Meet The Researcher

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Appointments:

- **Academic Head:** Dept. of Neurosurgery, Institute of Neurological Sciences, Prince of Wales Hospital
- **Neurosurgeon:** Prince of Wales Hospital and Sydney Children's Hospital
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Education:

- **B.Med.Sc.:** (Distinction) University of Queensland, 1986
- **M.B.,B.S.:** (Honours) University of Queensland, 1987
- **Ph.D.:** University of Adelaide, 1997; Thesis: "Pathophysiology of Syringomyelia"
- **F.R.A.C.S.:** Royal Australasian College of Surgeons, 1997

Selected Publications:

- Brodbelt AR, Stoodley MA, Watling AM, Brown CJ, Jones NR. **"Experimental excitotoxic post-traumatic syringomyelia." Journal of Neurotrauma** (In Press, accepted May, 2003).
- Brodbelt AR, Stoodley MA Watling AM, Tu J, Burke S, Jones NR. **"Altered subarachnoid space compliance and fluid flow in a model of post-traumatic syringomyelia." Spine** (In press, accepted 29 May, 2003).
- Stoodley MA, Jones NR, Yang L, Brown CJ. **"Mechanisms underlying the formation**

New Fluid Flow Model May Shed Light On Post-Traumatic Syrinx

Because Chiari related syringomyelia is more prevalent, it often receives the lion's share of attention. Post-traumatic syringomyelia, however, is all too common after spinal injury and can be very difficult to treat. A syrinx can form months to years after the initial trauma and research shows that fewer than half of these will respond to treatment. In addition, post-traumatic syringomyelia is often accompanied by arachnoiditis, an inflammation of the arachnoid. The combination of the syrinx and arachnoiditis can cause severe, chronic pain.

While there is substantial research into the use of genetic engineering and stem cells to treat theses conditions (especially the pain); at present, these treatment options are but a promise of the future. Current treatments, and even promising new ones, are limited by a lack of fundamental understanding regarding syrinx formation.

A research team from the Prince of Wales Medical Research Institute, in Australia, is working to change this and improve our understanding of how and why syrinxes form after trauma. Dr. Brodbelt, Dr. Stoodley and their colleagues reported some of their results in the June, 2003 issue of the European Spine Journal, in a paper titled, *Fluid flow in an animal model of post-traumatic syringomyelia*.

In the study, the researchers essentially created post-traumatic syringomyelia in 25 rats. Unlike Chiari related syringomyelia, where a syrinx forms in the hollow space of the central canal of the spinal cord, a post-traumatic syrinx will form in the spinal tissue itself (outside of the central canal) and is often accompanied by arachnoiditis. The researchers simulated this by injecting an acid into the spinal cords of the rats - there is often a release of acids at the site of a spinal cord injury in humans - to form a syrinx in the spinal tissue, and by injecting a second substance to inflame the arachnoid. They waited six weeks to allow for syrinx formation and then injected what's known as a tracer substance into the CSF system of the rats.

The tracer substance, by leaving a mark as it distributed throughout the CSF system, allowed the researchers to analyze how CSF flows in a spinal system with a syrinx. The team examined the rats at either 0, 3, 5, 10, or 20 minutes following the tracer injection.

What they found was that at each point of time, there was evidence that the tracer had reached the syrinx, so the question then was how did it get there? In more than 80% of the rats, there was evidence that the tracer was entering the spinal cord itself through the perivascular spaces around the main spinal arteries. In other words, CSF, and the tracer, was flowing into the spinal cord in the space around the arteries that supply the spinal tissue with blood. It is important to note that the CSF and blood don't actually mix; the arteries essentially create tunnels throughout the spinal cord, and the CSF flows along the outside of these tunnels. Not only was the CSF entering the spinal cord through these spaces, but there was evidence that this was the preferred route of fluid flow. While it was not proven in the study, the team believes it may be the artery itself that provides the energy necessary to push the fluid into the spinal cord along this route.

What are the treatment implications of these findings? Dr. Brodbelt notes, "If you could block or reduce the amount of fluid going into the spinal cord and syrinx, then you could stop syrinxes forming. A syrinx is a dynamic structure, with a constant circulation of fluid in and out."

Although the research shows how fluid may enter, and expand, a post-traumatic syrinx, it does not address why a syrinx forms initially. One theory proposes that bruising at the time of injury may create an initial cavity which is then filled and expands. However, there is no evidence to support this proposition and for now syrinx formation remains an unknown.

Luckily, Dr. Brodbelt and Dr. Stoodley continue their work into the biomechanics of syrinxes and the development of treatment options. "It is important to keep research progressing in this area," notes Dr. Brodbelt. "If we can find the cause, route, and driving force, then maybe we can stop syrinxes from happening in the first place, or improve treatment for when a syrinx does occur.

Editor's Note: Dr. Brodbelt completed his Ph.D. in Post-traumatic Syringomyelia and is now a neurosurgical trainee in the United Kingdom.

and enlargement of noncommunicating syringomyelia: experimental studies". Neurosurgical Focus 8(3):Article 2, 1-7, 2000.

- Stoodley MA, Gutschmidt B, Jones NR. **"Cerebrospinal fluid flow in an animal model of non-communicating syringomyelia."** Neurosurgery 44: 1065-1076, 1999.