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DISCUSSION

DR. ROBERT W. RAND (Los Angeles, California): Dr. Jannetta has introduced a unique and original concept of medulla oblongata vascular compression by the vertebral artery as one of the causes of hypertension. Further, he has demonstrated this neurovascular compression phenomenon can be surgically treated in certain instances by microvascular decompression. This experimental and clinical research has historic roots, as he mentioned, in the surgical treatment of the syndromes of trigeminal neuralgia and hemifacial spasm by Drs. Walter Dandy and W. James Gardner, respectively.

I would like to review briefly these operations. The original drawings by Dr. Dandy in the late 1920s depict his evidence that the looping cerebellar arteries can compress and distort the trigeminal nerve and thus cause tic douloureux or major trigeminal neuralgia.

Dr. Jannetta has expanded and popularized this concept of the neurovascular compression syndrome of Dandy as a mechanism of the cause of trigeminal neuralgia. About 103 of 206 instances described originally by Dandy were caused by some type of vascular compression of the trigeminal nerve. In addition to the arteries, Dandy also described venous neurocompression. As Dr. Jannetta has pointed out, not many surgeons believed that Dr. Dandy was correct at that time.

Dr. W. James Gardner in 1959 was the first surgeon to put an absorbable sponge between the compressing cerebellar artery and the distorted trigeminal nerve. The first patient treated by Dr. Gardner remained completely free of tic douloureux without neurologic loss for five years. Subsequent patients treated by Gardner were also relieved of the pain.

It has been 14 years since, with Dr. Jannetta, I operated on my first patient using microneurosurgical decompressive techniques of the trigeminal nerve. The patient remains completely free of tic douloureux and without sensory loss. Additional patients, like those of Dr. Gardner, have been successfully treated at UCLA in this manner by subtemporal transtentorial and suboccipital operations.

Dr. Gardner went on to work on the problem of hemifacial spasm, as Dr. Jannetta has indicated, using similar neurovascular decompression procedures he developed for trigeminal neuralgia. In 1961 Dr. Gardner's first group of seven patients with hemifacial spasm successfully underwent his neurovascular decompression operation without having facial palsy, and yet being relieved completely of the hemifacial spasm.

DR. BROOKE ROBERTS (Philadelphia, Pennsylvania): To those of us who are not neurosurgeons, this concept is almost completely new. I had been aware of Dr. Jannetta's work with regard to tic douloureux and some of the neuralgias of the cranial facial nerves, but this concept of hypertension explained on a similar basis is new and an extraordinarily exciting concept.

New concepts often have difficulty being accepted. It is fascinating when a group of diseases or entities whose cause is poorly or completely understood are brought together to show by a common cause that they can be explained and to have them explained in such a simple way.

What percentage of hypertension, so called essential hypertension, caused by this remains to be seen? Some of it is unquestionably true from the results which we have heard. I look forward to more work from this quarter.

DR. WILLIAM S. BLAKEMORE (Toledo, Ohio): I have watched Dr. Jannetta's work for a number of years. It is true that he has had to go through the criticism of his colleagues to establish the concept of the hyperexcitability of the endocranial portion of the cranial nerves. I wish especially to comment on this newly presented cause for hypertension. It evolves that after 25 or 30 years, when Smithwick and Zintel and Wolferth and others studied surgical treatment of hypertension, which in long-term follow-up studies has better results for the severely hypertensive patient than any medical treatment with ten year follow-up results. We have identified several

entities such as renal vascular hypertension or hyperaldosteronism, some of which were treated surgically but are now treated by more selective surgical procedures. Now we have another new disease entity that might also be treated surgically. I would like to ask Dr. Jannetta if he has any ideas on how we might diagnose this entity short of exploration of the interior of the skull. Is the mechanism humoral or neural, stimulating the pituitary or other end organ? Where does he think the next experimental models might be most effective for use by a number of young surgeons who might be particularly interested in developing further this intriguing concept.

I would also like to take license to present one more idea to this Association, the members of which are interested in training young surgeons. Dr. Jannetta is one of the first academic surgeons trained 20 years ago under a concept, which I had the opportunity to develop with others, including Dr. Carl Moyer, of including two years of laboratory experience in a basic science discipline. Despite the abortive attempt by the federal government to fund these programs, I would recommend to you that there are funds and methods of efficiently training young surgeons, especially those who have talents which promise them to be contributors in an academic setting and those who are interested in academic pursuits in surgical specialties and areas of academic surgery in which there is a manpower shortage.

DR. PETER J. JANNETTA (Closing discussion): Dr. Dandy's work was complicated by the fact that he put an entity that he called trigeminal neuralgia, which we have unfortunately lumped with atypical facial pain, in together with his series of classic trigeminal neuralgias, which he called tic douloureux. That has caused an on-going problem.

In regard to Dr. Roberts' comments, I do not know what percentage of patients will have essential hypertension. This was clearly present in 21 of 23 and in four more patients on whom I have the data. I can only follow up on William Henry Welch, who a long time ago said, "We must remember, gentlemen, that per cent means per hundred." When we have one hundred patients, I may be able to respond more clearly to that. I think it is common.

Dr. Blakemore, I think that a stereoscopic angiogram will be helpful in trying to elucidate this problem. Also, we need better testing of vagal function, other than what we test at present.

I think there are probably many epiphenomena that occur as a result of this pump, the left ventricle, being overdriven—humoral, reflex, even emotional and that, with further elaboration with the experimental model, we may be able to work this out. It will become complicated, because it appears to be both afferent and efferent, hyperactive and hypoactive, central and peripheral autonomic dysfunction, so we're dealing with six parameters.