Syringomyelia (cavity) is another complication of arachnoiditis, probably arising from the pressure dissociation between the subarachnoid space and the central canal.

In the 1999 Global survey, there were 6 cases of syringomyelia.

Inoue et al. ([i]) looked at 7 patients with syringomyelia associated with adhesive spinal arachnoiditis.

They found thoracic cavities in 5, cervicothoracic in 1 and an extended cavity from C4 to L1 in the remaining patient.

All cases showed cord deformity due to adhesion or displacement due to an associated arachnoid cyst.

Klekamp et al. ([ii]) described foramen magnum arachnoiditis without Chiari malformation as a "rare cause of syringomyelia".

They reported 21 cases, and noted accompanying hydrocephalus in one case, and arachnoid cyst in a further case.

In 1990, Caplan et al ([iii]) proposed that arachnoiditis causes syrinx formation by obliterating spinal blood vessels, thereby causing ischaemia.

Small cystic areas of myelomalacia may form, and these tend to coalesce to form cavities.

Alteration of spinal fluid dynamics due to scar tissue creating spinal block contributes to this process.

This was borne out by an animal study in 1992([iv]), which concluded from the data that "cavitation within the cord would be induced by the ischemia, and hydromyelia would be produced by the pressure dislocation between the spinal subarachnoid space and the central canal."

Williams ([v]) suggested that post-traumatic syringomyelia might be initiated by the cord being pulled open or by "the development of meningeal fibrosis and adhesion of the walls of the cord to the dura".

The subsequent spread of the cavity he ascribed to fluid dynamics within the cavity.

He had previously described these effects ([vi]), which he termed ?suck' which was the mechanism by which the cavity filled with fluid.

This was due to relatively long-standing pressure differences in different parts of the CSF due to arachnoiditis impeding the flow thus allowing pressure dissociation which sucks fluid into the cord in the low pressure area, and to rapid movement within these regions during coughing or sneezing, when the dura is abruptly compressed by distension of the epidural veins, and thus the subarachnoid space is also compressed, so that the fluid is impelled to sudden movement.

Williams called these ?rapid impulsive intra-cord fluid movements' "slosh".

He further remarked that in addition to the fluid movements, other pathological processes might be at work in situations such as arachnoiditis secondary to infection or to intrathecal streptomycin, when the commonest site was in the lower spine.

Cavities develop at the point where the cord is at its widest, (much like a balloon will inflate most readily at a point already partially inflated) so that any enlargement is likely to be a focus.

Milhorat et al. ([vii]) suggested 3 main types of syringomyelia:

1. dilatation of the central canal communicating directly with the fourth ventricle

2. non-communicating dilatation of the central canal below a normal segment of spinal cord

3. extracanalicular syrinx originating within the spinal cord parenchyma without communicating cavities

The third type tends to be found in ?watershed' areas of the cord and is often associated with myelomalacia; they may be produced by stenosis of the central canal.

Recently, a proposed study on syringomyelia sponsored by the National Institute of Neurological Disorders and Stroke (NINDS), has hypothesised that spinal syringomyelia results from obstruction of CSF flow in the subarachnoid space, which affects spinal CSF dynamics because the spinal subarachnoid space accepts the fluid displaced from the intracranial space as the brain expands during cardiac systole.

The reduced CSF compliance and capacity of the theca to dampen CSF pressure waves causes exaggerated waves to be produced with every heartbeat, acting on the spinal cord above the CSF block to drive fluid into the cord.

They also suggest a pre-syringomyelic stage when there is spinal cord oedema and progressive myelopathy. ([viii])

Australian authors Brodbelt et al. ([ix]) recently published the results of their rat study looking at the source and route of fluid flow in post-traumatic syringomyelia, which involved kaolin-induced arachnoiditis.

They reported,

"Fluid from perivascular spaces moves preferentially into extracanalicular syringes and the surrounding parenchyma.

Obstruction to CSF flow and loss of compliance from traumatic arachnoiditis might potentiate fluid flow in the perivascular space."

Non-traumatic syringomyelia

Parker et al. ([x]) conducted a retrospective study of 32 patients treated for syringomyelia associated with non-traumatic arachnoid scarring.18 had extensive scarring, of which 15 were post-meningitis (9 tuberculous, 3 listeria, 3 pyogenic) and 3 were post-subarachnoid haemorrhage.

There were 10 cases of focal arachnoid scarring, associated with spinal surgery in 5 (2 meningiomas, 2 neurinomas,1 thoracic disentomb), epidural anaesthesia in 1, thoracic disc herniation in 1, Pott's disease in 1 and 2 of unknown cause.

A third group had basal arachnoid scarring without hindbrain herniation, being associated with

birth injuries in 4 cases.

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[x] Parker F, Aghakhani N, Tadie M *Neurochirurgie* 1999 Jun; 45 Suppl 1:67-83 [Non-traumatic arachnoiditis and syringomyelia. A series of 32 cases.]