An improved clinical method for detecting meningeal irritation

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Abstract

Neck stiffness is the most important sign of meningitis. When the neck is flexed, the inflamed nerve roots and meninges of the cervical region get stretched. This causes protective muscle spasm manifesting as neck stiffness. Kernig's sign represents similar phenomena involving the distal spinal cord and related nerves. A manoeuvre that stretches the neural elements of the whole length of the spinal canal simultaneously will be a more sensitive test for meningeal irritation. Eliciting neck stiffness while the patient sits up with knees extended achieves this. This method is more sensitive, specific, and amenable to objective assessment.

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Beyond early infancy, neck stiffness is the most important sign of meningitis, and meningeal irritation is seldom suspected in its absence.¹ In patients with meningitis, neck stiffness was observed to become more marked in the sitting posture. This study evaluates neck stiffness in different postures with an aim to delineating the ideal method for eliciting neck stiffness and tries to formulate objective criteria for assessing meningeal irritation.

Subjects and methods

Neck stiffness was assessed in postures 1, 2, and 3 (figs 1, 2, and 3 respectively). Neck stiffness is produced by spasm of the extensors of neck. The degree of passive neck flexion possible (angle ABC, fig 4) will be a measure of the muscle spasm and hence that of neck stiffness

Figure 1 Posture 1 with the patient supine and mild neck stiffness. The photograph is of a patient recovering from pyogenic meningitis and was taken on the fifth day of treatment.

Figure 2 Posture 2 with the patient sitting on the edge of the couch with legs hanging down, hips and knees flexed, and absent neck stiffness. The photograph shows the same patient and was taken at the same time.

Figure 3 Posture 3 with the patient sitting up with knees extended and neck stiffness clearly evident. Note that the angle between the plane of the nape of neck and vertical is less than 45°, and that the patient needs support on the knees and back to avoid the tripod posture. Note also the tense muscles on the neck.

Rotate the picture clockwise through 90° to bring its right side down. Viewing the figure in this position, imagine the patient's back resting on a horizontal couch. Now, one can easily visualise his neck and lower limbs remaining in positions equivalent to eliciting neck stiffness and Kernig's sign simultaneously in a supine patient. The photograph shows the same patient and was taken at the same time.

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Figure 4 The angle ABC represents the degree of neck flexion. The line BC running parallel to the nape of neck connects the vertebra prominence and the external occipital protuberance. Line AB passing through the vertebra prominence represents the horizontal plane in posture 1 and vertical plane in posture 2 and 3.

also. For accurate measurement of neck stiffness, one can note the degree of flexion achieved for a given force applied to the head, with the neck fully relaxed. However, the application of sophisticated gadgets to the head to measure the force applied will frighten the child and jeopardise relaxation of the neck. Also, while flexing the neck in the supine posture, the weight of the head (which cannot be measured accurately) will have to be overcome; in the erect posture, this weight acts to favour neck flexion. Thus it is not practicable to measure neck stiffness by purely objective methods.

In patients with meningitis, an initial variable range of neck flexion will be observed to be free of muscle spasm. This is followed by a second phase characterised by a sharp and progressive increase in resistance to flexion. If forcible flexion is continued, the third phase is reached. This is characterised by increasing muscle spasm and pain and will be indicated by the child's facial expression. Also, to avoid pain, the child actively tries to extend the neck, and often overcomes the flexion already attained. With increasing neck stiffness, the muscle spasm characteristic of phase 2 begins earlier, so that phase 1 gets progressively shortened. Angle ABC was measured in patients and controls while keeping the neck passively flexed up to the beginning of phase 2, using an instrument similar to dividers with thin, rigid, straight, and blunt tipped arms. Its joint was placed on the vertebra prominence and one of its arms kept on the nape of the neck, parallel to the imaginary line BC (fig 4). The other arm was adjusted to overlie the imaginary line AB, by keeping it horizontal for posture 1, and vertical for posture 2 and 3. The accuracy of alignment of this arm was checked visually by comparing it with the plane of the couch for horizontal plane, and with a freely hanging straight wire suspended near by for the vertical plane. The instrument was then transferred on to a protractor to read off the angle between the arms. While measuring the angle in posture 1, the chest was not allowed to get lifted, and in postures 2 and 3, the tip of acromion was brought to remain vertically above the highest point of iliac crest. For

uniformity all the measurements were made by JV.

Children aged 2 to 12 years with symptoms suggestive of meningitis, and neck stiffness evident in posture 3, with or without neck stiffness in the other postures as per the subjective assessment of JV, were subjected to lumbar puncture. Those with pleocytosis and/or neutrophils detected in the cerebrospinal fluid were taken up for the study. Thus cases of pyogenic as well as aseptic meningitis were included. Children offering insufficient co-operation for eliciting neck stiffness, those having paralysis of limbs, decerebrate or decorticate posturing, or taking more than two weeks to be cured were excluded. Seventy five cases were studied. There were 22, 34, and 19 cases in the 2 to 5, 5 to 8, and 8 to 12 year age groups, respectively. Angle ABC was measured simultaneously in postures 1, 2, and 3 at the time of admission to hospital, and on subsequent days, whenever a change was appreciable in the degree of neck stiffness.

As a control, 900 children attending the outpatient department were studied for the normal free range of neck flexion possible in posture 3. All were examined while sitting on a firm couch, without tight clothing. They formed two groups with 450 cases in each, and each group had 150 cases in the 2 to 5, 5 to 8, and 8 to 12 year age groups. The first group (afebrile group) included patients who were unlikely to have any restriction of neck flexion because of disease; for this purpose, children with fever during the previous three days (a history or documented axillary temperature of more than 37.2°C), central nervous system disease, evident deformities of spine, and painful conditions in relation to the neck, trunk, or lower limbs were excluded. The second group (febrile group) included children with fever during the preceding 24 hours due to any cause, with or without other problems, but without neck stiffness in posture 3, as judged by the subjective assessment of one of the authors (JV).

Results

Neck stiffness was noted to be more marked in posture 3 than in 1 and 2. While sitting up from supine posture, patients with marked neck stiffness avoided kyphosis of the back, and in extreme cases kept the spine lordotically curved. They used hands on the bed for the tripod supporting posture, and could not sit erect unless the knees were drawn up (Amoss' sign²). While measuring neck flexion in posture 3, these patients were permitted just sufficient flexion of hips and knees, so that they could sit up to bring the tip of acromion vertically above the highest point of the iliac crest.

During recovery neck stiffness disappeared first in posture 1 and 2, but it persisted in posture 3 for another 1–2 days (or more) depending on the speed of recovery. Neck flexion measured simultaneously in the three postures on 178 occasions while the patients had varying degrees of neck stiffness ranged between 17° and 60° in posture 1 with a mean (SD) 42.56 (10.67)°, 24° and 76° in posture 2 with a mean

Figure 5 This photograph shows the same patient one week after stopping treatment for meningitis. As a normal child, his angle of neck flexion in posture 3 is well above 45° . Note the kyphosis of the back; compare this figure with figure 3.

(SD) $61.07 (9.80)^\circ$, and 5° and 50° in posture 3 with a mean (SD) $31.35 (11.55)^\circ$. The difference between the angles of neck flexion in postures 1 and 3 ranged between 4° and 22° , and this difference was highly significant ($p < 0.001$, t test).

Once neck stiffness was fully relieved, angle ABC in posture 3 became more than that in posture 1; this gave the impression that a greater degree of neck flexion would be possible in posture 3 in normals. This is false, and is due to the kyphosis generally noticeable when a patient free of meningeal irritation sits up (fig 5). This kyphosis causes the upper thoracic and consequently the cervical spine also to assume a forward inclination. This inclination, together with neck flexion, accounts for the exaggerated angle ABC in posture 3 in cases without meningeal irritation. In posture 1, as the back rests on the cot, there is little kyphosis and the angle is due to neck flexion alone, and is hence less than that in posture 3.

Neck flexion was most free in posture 2. The difference between the angles of neck flexion in postures 1 and 2 ranged between 6° and 31° , and this was also highly significant ($p < 0.001$, t test).

Among the afebrile control children, neck flexion in posture 3 varied between 50° and 104° with a mean (SD) $78.81 (7.88)^\circ$. Neck flexion in febrile controls ranged between 48° and 97° with a mean (SD) $74.27^\circ (9.66)^\circ$.

Discussion

The genesis of meningeal signs is best explained on the basis of mechanical factors.¹⁻³ The spinal canal being posterior to the vertebral bodies, the former lengthens, and its contents get stretched when the spine is flexed. As the brain stem is relatively immobile the cord gets pulled upwards when stretched by neck flexion. The resultant tension is transmitted along the cauda equina to the femoral and sciatic nerves. The femoral nerve passes anterior to the hip joint and the sciatic posteriorly. Hence, the sciatic gets stretched by hip flexion, and femoral by extension beyond the neutral position of the thigh. The opposite movements relax them. As the downward continuation of the sciatic is along

the back of the knee it gets stretched by extension and relaxed by flexion at the knee. However, the femoral nerve is little disturbed by movements at the knee as its downward course is along the medial aspect of that joint. Both the nerves are maximally relaxed by keeping the hips and knees flexed (posture 2 and the tripod posture with knees drawn up). Here, though hip flexion stretches the sciatic, it gets relaxed by flexion at knee. In the neutral position of the lower limbs (posture 1 and the standing posture) the lower spinal roots also remain relaxed. Flexion of the hip with extension of the knee (straight leg raising and Kernig's sign, and posture 3) stretches the sciatic and lower spinal roots maximally, though the femoral remains relaxed. Manoeuvres that stretch the inflamed neural elements and meninges of the spinal canal induce pain and protective muscle spasm (for example, neck stiffness consequent to passive neck flexion) and lead to postures designed to minimise tension on the inflamed structures²⁻⁴ (for example, on attempting to sit up, the patient adopts the tripod supporting posture with back and neck extended, and hips and knees flexed). These phenomena characterise all meningeal signs.

The augmentation of neck stiffness occurring in posture 3 can be explained as follows: in this posture the lower limbs, which remain flexed at hips and extended at knees, are in a posture equivalent to bilateral elicitation of Lasègue's (straight leg raising) or Kernig's signs. This causes the distal spinal cord and related nerves to remain stretched and tethered down. Sitting with a kyphosis stretches the cord at its middle. If the neck also is flexed now, the cord gets pulled upwards also. This simultaneous upward and downward pull on the cord causes more severe pain and muscle spasm than if the pull were from one end only, in which case the other end could allow for some relaxation. Brudzinski's neck sign substantiates this integration between movements of neck and lower limbs in meningitis. Here, neck flexion causes flexion of hips and knees. The latter is a compensatory movement of the lower limbs to relax the cord stretched by neck flexion.

Testing for neck stiffness in posture 3 is equivalent to eliciting Kernig's sign and neck stiffness simultaneously—with one augmenting the other. Eliciting neck stiffness in other postures pulls the cord upwards only, leaving its lower end free. Textbooks of clinical medicine, paediatrics, and neurology either advise neck stiffness to be elicited in the supine posture,¹⁻⁵⁻⁸ or do not specify the posture.²⁻⁹ Opinion to the contrary, that neck stiffness is better elicited in the sitting posture, has been expressed by Wehrle¹⁰ and by Illingworth¹¹ independently and is based on personal observations (personal communications). The present report is in agreement with this view.

As a test of neck stiffness it has been suggested that the ability to approximate the chin to the chest is to be looked for.¹⁻²⁻⁷⁻⁹ This, however, would not be a satisfactory criterion for assessing neck stiffness. Because of their short neck, children can approximate the chin to the chest by tilting the face downwards

(nodding movement). This movement involves flexion at the atlanto-occipital joint only, and does not cause a true flexion of the whole length of neck. Consequently, significant stretching of the cord does not occur and it can be carried out despite mild meningeal irritation. Only true neck flexion—and not the nodding movement—alters angle ABC. Hence, this angle, which reflects true neck flexion and the resultant stretching of the cord, is a better measure of neck stiffness than the position of chin in relation to the chest.

The angle of neck flexion as obtained in posture 3 is modified by, and is the aggregate effect of flexion of neck, flexion of trunk, and flexion of hips with extension of knees. All these movements together stretch the neural elements of the whole length of the spinal canal. Hence, this angle integrates all the important signs of meningeal irritation (neck stiffness, Kernig's sign, and Amoss' sign) and is a composite, single, and more sensitive index of meningeal irritation. It also forms a criterion suitable for objective assessment of meningeal irritation at the bedside. This method also obviates the need to elicit Amoss', Brudzinski's, and Kernig's signs separately.

For these reasons, neck stiffness due to meningitis, meningism, and disease involving the neural elements of the craniospinal axis should become more marked in posture 3, and neck stiffness due to local painful conditions in the neck would not show this postural variation. Thus this method scores advantageously in the matter of specificity also.

Though one of the authors (JV) could subjectively appreciate neck stiffness on a few occasions in children who could flex the neck beyond 45° and up to 50° in posture 3, for practical bedside purposes meningeal irritation can be assumed to be absent if a child can sit erect with knees extended and then allow passive neck flexion to the extent that the nape of neck forms an angle of more than 45° with vertical. The presence of pain and/or muscle spasm within 45° of flexion is indicative of meningeal irritation. All control children could achieve neck flexion beyond this.

Being more sensitive this method helps to detect pyogenic meningitis earlier. Detection of aseptic meningitis with minimal meningeal irritation is also made possible. Naturally, more cases of meningism will also be picked up. However, giving due consideration to all clinical aspects in evaluating these cases with mild neck stiffness, and observing the evolution of the illness over the ensuing 12–24 hours will help to avoid lumbar puncture on many occasions. In this study, neck stiffness that was evident only in posture 3 led to the suspicion and subsequent confirmation of the diagnosis of meningitis in 18 patients. As this method combines all the important signs of meningeal irritation into one objectively assessable and more sensitive test, the absence of neck stiffness in posture 3 will be strong evidence on firm grounds to exclude meningitis.

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