Microvascular Decompression for Control of Essential Hypertension

ARTICLE BY TERI JUNGE, CST/CFA

Peter Jannetta, MD, chairman of the Department of Neurosurgery at the University of Pittsburgh, is a pioneer in the use of microvascular decompression in the treatment of neurogenic hypertension. Through research conducted in 1973, following two disastrous incidents involving patients undergoing microvascular decompression for other reasons, Dr Jannetta and associates discovered that a hyperactive autonomic dysfunction involving both an artery in the medulla oblongata of the brain stem and the left vagus nerve may be a factor in causing neurogenic hypertension.[1] This phenomenon more commonly affects the vascular and neural structures on the left side of the body, the reason for which is unknown.[2]

History

To test his theory, Dr Jannetta and his associate, Dr Ricardo Segal, conducted an animal study in which baboons were used. In order to manifest the characteristics of essential hypertension in a baboon, a double-balloon catheter was inserted into the animal to simulate arterial pressure on the ninth (glossopharyngeal) and tenth (vagus) cranial nerves (Figure 1).[3] One of the inflated balloons was inserted into the aorta and the other into the cephalic subarachnoid space, tunneling the remainder of the catheter subcutaneously. The pulsing of the aorta against the inflated balloon caused a transference of pulsatile compression to the ninth and tenth cranial nerves, raising the animal's blood pressure as expected. Several days after the balloons were deflated, the baboon's blood pressure returned to normal. These events supported Dr Jannetta's theory that essential hypertension may have a neurovascular etiology.[4]

Although other causes of idiopathic hypertension may exist, the medical community is beginning to recognize the benefit of microvascular decompression in treating this disease. Furthermore, the research performed by Dr Jannetta and his colleagues may lead to the development of more effective drugs for treating hypertension; this may take the form of pharmacotherapy designed to reduce abnormal nerve transmissions, resulting in lowered blood pressure.[5]

The next step in Dr Jannetta's research was to identify a relatively inert substance that when implanted would serve to cushion and protect the nerve from the pulsations of the artery. Initially, several products were evaluated, including polytetraflouroethylene (Teflon). Already being used successfully in plastic, cardiac, vascular, and neurosurgery since the 1960s, Teflon was tested for this application in a cooperative study that involved the Department of Neurosurgery of the College of Medicine and Medical Sciences at King Faisal University, Dammam, Saudi Arabia; and the Department of Neurobiology, Anatomy, and Cell Science and the Department of Neurosurgery of the University of Pittsburgh School of Medicine in Pittsburgh, Pennsylvania. The study found that Teflon serves well as a permanent implant material in neurosurgery because it is relatively inert and nonadhesive to neural cells, physically stable inside the body, nontoxic, nonstimulating, noncarcinogenic, and compatible with surrounding tissue. For these reasons, Teflon is a suitable material for use in microvascular decompression procedures.[6]

Anatomy and Physiology

There exist twelve pairs of cranial nerves, which are identified by name or Roman numeral (Figure 2). Since this article pertains specifically to the ninth and tenth cranial nerves, following is a brief description of their location and function.

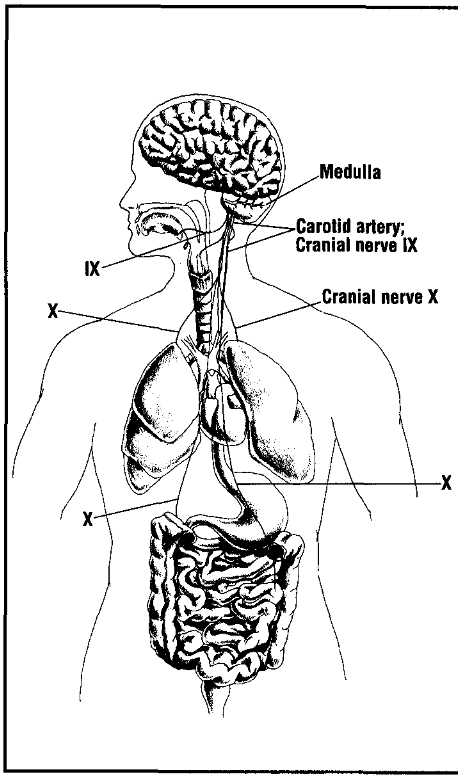

Figure 1. Location of cranial nerves IX and X.

The glossopharyngeal (IX) nerve originates in the medulla oblongata and is responsible for the sense of taste in the posterior one-third of the tongue, innervation of the tonsils and the pharyngeal region, including the carotid sinus, and partial innervation of the pharyngeal muscles (see Figure 1).[7] Its sensory function also includes blood pressure regulation.[8]

The vagus (X) nerve also originates in the medulla oblongata; its many functions include innervation of the pharyngeal and laryngeal muscles, secretion of digestive fluids, and most important for our purposes, providing parasympa-

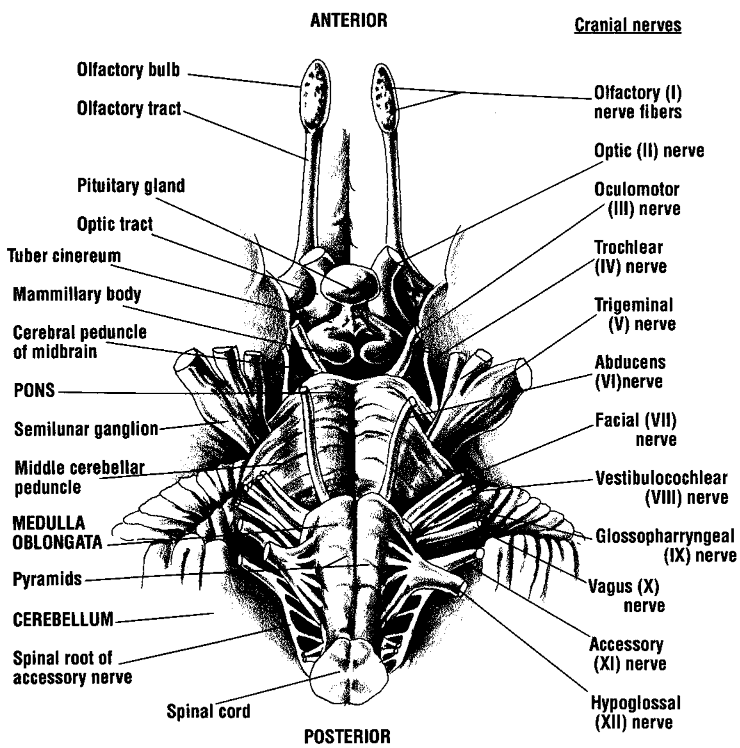

Figure 2. Location of the cranial nerves.

thetic stimulation that affects heart rate (see Figure 1).[7,8]

More than 61 million Americans suffer from hypertension.[5] Of these, approximately 10% have an identifiable cause for their elevated blood pressure.[3] In persons under age 50, normal blood pressure is less than 140/90 mm Hg, or less than 150/95 mm Hg in persons older than age 50.[9]

Hypertension is defined as an intermittent or sustained elevation of the systolic or diastolic arterial blood pressure. The two major types of hypertension are essential, or primary, hypertension (also termed neurogenic hypertension by Dr Jannetta), which is the most common type and has no discernable cause; and secondary hypertension, which is a manifestation of other problems that can be identified.[9]

Formerly, it was believed that such characteristics as family history, obesity, smoking, stress, poor diet, advanced age, and sedentary lifestyle were significant contributing factors in essential hypertension.[9] However, the theory espoused by Dr Jannetta has shed new light on a possible physiologic cause for this mysterious disease.

Traditional treatments for hypertension include modifications in diet (reduction of fat and sodium intake), lifestyle changes (exercise, weight loss, and stress reduction), and drug therapy, usually beginning with a diuretic and adding beta-blockers and vasodilators as needed.[9] Fortunately, microvascular decompression may now be regarded as one of the established protocols for treating this debilitating disease.

Among the diverse causes of secondary hypertension are renal vascular disease, metabolic imbalances, pregnancy, and neurologic disorders. Specific treatments are associated with each of these disorders and will not be discussed in this article.[9]

Uncontrolled hypertension can lead to severe complications, such as stroke, heart attack, blindness, and renal failure.[9]

Patient Selection/Indications

When hypertension can not be controlled by means of medication or through lifestyle improvements, microvascular decompression may be indicated. According to Dr Jannetta, clinicians may overlook a neurogenic etiology in essential hypertension, failing to diagnose quickly enough those patients in whom this type of hypertension exists before a critical stage in the disease has been reached. Such patients achieve the greatest benefit when the processes of diagnosis and treatment are not delayed. Certainly, not all patients diagnosed with hypertension are candidates for surgical treatment, and the criteria for selecting eligible patients are stringent. "An appropriate candidate [for microvascular decompression] is someone who has been through the gamut of medicine and whose hypertension is still uncontrollable," states Dr Jannetta.[5]

Such diagnostic tests as computed tomography (CT) scan, magnetic resonance imaging (MRI) scan, and angiogram are useful in determining if microvascular compression is a factor in uncontrolled hypertension.[10]

Operating Room Setup

The instruments, equipment, and supplies (Table 1, p12, and Figures 4-6, p13) are gathered, and the equipment in the operating room is arranged so that optimal positioning of the patient can be achieved (Figure 3, p12). All equipment, particularly the microscope, camera, and monitor, are pretested to ensure their functional integrity.

Patient Preparation

After the patient has given his/her informed consent to undergo surgery, and the preoperative interview and teaching have been done, he/she is brought into the operating room. Preoperative medication may be used that reduces patient anxiety and lowers blood pressure. The patient is temporarily placed in the supine position on the operating table (padded with an egg-crate mattress for extra comfort) to facilitate placement of the monitoring devices, both invasive and noninvasive, and the administration of general endotracheal anesthetic. The TED hosiery, pneumatic sequential compression stockings, grounding pad, and foam elbow and heel pads are applied; a Foley catheter is inserted, and the Mayfield head holder is applied.

The patient is now ready for reposi-

Table 1. Supply List

Items for Positioning:
- Egg-crate mattress
- Foam elbow and heel pads
- Axillary roll
- Mayfield head holder with attachments and skull pins
- Pillows
- 3-in. adhesive tape

Equipment:
- Hair clippers and razors
- Headlight
- Operative microscope with video camera and monitor
- External thermal blanket with machine
- Electrosurgery unit for monopolar and bipolar applications
- Pneumatic sequential compression stockings, machine, and TED hose
- Foot pedal for drill
- Extension cords
- Patient's X-ray films, CT scan, MRI scan, and angiograms

Instruments and Supplies:
- Foley catheter with urimeter and insertion kit
- Prep tray
- Craniotomy pack
- 1010 Steri-drape
- 1050 Steri-drape
- Two utility drapes
- Three gowns
- Basin set
- Towels
- Craniotomy set
- Neuro micro set (Figures 4 and 5)
- Raney rongeur
- Greenburg retractor
- Bipolar cord
- Cautery pencil
- Peanuts
- Bone wax
- Weck clips - small (yellow)
- Weck clips - medium (blue)
- Gelfoam
- Avitene sheets
- Surgicel
- Microscope drape
- 14-g angiocath
- 20-cc syringe
- 7-mm Jackson-Pratt drain with reservoir
- Anspach neuro drill (Figure 6)
- Drill bit - silver (No. 32)
- Drill bit - green (No. 1)

Suture and Needles:
- Dura:
  - 4-0 silk
  - 4-0 silk 18-in. strands
  - French-eye needles
- Bone flap:
  - 0 silk 18-in. strands
  - No. 5 Mayo taper needle
- Closure:
  - 3-0 polyglactin pop-offs
  - 4-0 nylon
- Drain:
  - 2-0 silk
- Dressings:
  - Cotton balls
  - Betadine ointment
  - Adaptic
  - 4 x 4's
  - Abdominal pads
  - Two Kerlix rolls
  - 1-in. paper tape

Medications:
- 1-mg adrenalin diluted in 150 cc normal saline for local injection prior to incision
- Bacitracin 100,000 units in 1,000 cc of normal saline for irrigation
- Topical thrombin 10,000 units diluted as directed for soaking Gelfoam and cottonoids

tioning in the lateral position. Mindful of good body mechanics and alignment, at least four members of the operating room team turn the patient onto his/her right side. The table is turned 90 degrees with the patient's face turned toward the anesthesiologist. Great care is taken to avoid dislodging the endotracheal tube or any of the lines and monitoring devices. An axillary roll is placed in the right axilla to reduce pressure on the arm. The arms are placed on a double armboard with a pillow supporting the upper arm and secured with Velcro arm straps. The head is then positioned with attention to keeping the neck and spine in proper alignment. The shoulders and hips are secured with adhesive tape as necessary. The lower leg is flexed at the knee and hip, and the upper leg is kept straight. Pillows are placed between the legs and secured with the table strap. A lower-body thermal blanket is applied at this time.[7]

The patient's hair is clipped and the scalp is shaved, and a 10-minute chemical/mechanical prep is carried out. Draping is done according to hospital policy. The equipment is moved into position and the cautery, suction, bipolar cautery, and neuro drill are connect-

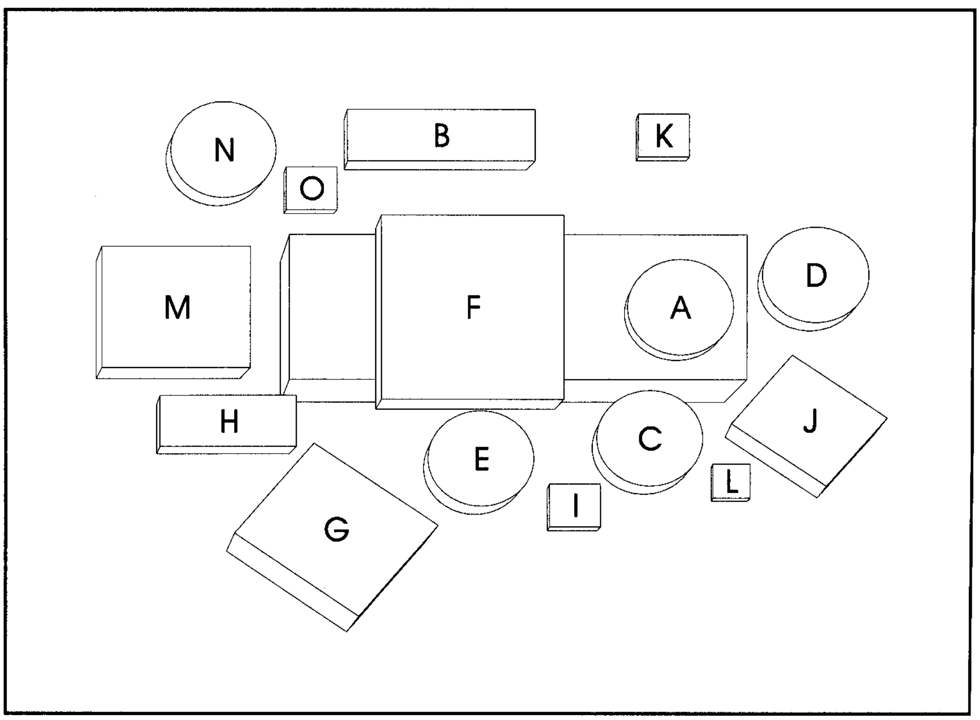

Figure 3. Operating room setup. A, Patient (positioned lying on right side facing anesthesia); B, Anesthesia; C, Surgeon: D, Assistant; E, Surgical technologist; F, Mayfield table; G, Sterile instrument table; H, Double ring stand; I, Small table for drill; J, Microscope base with camera; K, Video monitor; L, Headlight source; M, Electrosurgery unit; N, Suction units; O, External thermal unit.

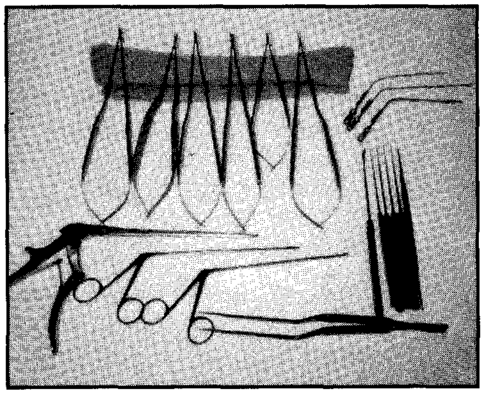

Figure 4. Neuro microinstruments.

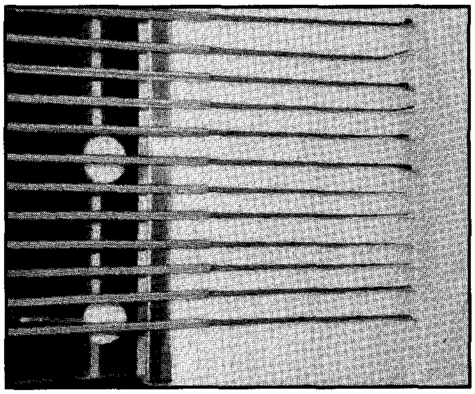

Figure 5. Neuro microinstruments.

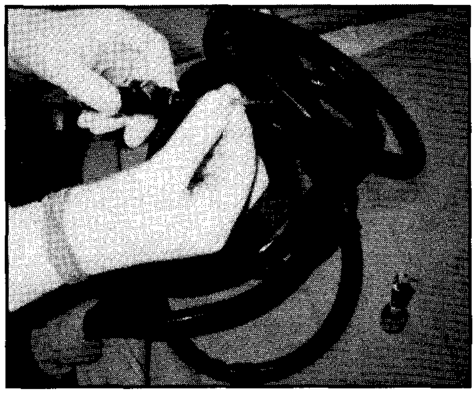

Figure 6. Assembly of power equipment.

ed, activated, and pretested. A marking pen is used to outline the anticipated incision line, and the adrenalin solution is injected along the incision line to cause vasoconstriction, thus reducing blood loss during incision.

Operative Procedure

The retromastoid craniotomy is initiated with a vertical incision made behind the mastoid process. Raney clips are applied and cautery is used as necessary to minimize bleeding (Figure 7). A self-retaining retractor may be inserted. The incision is carried down to the periosteum where a periosteal elevator is used to clean the bone. A No. 32 silver Anspach burr is used to make a burr hole (Figure 8). It may be necessary to use bone wax to control any osseous bleeding. The dura is gently removed from the edges of the burr hole with a Penfield elevator or dural separator to prevent tearing as the burr hole is enlarged. Such enlargement is achieved with a No. 1 green Anspach bit or a rongeur, and the bone edges are smoothed (Figure 9).

The exposed dura mater is gently lifted with a dural hook and incised with a No. 11 blade. The dural incision can be enlarged using a grooved director and the blade, or it may be held with Gerald teethed forceps and cut with small scissors (Figure 10, p14). Cautionary measures are taken to control any bleeding: bipolar cautery, Gelfoam soaked in topical thrombin, or Surgicel may be used for this purpose. Traction sutures of 4-0 silk loaded on French-eye needles may be used to help retract the dura. Cottonoids may be placed at the wound edges to keep the field moist.

At this point, the microscope is dressed and positioned for use (Figure 11, p14). It is important to ensure the surgeon's comfort and help prevent his/her fatigue. The surgeon may be seated at this time, and portions of the Greenburg retractor may be assembled for use as a handrest by the surgeon.

Using brain retractors over cottonoids, the cerebellum is gently elevated upward and toward the midline (Figure 12, p14). The ninth and tenth cranial nerves are consecutively identified and defined with bayonet forceps, micro nerve hooks, and fine dissectors. The offending arteries are dissected in the same fashion. Small pieces of Teflon are inserted as necessary to cushion the nerve from the pulsation of the artery.

The wound is then irrigated with the antibiotic solution and a final check for hemostasis is performed. The microscope is removed from the operative field and wound closure is begun. While the dura is being closed with 4-0 silk, the first closure count is taken. A drain is inserted and previously removed bone is replaced using 0 silk loaded on No. 5 Mayo taper needles that are threaded through drill holes in the cranium and bone flap. In the absence of a bone piece, a large defect can be filled in with titanium mesh or Silastic buttons. The fascia and skin are closed, a final count is taken, and the dressing is applied.[7]

The patient is taken to the postanesthesia care unit and will probably spend one night in the intensive care unit. The patient's neurologic status and vital signs are monitored closely. In approximately 1 week, the patient's blood pres-

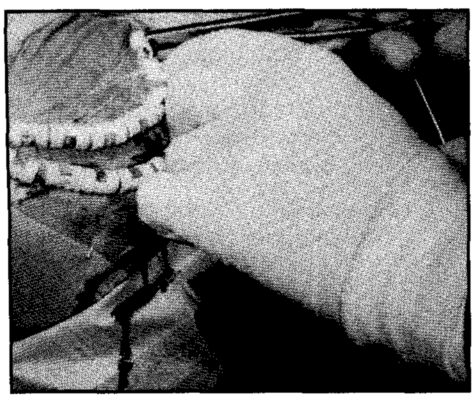

Figure 7. Application of Raney clips immediately following incision.

Figure 8. View of primary burr hole.

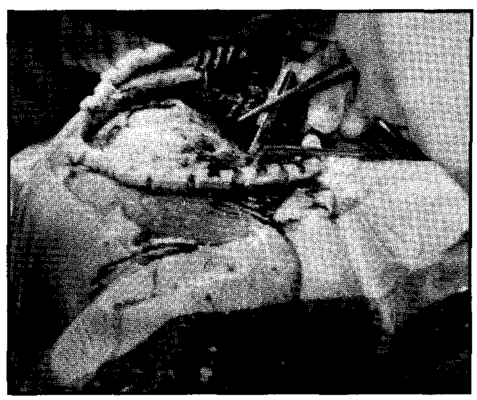

Figure 9. Bone flap enlargement with power equipment.

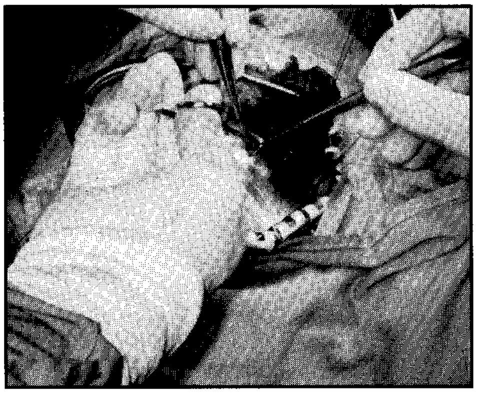

Figure 10. Incising the dura maters.

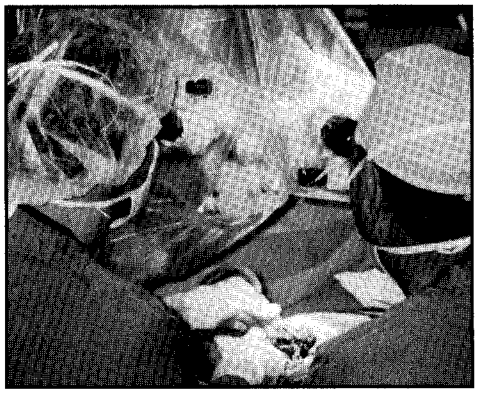

Figure 11. The microscope is positioned for use.

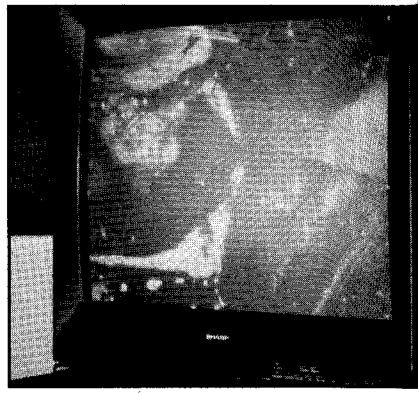

Figure 12. View of brain with dura retracted.

sure is expected to stabilize within normal limits. Prescription of postoperative hypertension medication is based on factors elicited during the patient's convalescence. Normal activity can be resumed within 4 to 6 weeks after surgery.[5]

As with any procedure, there are potentially fatal risk factors involved with the anesthetic and the operation. The possibility of excessive blood loss and permanent nerve damage exist. These risks are minimized by the use of the operative microscope and fine instruments.

Conclusion

Microvascular decompression is not yet widely used, but is gaining recognition as a viable treatment method for qualified patients diagnosed with essential hypertension. In fact, Dr Jannetta has shown that with careful patient selection, nearly 86% of hypertensive patients treated with microvascular decompression achieve improvement.[11]

Author's Note

Believe it or not, the idea for this article began with a story in the National Enquirer. [11] A friend mailed me a copy of the story in hopes that a mutual friend who suffers from essential (or neurogenic) hypertension could benefit from the surgical treatment described as "microvascular decompression" by Dr Jannetta.

To begin my research, I contacted Dr Jannetta's office. Then, with the help of a medical librarian, a computer search of the medical literature using Medline was done. Both of these sources found titles of several articles pertaining to the subject, which were gathered from our medical library or requested from other libraries in the region.

With the information from the research and after consulting with a neurosurgeon in our area, it was determined that my friend could not be helped by microvascular decompression, although the procedure is valid and has a very high success rate. This article is the result of what I learned while doing this research.

Acknowledgements

I wish to express my gratitude to Barbara Bateman, director of perioperative services, and Amanda Rathbun, medical librarian, Palomar Medical Center, for their support with this project, and to Sharon C. Sell, RN, University of Pittsburgh School of Medicine, Department of Neurological Surgery, for providing valuable information.

Thanks also to my family and all of the Palomar Medical Center surgery department staff who put up with me and assisted with photography during this effort, especially Marquelle Hudson for assisting with the illustrations used in the article and the physicians who patiently answered all of my questions regarding this project. Δ

References

1. Jannetta P, Segal R, Wolfson S. Neurogenic hypertension: Etiology and surgical treatment I. Observations in 53 patients. *Ann Surg.* 1985; 201:391-398.
2. Naraghi R, Geiger H, Crnac J, et al. Posterior fossa neurovascular anomalies in essential hypertension. *Lancet.* 1994;344:1466-1470.
3. Thomas C, ed. *Taber's Cyclopedic Medical Dictionary.* 12th ed. Philadelphia, Pa: F. A. Davis Co; 1973. H-76, C-123.
4. Jannetta P, Segal R, Wolfson S, et al. Neurogenic hypertension: Etiology and surgical treatment II. Observations in an experimental nonhuman primate model. *Ann Surg.* 1985;202:253-261.
5. Fischman J. Cutting edge: Brain surgery for high blood pressure. *Discover The World Of Science.* May 1990, pp 54-60.
6. Ammar A, Lagenaur C, Jannetta P. Neural tissue compatibility of Teflon as an implant material for microvascular decompression. *Neurosurg Rev.* 1990;13:299-303.
7. Rothrock JC, Meeker MH. *Alexander's Care of the Patient in Surgery.* Mosby-Year Book. 9th ed. 1991;150-153, 332-335.
8. Tortora G, Grabowski S. *Principles of Anatomy and Physiology.* 7th ed. New York, NY: HarperCollins College Publishers; 1993:434,637.
9. McVan B, Gyetvan M, McCann J. *Diseases & Disorders Handbook.* Springhouse, PA: Springhouse Corp; 1988:377-379.
10. Kleineberg B, Becker H, Gaab MR, et al. Essential hypertension associated with neurovascular compression: Angiographic findings. *Neurosurgery.* 1992;30:834-841.
11. Montgomery C. A tiny piece of Teflon can cure high blood pressure. *National Enquirer* May 2, 1995, p 36.

***Teri Junge**, CST/CFA, has worked in surgery since 1975. She became certified as a CST in 1985 and passed the first assistant certifying examination in 1992. Teri has worked at Palomar Medical Center in Escondido, California, for 12 years. This is the second continuing education article that Teri has written for the journal.*