Dura-arachnoid lesions produced by 22 gauge Quincke spinal needles during a lumbar puncture

M A Reina, A López, V Badorrey, J A De Andrés, S Martín

Aims: The dural and arachnoid hole caused by lumbar puncture needles is a determining factor in triggering headaches. The aim of this study is to assess the dimensions and morphological features of the dura mater and arachnoids when they are punctured by a 22 gauge Quincke needle having its bevel either in the parallel or in the transverse position.

Methods: Fifty punctures were made with 22 gauge Quincke needles in the dural sac of four fresh cadavers using an “in vitro” model especially designed for this purpose. The punctures were performed by needles with bevels parallel or perpendicular to the spinal axis and studied under scanning electron microscopy.

Results: Thirty five of the 50 punctures done by Quincke needles (19 in the external surface and 16 in the internal) were used for evaluation. When the needle was inserted with its bevel parallel to the axis of the dural sac (17 of 35), the size of the dura-arachnoid lesion was 0.032 mm² in the epidural surface and 0.037 mm² in the subarachnoid surface of the dural sac. When the needle’s bevel was perpendicular to the axis (18 of 35) the measurement of the lesion size was 0.042 mm² for the external surface and 0.033 mm² for the internal. There were no statistical significant differences between these results.

Conclusions: It is believed that the reported lower frequency of postdural puncture headache when the needle is inserted parallel to the cord axis should be explained by some other factors besides the size of the dura-arachnoid injury.
was mounted on three aluminium rods covered with latex (fig 1). The dura mater was kept in a physiological environment by continuously spraying a saline solution to avoid any biological changes. Under these conditions, we did not find any histological changes in the samples. A 22 gauge Quincke needle (Yale Spinal Becton Dickinson needle made in Spain) was used to perform the punctures. Twenty five of fifty punctures were done in a parallel approach to the axis and the other 25 in a perpendicular one. Fifteen minutes after the punctures small dural sac samples were cut for examination and immersed for four hours in 2.5% glutaraldehyde with phosphate solution buffered to a pH of 7.28–7.32. They were then progressively dehydrated by immersion in solutions containing an ever increasing concentration of acetone (up to 100%). The acetone from the samples was exchanged with carbon dioxide in a pressurised chamber (Balzers CPD 030 Critical Point Dryer, Bal Tec AG, Fürstentum, Liechtenstein) until a critical pressure (73.8 bar) and temperature (31°C) were reached. A carbon layer was then put on the samples to obtain a thickness of less that 200 Armstrong with a Balzers MED 010 Mini Deposition System (Balzers, Bal Tec AG, Fürstentum, Liechtenstein). The carbon evaporation was done by passing an electrical current through a graphite electrode within a 10⁻⁵ mill bar vacuum chamber. The samples were then covered with a golden microfilm by passing a 20 amp electrical current through a gold electrode within a vapourisation chamber SCD 004 Balzers Sputter Coater (Balzers, Bal Tec AG, Fürstentum, Liechtenstein) regulated with a vacuum pressure of 0.1 mill bars. The samples were studied in both the outer epidural surface and the inner subarachnoid surface with a JEOL JSM 6400 Scanning Electron Microscope (JEOL Corporation, Tokyo, Japan). The area of lesion of the DAL was calculated according to the photographic pictures taken once they had been amplified up to 600 times. Each lesion was an irregularly shaped geometrical figure. Its area was calculated by dividing the irregular picture into multiple triangles (30–35) and adding them up to get the final area. The total measured error on the surface of each lesion was 2%. The thickness of the dura-arachnoid membrane was measured by using a Mitutoyo micrometer 102-101 N, 0–25 mm, Mitutoyo Corporation, Japan. Six measurements were performed in each of the corpses.

The needle used in all the procedures was the 22 gauge bevelled cutting Quincke needle, as neurologists do not usually use thinner needles when performing a diagnostic lumbar puncture. The data are normally distributed. Values are expressed as means and 95% confidence intervals. Student’s t test was used for statistical analysis and a p value of less than 0.05 was considered significant.

RESULTS

Of 50 punctures done by Quincke needles 35 (19 in the external surface and 16 in the internal surface) were analysed. The remaining 15 were not included because of the lack of quality of the histological preparation. Seventeen punctures were performed with the needle inserted in a parallel direction to the cord axis and 18 in a perpendicular direction. The area of the DAL when the needle bevel was in a parallel direction was 0.042 mm² (95%CI 0.026 to 0.040) for the internal (fig 3B). We found no statistically significant difference between the areas.

The average thickness of the dura-arachnoid membrane was 0.36 mm (95%CI 0.30 to 0.42). The morphology of the DAL caused by Quincke needles had either a “U” shape (figs 2 and 3) or a semi-lunar shape (fig 4A). The lesion generates a flap of dural membrane with a clean cut edge that resembles the lid of a partially opened tin. The needle orientation had no influence on the morphology of the lesion (fig 4A). When the internal surface of the dural sac was studied, we found in some of the samples elastic fibres and broken neurothelial cells around the edge of the DAL (fig 4B). In the partially closed lesions, (like a semi-open tin) we could identify dural laminas inside the thickness of the dural sac. These laminas were almost 5 microns thick.

DISCUSSION

This study has allowed us to confirm the shape and size of the DAL 15 minutes after withdrawing the spinal needle. It is accepted wisdom that the size of the dural puncture and PDPH are directly related. Therefore, the bigger the dural puncture the greater the chance of PDPH. We have shown the morphology of the DAL with a non-damaged needle pointing at different angles. When an “in vitro” puncture is performed, the tip of the needle makes a straight contact with the dural sac. In clinical practice, the needle gets through the skin and spinal ligaments before it reaches the epidural space. It can often hit rigid structures such as the vertebral laminas and the spinous processes causing damage to the needle’s bevel.13–15 Our results showed no statistical differences in the size of the DAL when the needle’s bevel was pointed in directions, parallel and perpendicular to the axis.

This conclusion is not supported by Franksson et al,6 who using an “in vitro model” and the help of a light microscope, stated that fewer fibres were damaged when the needle’s bevel was inserted in a longitudinal way than when it was done transversely. This seminal study established the accepted clinical guidelines: to point the bevel in a parallel direction. Further studies are needed to see if this also occurs in the human spine.
direction to the spinal cord axis results in a smaller dural sac hole and less CSF leakage less than when the bevel is pointed perpendicularly. However, their study cannot be compared with ours as it was undertaken using different needles although they were called the same. A detailed look at Franksson and Gordh’s research7 reveals that the non-disposable bevelled needles named by them as "Quincke", are very different from the disposable "Quincke needles" used nowadays. The DAL caused by the "old Quincke needles" from the forties was of a different morphology. Those needles with no adequate bevel edge warranted larger lesions caused by a tearing action rather than a sectioning action whereas the use of "modern Quincke needles" results in a clean U shaped lesion.

Old “Quincke” needles ended having the bevel completely modified as the result of the continuous use, the crashing into the vertebras, and the sterilisation techniques. Their bevels were blunt rather than cutting. We were able to prove all these features by analysing Quincke needles with scanning electron microscopy (SEM) and observing the bevel surface and its morphological details and by comparing the differences between Quincke needles made by different manufacturers.16

Other investigators have studied the DAL by light microscopy.17 18 SEM allows us to assess the size and morphology of the dural and the arachnoid injury separately as well as verifying the lesion along its thickness and the dural laminas section. The dural sac is made on its 90% outer aspect of dural lamina and on its 10% inner side of neurothelial and arachnoid cells. Its innermost layer is made of arachnoid cells (arachnoid lamina) in close contact with the CSF.19–21 When puncturing the dural sac, Quincke needles cause less “tent effect” and overstretching of the fibres than “pencil point” needles because of their cutting action. The result is a clean, U shaped lesion or flap resembling the lid of an open can (hood mechanism).12 As the needle is withdrawn, the U shaped flap tends to return to its original position with the help of the CSF pressure and the viscoelastic properties of the dura mater.
The development of PDPH is multifactorial. Some of the following alone or in combination may contribute to its appearance: dragging of skin cells, number of failed puncture attempts, tip deformation when hitting bone, use of defective spinal needles, degree of previous cerebral vasodilatation, CSF pressure, transverse and parallel puncture, unexpected movement of the patient during the puncture, patient’s position and degree of flexion of the vertebral spine, previous hydration status, abnormal distribution of cranio-spinal elasticity, substance P concentration, and operator skills.

Nevertheless, we must remain cautious when establishing a cause-effect relation in PDPH. In a meta-analysis looking at the relation between PDPH and different types of needle, of 46 articles 30 were rejected by the authors because of unsatisfactory study design or methodology.

Our study contains a number of limitations. Firstly, it is an “in vitro” study. The samples were not exposed to traction forces in a cephalic and caudal way on performing the punctures. However, in the clinical setting some patients are placed in the hyper flexion position leading to a greater DAL. Our conclusions can only be extrapolated to clinical practice if patients are placed on slight flexion of the vertebral spine and when aspiration of CSF comes from a clean, non-traumatic, and one single attempt puncture.

Secondly, we were unable to assess the role of CSF. In vivo, CSF pressure may affect the time the dural hole remains open. We did not measure pressure gradients across the dura-arachnoid membrane.

We conclude that in isolated dural sac samples, puncturing the dura either perpendicular or parallel to the long axis of the dural sac does not affect the size of the lesion produced by the bevel. Further studies are warranted to confirm or disprove the present clinical practice recommendations concerning the insertion of needles for spinal puncture.

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Authors’ affiliations
M A Reina, A López, Department of Anaesthesiology and Critical Care, Hospital de Móstoles, Madrid, Spain
M A Reina, A López, V Badorrey, Department of Anaesthesiology, Hospital Madrid Montepríncipe, Madrid, Spain
J A De Andrés, Department of Anaesthesiology, Critical Care and Pain Therapy, Hospital General Universitario, Valencia, Spain
S Martín, Department of Anaesthesiology and Critical Care, Hospital Ramón y Cajal, Madrid, Spain

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Primary lateral sclerosis and Pierre Marie

Primary lateral sclerosis (PLS) is an idiopathic non-familial neurodegenerative disorder of upper motor neurons. It presents as a slowly progressive pyramidal tract syndrome, sometimes with marked pseudobulbar symptoms.

The diagnosis is based on exclusion of other causes. Pringle and colleagues' proposed diagnostic criteria in 1992 for PLS: adult onset; negative family history; duration of at least three years (to exclude motor neurone disease (ALS)); and normal blood, CSF, EMG, and MRI results. However, it remains an enigmatic, heterogeneous syndrome of uncertain nosology, for some clinically similar patients have varied patterns of symptom progression and physiology. MRS may show a reduction of N-acetylaspartate/creatinine in the motor cortex. Modern technology can exclude other disorders with an accuracy of about 90%. It is now frequently accepted that PLS is an idiopathic non-familial neurodegenerative disorder composed of different subtypes with the exception of congenital spastic rigidity of the limbs, which he recognised as Little’s disease.

It remains mandatory to exclude: compressive lesions at the foramen magnum and cervical cord, MS, Chiari malformation, and human immunodeficiency virus or human T-lymphotrophic virus type I. In PLS and in ALS morphometry shows small pyramidal cells in the precentral gyrus; and in PLS, quantitative histopathology shows the neuronal degeneration confined to the corticospinal system, without involvement of lower motor neurones. Its place in neurological taxonomy is still unclear.

J M S Pearce

304 Beverley Road, Analby, Hull HU10 7BG, UK; jmspearce@freenet.co.uk

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