Grant Winner, Discusses His Research

November 20, 2006 -- This month, we took the opportunity to talk with one of the Research Grant winners to learn more about his past work and his research goals moving forward.

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Dr. Marcus Stoodley is an Associate Professor of Neurosurgery at the University of New South Wales in Australia. He has researched and published extensively on syringomyelia and serves on the Editorial Board of Pediatric Neurosurgery. We put Dr. Stoodley In The Spotlight...

Q: You have studied syringomyelia for quite some time, why did you choose to focus on it?

I wanted to do research that made an original contribution to neurosurgical knowledge. Syringomyelia stood out as one of the most enigmatic conditions we encounter, and the results of treatment are not always satisfactory. There were lots of "armchair theories" on what caused the cysts, but very little scientific research. My desire to understand the cause of syrinxes has only increased with time.

Q: A lot of your work has been on Post-Traumatic SM. Is PTS fundamentally different than Chiari related SM, or are they similar?

I view syringomyelia as a secondary event occurring in a wide array of conditions. Post-traumatic and Chiari-related are the commonest types; other causes are tumors, infections, arachnoiditis, and some have no detectable underlying cause. Although there are similarities in that any type of syrinx can cause pain, numbness, and weakness, the underlying mechanisms of cyst formation in each type are likely to be different. The cysts form in slightly different parts of the spinal cord in each type. One focus of my group's research is to determine whether the source of fluid in each type is similar. If we can understand the mechanism of cyst formation in each type, this will hopefully allow us to develop appropriate treatments.

Q: From a clinical point of view, how successful are treatments for PTS?

This is one of the most difficult types of syrinx to treat. In general, treatment is aimed at preventing further loss of function, rather than trying to restore function. Short term results are often good, but in the long term up to 50% of patients will continue to lose function. An ideal treatment would be one that not only prevented loss of function, but also restored function.

Q: In the Chiari world, Oldfield's Piston Theory has gained some popularity, what do you think of this theory?

This theory has considerable merit. We know that Chiari malformations do cause syrinxes, often some distance from the Chiari itself. A change in pressure or compliance in the subarachnoid space is likely to be a part of the link, and pressure transmission by the piston effect is a good explanation of this. However, I don't think this is the total explanation. For example, the theory does not explain how fluid enters the spinal cord: is it diffuse flow through the cord surface or are there specific pathways? The theory also doesn't explain syrinxes in association with other conditions, such as spinal trauma.

Q: Do you think there are multiple mechanisms at play in syrinx formation? If so, what are some possibilities?

Definitely! There is likely to be a somewhat different mechanism for each of the associated conditions. In each situation, there may be different causes for cyst initiation and cyst enlargement. After trauma, for example, cysts may be initiated by death of tissue from the direct trauma or hemorrhage into the cord. For a syrinx to enlarge, there must be an imbalance between fluid inflow and fluid outflow. Possible sources of increased fluid inflow include:

- a) increased pressure in the subarachnoid space forcing fluid into the spaces around blood vessels entering the cord;
- b) scarring around the cord from infection or trauma blocking the normal flow of fluid in the subarachnoid space;

c) fluid leaking from damaged blood vessels inside the spinal cord

Q: With this grant, what will you be doing and what do you hope to accomplish?

We have two main questions that we are addressing in this project. The first is to investigate whether there is any contribution to cyst fluid from leaky blood vessels in the spinal cord. Normally the blood vessels in the brain and spinal cord have a special lining that prevents fluid crossing them. It is known that after trauma this lining is damaged, allowing fluid to leak out. Normally the lining repairs itself, but it is possible that in some cases this does not occur, allowing fluid to continue leaking out of the vessels. It is even possible that as syrinxes enlarge, they cause further damage to the blood vessels, allowing even more fluid to leak out. These questions have never been addressed before. Our second line of investigation is to investigate how the pressure changes from a Chiari malformation might increase fluid flow into the spinal cord. We will be establishing computer models to simulate the pressure changes in the subarachnoid space and how these changes impact on fluid flow in the tiny spaces around blood vessels entering the spinal cord.

Q: How will this work impact patients?

The results from this project will improve our understanding of how syrinxes form. I doubt that there will be any significant advances in

treatment or prevention until we have a good understanding of the mechanisms underlying syrinx formation.

Q: What are the next logical steps in this line of research?

There are further questions to be answered regarding syrinx and Chiari pathophysiology, such as the anatomical pathways of fluid outflow from syrinxes and whether any obstruction of this flow contributes to syrinx enlargement. What is even more exciting though is that as we build an understanding of syrinx mechanisms, it will be possible to look at the effects of treatment. For example, if we show that fluid leaking out of blood vessels contributes to syrinx formation, the logical step is to investigate the effects of preventing blood vessel damage or improving the repair mechanisms. I'd also like to look at the effects of altering subarachnoid compliance on fluid flow into the cord: this is particularly important for those patients who do not respond to standard Chiari decompression surgery.

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