anterior inferior cerebellar artery and the posterior inferior cerebellar artery in the medulla.

Mid brain paramedian arterial territory syndrome

(a) “Locked in” syndrome

Occlusion of midbrain paramedian arteries can give rise to a “locked in syndrome with ocular palsy”16. Such patients have a sudden onset of vomiting and they may lose consciousness transiently but the most important feature is acute onset quadriplegia with bilateral facial paralysis, anarthria, and loss of voluntary eye movements. The eyes are often directed laterally and downwards. In the usual “locked in” syndrome the infarct is located in the upper part of pons. In the pontine “locked in” syndrome however the oculomotor nerves are not involved and the patient although speechless, can move his eyes voluntarily. In the midbrain “locked in” syndrome, patients have bilateral nuclear third nerve palsy and the quadriplegia is due to involvement of corticospinal fibres bilaterally, because the pyramidal tracts are supplied by paramedian arteries.

(b) The syndrome of “covergence spasm” and ataxia. (pseudo 6th nerve palsy syndrome).
These patients usually have a sudden onset double vision, unsteady gait and unilateral clumsiness of limbs. They also have reduced or absent eye abduction, upbeat nystagmus and "convergence spasm" which means that the eyes converge. Convergence spasm appears, when there is decreased eye abduction. Superficially it looks like 6th nerve nuclear involvement but often it is not and that is why it is also called “pseudo 6th nerve palsy” syndrome. The detection of convergence spasm is diagnostically important because it immediately indicates a midbrain lesion.

Just to summarise again, the value of oculomotor signs in the paramedian mesencephalic syndromes, one can say that if the lesion is in rostral midbrain, it gives rise to vertical gaze palsy; if it is in the caudal midbrain, it leads to a nuclear 3rd nerve palsy or a fascicular 3rd nerve palsy and if it is bilateral, it might produce a mesencephalic "locked in" syndrome.

About 30% of these small infarcts are due to the atherosclerotic stenosis and about 20% are caused by cardio-embolism and another 20% are due to small vessel disease.

**Individual brain stem arterial syndromes:**

**a) Syndrome of superior cerebellar artery**

This is a syndrome caused by occlusion of the superior cerebellar artery. Patients usually present with sudden nausea, vomiting, ataxia and unilateral incoordination of limbs. It can present as a pure cerebellar syndrome when it can be a little difficult to localise, because ataxia is a nonspecific feature. But the brain stem may also be affected because superior cerebellar artery also sends branches to the pons which may give rise to brain stem signs as well. So one can see an ipsilateral ataxia which is not only due to cerebellar involvement but also due to superior cerebellar peduncle involvement. There may be a Horner’s syndrome, because, the sympathetic fibres may be involved and there may be contralateral pain and temperature loss because the spinothalamic tract may be affected. The touch, joint sensation and vibration are not usually affected because the lesion usually does not extend to the medial lemniscus. In over 70% cases the cause of these occlusions is cardioembolism, but one can occasionally see an artery to artery embolism from the vertebral artery.

**b) The syndrome of anterior inferior cerebellar artery**

These patients present with sudden onset vertigo, vomiting and falling to one side. They have usually marked horizontal nystagmus but the characteristic features are absent sensation on one side of the face, ipsilateral facial paralysis and ipsilateral hearing loss. They also lean to the affected side when you make them sit. The ipsilateral loss of sensation in the face may easily lead one to make a misdiagnosis of Wallenberg syndrome, i.e., the lateral medullary syndrome, but facial paralysis and hearing loss are not features of Wallenberg syndrome. The combination of ipsilateral hearing loss, facial paralysis and loss of facial sensation is a very typical feature of anterior inferior cerebellar artery occlusion syndrome. The 7th nerve is at the pontomedullary junction and the anterior inferior cerebellar artery affects the anterior and inferior part of pons, so it often involves the exiting fibres of the 7th nerve and the neighbouring auditory nerve nucleus and the 5th nerve nucleus. The facial sensory loss is multimodal and affects all modalities including touch, pain and temperature. So a combination of vomiting, ataxia, nystagmus, and ipsilateral 5th, 7th and 8th nerve dysfunction, typically suggests anterior inferior cerebellar artery syndrome. If it is a pure anterior inferior cerebellar artery syndrome it is usually due to atherosclerosis of the artery in the background of diabetes. If it is combined with other infarcts in the brain...
stem or the cerebellum, the cause is usually basilar artery disease.

c) The lateral medullary syndrome of Wallenberg: (Or the syndrome of posterior inferior cerebellar artery.) It is one of the most common of the brain stem syndromes. These patients have sudden onset nausea, vomiting, and dizziness or vertigo. They tend to fall over to the affected side when they are made to sit in the bed. Falling to one side, when sitting is called “lateral pulsion”. They may also complain of a spinning sensation and falling to one side, while standing or trying to walk. They often have double vision which is usually vertical, i.e., the images are one above the other. They also have a down beat nystagmus and decreased pin prick sensation on one side of the face. There is also loss of pain and temperature sensation on the opposite side of the body, i.e., in the opposite arm and lower limb. There is no motor weakness but incoordination of limb is present ipsilaterally along with Horner’s syndrome, hoarseness of voice, and dysphagia.

These findings are very characteristic of the lateral medullary syndrome. One thing which is remarkably absent, is the involvement of the corticospinal tract, such as an extensor plantar response either unilaterally or bilaterally. This immediately points to the fact that the lesion is not located in the anterior part of brain stem where the corticospinal fibres are concentrated and that it is in the relatively posterior part and this is how one localises it dorsoventrally. Downbeat nystagmus, nausea, vomiting, and dizziness are typical features of lesions in the lower part of brain stem that is in medulla. Numbness of the face suggests involvement of trigeminal nerve nucleus and the quinto-thalamic tract which are located laterally in the medulla. The presence of Horner’s syndrome, dysphagia, and hoarseness suggests involvement of the descending sympathetic fibres and the 10th nerve nucleus, both of which are also laterally located. The spinothalamic tract which carries the pain and temperature sensation from the body is also laterally located. All these features strongly point to the lateral and relatively posterior and inferior location of the lesion.

One can also see in these cases vertical nystagmus instead of a rotatory nystagmus. One can occasionally see a loss of pin prick sensation in the contralateral face as well as body and this is because of involvement of fibres carrying facial sensation which have already crossed over to the other side. Although this syndrome was originally thought to be caused by occlusion of the posterior inferior cerebellar artery, in majority of the cases, the occlusion is in the vertebral artery. Nearly 60% of these cases are caused by thrombosis, in the background of atherosclerosis and nearly 35% are due to embolism.

Multiple simultaneous brain stem infarcts

Infarcts involving multiple brain stem lesions are common. They can be divided into 3 main such syndromes, the first among these, is the “top of the basilar syndrome”, the second is the group of multiple vascular territory syndrome and the third, a “syndrome of brain stem compression from a swollen large cerebellar infarction”. Of all these, the second one is one of the most difficult to diagnose and separate from each other, because in the final syndrome that the physician sees, there is a lot of overlap with the other ones. So it is a mixed syndrome made of several small infarcts in the distribution of more than one artery.

a) The “top of the basilar” syndrome

It is a syndrome that is associated with multiple infarcts in the territory of the basilar artery. One might see a combination of an infarct in the posterior cerebral artery, associated with a thalamic infarct, because an occlusion of the top of the basilar artery may simultaneously affect the posterior cerebral artery, as well as the penetrating arteries that go to the thalamus. If there is a hemianopsia, it suggests the syndrome of top of the basilar occlusion. These patients may have
cortical blindness as well, when they have bilateral posterior cerebral artery occlusion. If they are cortically blind, sometimes patients may have a drowsy look.

“Multiple vascular territory overlapping” syndromes:

Infarcts involving multiple brain stem levels may have overlapping features and are very difficult to diagnose and localise. They may present with coma and quadriplegia. These overlapping syndromes may have combinations of superior cerebellar artery infarct, paramedian midbrain infarcts, lateral medullary infarct.

If one looks at their eyes, one may find vertical or lateral conjugate gaze palsy or hyperconvergence of the eyeballs. One may also see loss of immediate and remote memory, somnolence and akinetic mutism. These symptoms are often due to infarcts in the midbrain and the thalamus and the medial temporal lobe. As told earlier, it may be difficult to localise all these simultaneously occurring brain stem lesions. The main thing in recognizing them is to look for a striking brain stem sign such as nystagmus, because once a clear nystagmus is noticed, it is most likely to be due to a brain stem lesion. In this background, if the patient is comatose or quadriplegic, it indicates multiple level involvement. If the patients are examined carefully there may be some features of lateral medullary syndrome as well which indicates that the vertebral artery may be blocked. So if one sees a combination of superior cerebellar artery syndrome, lateral medullary syndrome, paramedian midbrain territory syndrome, it should immediately suggest multiple level infarcts and also that probably the vertebral artery is occluded. Because what may happen in these vertebral artery occlusions, is that the occluding thrombus may propagate to involve other arteries which are distally located or there may be multiple emboli from the main thrombus in the vertebral artery, which travel further occluding other smaller branches of the vertebral and the basilar artery that supply the brain stem.

Syndrome of “brain stem compression by a swollen cerebellar infarct”

It usually happens in the background of a combination of a posterior inferior cerebellar artery infarct and superior cerebellar artery infarct. A combination of these two infarcts may give rise to severe swelling of the cerebellum. This syndrome typically presents as progressive loss of consciousness with ipsilateral gaze palsy. These patients begin to be drowsy and then develop a gaze palsy. A 6th nerve palsy may also be present. As a matter of fact other than a cerebellar haematoma, this may be the most important cause of a rapid onset ipsilateral gaze palsy, development of long tract signs and deteriorating consciousness. A surgical decompression can be life saving and therefore they must be diagnosed immediately with at least an urgent CT scan of the head.

The key investigation in localising the brain stem vascular syndromes is MRI, which may be followed by MR angiography which helps to localise the lesion or lesions anatomically. A CT scan usually does not see small infarcts, but it is a good device to exclude haemorrhagic strokes.

In summary therefore, what does a clinician need to know to diagnose brain stem infarcts? One needs to know, the brain stem infarcts can be missed easily unless one has a high suspicion index. Once the physician suspects a brain stem stroke, he needs to localise it and to do so, one of the first questions that he needs to ask “does the clinical picture fit into a known syndrome”. If the physician is aware of one of these distinctive brain stem syndromes, he may be able to fit that case into one of them. If he can’t fit into one of the known syndromes immediately, his second important step is to localise the lesion to try to find a rostro caudal level, whether the causative lesion is in the midbrain, pons or the medulla. He does it by looking at the cranial nerve deficits.
The third nerve involvement signifies midbrain location; the 5th, 6th, 7th, and 8th nerve involvement signify the pontine location; and the 9th, 10th, 11th and 12th nerve involvement indicates medullary involvement. The third step is to find an antero-posterior localisation. And to do that, he has to ask a few questions again. Is it pure motor? Is it sensory as well as motor? Is it pure sensory? With the help of these rostro-caudal and antero-posterior level findings, one would know the anatomical location and the probable vascular territory involved. If the lesion is posterior, inferior and lateral it would probably be in the posterior inferior cerebellar artery or the vertebral artery. Once he determines the vascular territory, one should try to know what types of arterial disease the patient may have to lead him to this syndrome. And finally, one will infer what investigations need to be done.

References