A Pioneer of the Modern Microvascular Decompression Surgery: Peter Jannetta

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ROLLING MEADOWS, Ill. (Aug. 2, 2017) — The condition of trigeminal neuralgia (TN) has been recognized for many years. Prior to 1960, the treatment consisted of either medical therapy or deliberate injury to the trigeminal nerve. The nerve was sectioned and injected with absolute alcohol or irradiated to ablate the nerve. The object of these treatments was to trade the patient’s pain with numbness. Several serendipitous events came together in 1966 that led Peter J. Jannetta, MD, FAANS(L), to develop his understanding of neurovascular compression (NVC) disorders. While many of his findings were previously noted by others, his discovery and concept development were his own, as was his singular success in bringing Microvascular Decompression (MVD) into the mainstream of modern neurosurgical practice.

Jannetta was a resident of neurosurgery at UCLA in 1965, and one of his roles was to provide anatomy instruction to dental students. In the process of preparing prosected cadaver specimens of the cranial nerves, he noted the fine accessory sensory branches of the trigeminal nerve. This led him to suggest treatment of trigeminal nerve (TN) by sectioning of the portio major with preservation of the motor fibers of portio minor as well as the accessory fibers, thereby avoiding complete facial numbness.

Together with his mentor, Dr. Rand, they tested a sub-temporal trans-tentorial approach to the trigeminal nerve root entry zone (TNR) and trigeminal transection. With the recent introduction of the operating microscope, this surgery was hoped to provide more accurate sectioning and a lower risk of surgical morbidity. Their first surgery was not conducted at UCLA, as Jannetta and Rand were forbidden to undertake such a rash new procedure there. Instead, the patient was under the care of John Alksne, Chief of Neurosurgery at Harbour General Hospital (HGH). The patient was a 52-year-old man who had been admitted with an acute flair-up of progressive right-sided TN, despite temporary relief from alcohol injections and medication over the preceding years. Consent for a selective sensory rhizotomy was obtained and performed by Jannetta, with Alksne assisting and Rand observing. For this procedure, the operating microscope, which was personally purchased by Rand, was driven from UCLA in Jannetta’s 1957 Ford station wagon and reassembled at HGH for the surgery. Through the transtentorial approach, Jannetta visualized the trigeminal nerve root and noted, ‘The superior cerebellar artery was seen to pulsate into and compress the nerve. I said, ‘That’s the cause of the trigeminal neuralgia.’ Alksne also understood immediately. Although they proceeded with the planned selective sensory rhizotomy, Jannetta quickly became convinced he had observed the cause of TN and recognized a potential to cure for the disease.

The use of the microscope provided excellent visualization not previously appreciated. With its use, Jannetta observed in four consecutive patients the presence of the superior cerebellar artery (SCA) compressing the trigeminal nerve at its entry into the brainstem. He had not seen a similar compression in his cadaveric dissections, leading him to hypothesize that the pulsatile compression on the TNR may be the cause of trigeminal neuralgia. This had, of course, been proposed by prior generations of neurosurgeons, including Dandy and Gardner, although this was unknown at the time to the young neurosurgical trainee.

Jannetta’s first MVD, however, was not for treatment of TN, but rather another cranial nerve hyperactivity disorder. He would tell the story of seeing a patient approaching down the hall towards his clinic. The man suffered from uncontrolled episodic spasms of the left face and Jannetta had the epiphany that the man suffered from the same hyperactivity disorder underlying TN, but affecting the facial nerve instead. Convinced already of the vascular compression etiology of TN, he was sure a similar culprit would be at the source of this patient’s affliction. The patient consented to what would become
the first MVD surgery, later called the Jannetta procedure. The operation was performed on June 2, 1966, at UCLA when the senior faculty members who had forbidden such surgery were away. The supervising neurosurgeon, Paul Crandall, agreed to Jannetta doing the procedure. A retromastoid approach in sitting position was employed as well as use of the operating microscope. Jannetta found an artery coursing around the distal facial nerve but was not convinced this was the culprit cause of the hemifacial spasms (HFS). He explored further and found a vein running across the facial nerve root entry at the pons. He divided and coagulated the vein and the facial nerve moved slightly, released from the vascular compression. The patient recovered well, and the HFS ceased the following day not to return. Thirty years later, when giving a presentation at the University of Calgary, Jannetta recounted the procedure and happy follow up of the patient whom he had spoken to the prior week. One can only wonder when or if another neurosurgeon would ever venture to test the NVC theory, and what the future of this surgery would have been if the first operation had been unsuccessful. It was rare that the HFS in that first case was due to a vein, although a great testament to Jannetta’s insight and skill to recognize the presumed culprit and safely alleviate the compression.

Months later, August 8, 1966, a vascular decompression surgery for TN was performed at UCLA hospital for a private patient of Rand’s. In Mark Shelton’s book, Working in a Very Small Place: The Making of a Neurosurgeon, Jannetta is described to have performed the surgery with Rand assisting while Rand’s account suggests the opposite. While accounts of that case may be disputed, there is no doubt Jannetta further pursued his NVC concept and pioneered the Jannetta MVD procedure as now performed worldwide.

Jannetta would endure decades of nay-sayers and critics determined to discredit the new “cure” of the old disease. He was not the first to describe the occurrence of NVC in TN or HFS, nor hypothesize these were the cause of TN, HFS and other cranial nerve disorders. He was, however, able to make these observations and develop his procedure independently. He persevered through attacks upon his character and integrity that sometimes became personal, but came to accept these challenges in his role as innovator and educator. Eventually, the body of surgical experience and clinical follow up presented by Jannetta convinced a critical mass of adopters that MVD was an effective treatment, and surgeons worldwide began to replicate his good results.

Patients from around the world came seeking consultation with Jannetta. He treated them all with great compassion: the wealthy, well connected and working-class were extended his full attention and respect. His charisma was legendary, and he enjoyed the attention of his fans and gratified patients. This was, however, always balanced with a humility and earnest desire to help. To every patient that came under his care he promised to do his best, and that was usually the happy beginning of the rest of their lives. Even those unfortunate few who were not cured by an MVD or not even candidates for the surgery, Jannetta connected with them and they realized his desire to help. He always promised to those he had no treatment to offer that if a new approach or intervention was developed he would contact them and he maintained a list of those with “facial pain of obscure origin” in hopes of someday having a solution.

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