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Experimental determination of the effect of progressive sharp-angle spinal deformity on the spinal cord

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Abstract Neurological deficit is a serious though not well-known complication associated with spinal deformity. Sharp-angle kyphosis may be congenital, traumatic, degenerative, infectious, or iatrogenic in origin. Many kyphotic deformities are underestimated, thus leading to severe neurological deficit. In order to determine exactly what procedures of angulation the patients should undergo to stabilize the spine, which are major operations, the authors analyzed in an experimental model the effects of progressive sharp angulation on the anatomy of spinal canal and cord. We found that sharp anterior angulation of 50° causes anterior-posterior stenosis and the dura will touch the spinal cord. At 90° of angulation, the spinal cord will be squeezed and the pressure in the canal will be double what it was initially, probably leading to ischemia. The experimental confirmation (determination) of these angulations allows the physician in charge to define early in the treatment program when a surgical stabilization procedure should be included, before the angulation causes any neurological damage.

Key words Spinal diseases · Spinal cord injuries · Spinal cord compression · Kyphosis

Introduction

Progressive deformity of the spine such scoliosis, lordosis, and kyphosis may result in a variety of complications. The effects of the curve on the respiratory function range from a minimal limitation for physical exercises to cor pulmonale and death [3–5, 9, 22, 27, 29]. The discal and facet degeneration may result in chronic back pain [2, 19, 28]. The external appearance may cause severe psychological disturbances [6, 13, 16]. A lesser known complication is the neurological deficit [1, 8, 23, 24, 32]. This is probably related to the fact that this is a rare complication that has not yet been analyzed enough. How and when does the deformity cause neurological deficit? The answer to these questions will allow those dealing with such patients to define the right moment and the technique to use in order to stop development of a spinal cord deficit. An understanding of these factors will certainly influence the treatment of other pathologies originating from spinal deformity, such as infections, tumors, degeneration, and iatrogenic insult, the last more frequent after wide laminectomies. We developed this experimental model in order to elucidate these questions.

Material and methods

A dural sac model was made of fascia lata. The diameter used was 14 mm, corresponding to the diameter of the sixth thoracic vertebra, the most frequent location of spinal deformities associated with neurological deficit [13, 19, 23, 32]. Fascia lata was chosen for its similarity to the dura. We placed a distensible balloon inside this tube, filled it with water, and connected it to an external system allowing verification of pressure during the changing of angulation (Fig. 1). This model underwent sharp angulation up to 110°. We adopted this mark on the basis of the variations referred to in the literature where the maximum kyphotic curve reported is 110° [23, 24, 32]. During the changes in the shape of the tube we determined its anterior-posterior and lateral-lateral diameters and internal pressure.

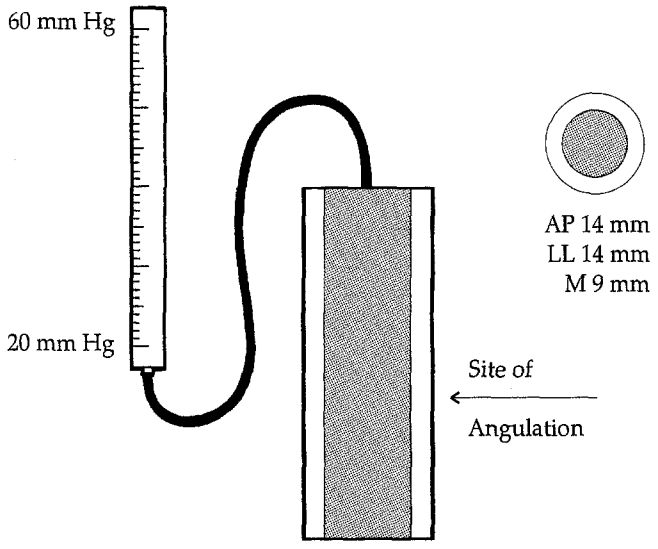

Fig. 1 The experimental model. Anterior-posterior and lateral-lateral diameter, 14 mm; initial pressure inside the balloon, 20 mm Hg; spinal cord diameter, 9 mm

Results

We observed that angulations from 20° to 110° resulted in a progressive reduction of the anterior-posterior diameter and an increase in the lateral-lateral diameter of the dural sac. At an angulation of 50°, the anterior-posterior diameter of the dural sac was equal to the diameter of the spinal cord (9 mm), and from that point onwards the spinal cord was permanently under pressure (Figs. 2, 3).

Discussion

The relation between neurological deficit and acquired deformity of the spine was first evidenced by MacEwen in 1888 [26]. From 1889 through 1990, Lonstein, Winter, and colleagues [23] analyzed 286 cases of spinal deformities associated with neurological deficit, etiologically classified as follows: congenital deformities (39%), neurofibromatosis (16%), idiopathic deformities (14%), tuberculosis (11%), and others (20%). Sixty-eight percent of the patients presented kyphoscoliosis, 17% kyphosis, and 15% scoliosis. The thoracic spine was involved in 81% of the cases, generally at the T6 level, the median angulation of kyphosis was 95° (variation from 50° to 180°), and the median angulation of scoliosis was 60°. Fifty-nine percent of the cases presented paraparesia and 41% paraplegia. Our clinical experience was made up of 10 patients with congenital kyphoscoliosis with associated neurological deficit (50% male patients, with age ranging from 9 to 21 years) treated surgically by the anterior approach at the SARAH Hospital from 1981 to 1990. These data called our attention to the progressive neurological deficit associated with deformity with sharp angulation of the spine and made us ask what would have been the right time for treatment to avoid the progressive neurological deficit.

In cases of sharp kyphosis, the spinal cord is stretched out upon the posterior side of the vertebra [11]. In our model, from 50° of sharp angulation onwards, the spinal cord is subjected to posterior compression from the (not very flexible) dura mater fixed on the proximal side to the skull and on the distal side at the emerging nerves [7]. An incision in the dura mater may reduce the pressure but this is only possible through laminectomy, which has the disadvantage that it weakens the posterior components of the spine and so aggravates kyphosis and scoliosis. In 1967, Maxwell and Khan [25] reported a dramatic case of paraplegia developing after a thoracic laminectomy that intensified the kyphosis. The deficit involved the upper limbs and caused respiratory failure. The patient recovered normal respiratory functions and use of the upper limbs after renewed surgery to cut the spinal cord at the kyphotic level. In this case the effects of traction were not

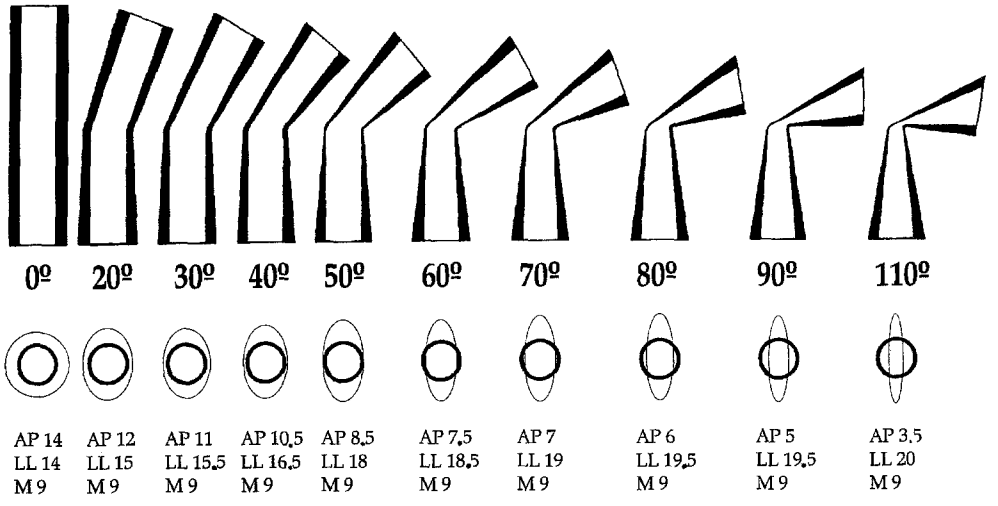

Fig. 2 Synthesis of angulation effects on the model at each stage of angulation. *AP*, antero-posterior diameter; *LL*, lateral-lateral diameter; *M*, spine cord diameter

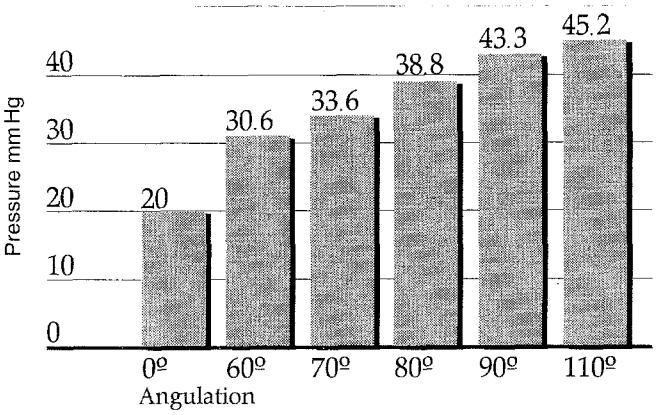

Fig. 3 Relationship of pressure in relation to angulation

only local but also involved the cervical spinal cord and medulla.

As we demonstrated, this combination of angulation, compression, and traction affects the vascularization of both spinal cord and nerves. Dommisse [12] analyzed the vascularization of the thoracic spinal cord. A deformity at this place strikes at the most fragile part of spinal cord vascularization. There are few arteries, and the blood supply is a balance of the flow between the anastomosis of the cervical and lumbar vascularization. During the rapid development during adolescence, the sharp angulation associated with the deformity causes compression on the spinal cord, which explains the appearance of deficit during the second decade of life [23, 32]. According to our findings, the reduction of the dural sac diameter is proportional to the increase in the angle of kyphosis, and at an angulation of 50° the anterior-posterior diameter of the dural sac is equal to the diameter of the spinal cord, which probably produces initial venous compression. The pressure analysis showed that a 60° angulation results in venous stasis and a 10-point increase in pressure. Arteries are subjected to compression and distention. At 90°, the pressure becomes twice as much, possibly causing spinal cord ischemia. This is the reason why patients with over 90° angulation frequently do not recover even after surgical decompression. It must be taken into account that the slow evolution of angulation allows accommodation mechanisms to establish themselves, sometimes permitting amazing recovery processes despite sharp angulation. As demonstrated by Maiman and colleagues [24], patients with late decompression may recover. Certain mechanisms like rotation, traction, and inclination may be associated with the development of the neurological lesion, but because of the complexity of observing them, we did not include them in our model. Traction is a well-studied cause of spinal cord deficit as it is associated with tethered cord syndrome. Rotation and inclination do not seem to be the main causes of deficit as they are present in many cases of scoliosis and do not produce neurological deficit [10].

Early diagnosis and appropriate treatment of the patient with spine deformity can inhibit the appearance or progression of a neurological deficit. The analysis of results of experimental work with an inflated balloon in the epidural space proved that pressure and the duration of compression are important [30]. There is a multiplicity of options for treatment [13–15, 17, 18, 20, 21, 30, 31], and some patients develop deficits that in our opinion could be avoided entirely. Perhaps our study, establishing the sharp angulation of 50° as the beginning of spinal cord compression and 90° as severe spinal cord compression, could contribute to avoiding that kind of delay and underline the necessity of early use of the procedures referred to in the treatment of patients with sharp-angle spinal deformities.

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