CHAPTER

1

Presidential Address: A Neurosurgical Approach to the Therapy of Extracranial Occlusive Disease

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Mr. Chairman, founders, members, guests, Dr. William Sweet, our Honored Guest, welcome to the Silver Anniversary Meeting of the Congress of Neurological Surgeons. It is my sincere wish this meeting will be scientifically stimulating and socially satisfying. During the last 25 years, this Society has become the largest of all neurosurgical organizations but continues to be dedicated to the education of the young neurosurgeon. It is my particular honor, an occurrence largely of chance, to serve as President for this anniversary gathering and to have as our special honored guests many founders of this Society and many former presidents. Since the founders formed the Society in St. Louis and the first meeting was held in Memphis, our meeting site is most appropriate. With pleasure, I welcome you to this gracious and exploding queen of southern cities, Atlanta. Sherman's ashes have literally turned to gold, and southern hospitality and leadership are graced by your presence.

These are trying times for the medical profession, and particuarly for neurosurgeons. The interference of government, the problems of recertification and licensure, the restrictions imposed on clinical research, and the aspects of extortion implied in current liability insurance practice concern, confuse, and frustrate everyone. Your Society, by the dedicated, unselfish hard work of the Socio-economic Committee under Dr. Ed Amyes' leadership, is educating and representing neurosurgery regarding these matters in a superb professional manner. We have combined our efforts with a similar committee of the American Association of Neurological Surgeons under Dr. Russel Patterson's chairmanship. United we stand. These positive steps should lead to honorable problem solving, but the work is expensive and demands increasing time. A central office has been established, under the direction of Dr. George Tindall, to represent neurosurgery in continuing education and to coordinate all educational seminars. In our direct negotiations with government, this office could have increasing influence of an unknown

magnitude and require financing that may not be entirely tax exempt. Whatever the cost to survive, I am confident that our profession must and will stand the heat, stay in the kitchen, and preserve the free practice of medicine.

The President of this Society is allowed two special privileges. The first privilege is the selection of the Honored Guest for the Annual Meeting. Dr. William Sweet, our guest this year, is a neuroscientist of outstanding stature, a man of integrity and courage, an outstanding neurosurgeon, an excellent teacher, and a friend. The second privilege is the selection of the theme for the Annual Meeting. This year's program is dedicated to the neurosurgeon's interest in the prevention, research, and medical and surgical therapy of stroke.

If the comments I make have a bias or reflect any point of view that is worthwhile, I wish to give credit to my chief, Dr. Francis Murphy, a truly great clinical neurosurgeon, and to my colleagues in Memphis, particularly the members of the Semmes-Murphey Clinic.

Stroke due to occlusive disease is epidemic in this long living society. According to Dr. Cone Pevehouse, an excellent neurosurgical economist, stroke diagnosis and treatment constitutes a significant segment of the patient load in neurosurgical practice (21). It is a clinical challenge and the third leading cause of death in the United States (11.3%), killing at least 200,000 people per year. It is one of the leading causes of personal and family tragedy (12, 26). Stroke victims overflow our nursing homes. Stroke extracts an untold socio-economic drain on this nation. In minutes an occlusive stroke can change an active and intelligent executive into a dependent, demented, locked-in individual. Dr. Miller Fisher has appropriately defined the victim of a massive stroke as an individual in the state of "super death" (8).

Stroke demands the attention of all neurosurgeons, not only in diagnosis but in active medical and surgical therapy. It is not only a great challenge, but the results of preventive surgery are better than they are in surgical therapy of aneurysms or brain tumors. Recently, our specialty has become very aggressive in establishing neurosurgeons as stroke physicians. Due to the leadership of Dr. John Tew of Cincinnati and Dr. Thor Sundt of Rochester, a Cerebrovascular Section of the American Association of Neurological Surgeons has been formed and they will have their first meeting with the American Heart Association in Dallas in eary 1976. Dr. Steve Gurdjian, a member of the Congress and a former honored guest, has just directed an outstanding postgraduate course on the intra- and extracranial procedures for cerebral ischemia at the American College of Surgeons Meeting in San Francisco. If a few of you are less than qualified in the treatment of this very common disease

entity, you are urged to attend clinics or continuing education programs or work with vascular surgeons to become expert in stroke care. Our speciality can hardly be concerned about the brain without being concerned about its blood supply. Indeed, what other speciality has the potential to deal with this problem as we do?

In the time allowed for this presentation, it is impossible to discuss all aspects of stroke therapy, but the following comments are submitted for your review and consideration:

1. More than half of all deaths are due to the combination of cerebrovascular disease, coronary artery disease, and hypertensive cardiovascular disease (12).

2. Cerebral infarction accounts for nearly two-thirds of the fatal vascular brain disorders.

3. A completed stroke is frequently (40 to 80%) preceded by warning attacks. These transient ischemic attacks are defined as the sudden onset of a focal, usually painless, neurological deficit that clears completely, often in minutes, but certainly within 24 hours (1, 3, 13, 18, 20, 24). Although an individual literally may have had a hundred attacks, there is no correlation as to the number of attacks or when the stroke will occur (8). Vertebrobasilar ischemic attacks have a better prognosis than carotid ischemic attacks. These attacks demonstrate the difference between physiological paralysis and anatomic infarction. The medical profession and the public must be educated repeatedly to recognize these warning bells and take preventive action.

4. Once transient ischemic attacks have occurred, a stroke occurs in an instance of 4 to 10% per annum (20 to 50% within 5 years) (1, 3, 9, 13, 14, 17). The prognosis is still guarded when the arteriograms are normal (16).

5. There are syndromes suggesting cerebrovascular insufficiency; for example, acceleration giddiness, difficulty in rapid thought, slowing of mental function, stepwise dementia that may be related to occlusive vascular disease both intra-and extracranially. These hypoperfusion syndromes require more study and documentation (9, 24).

6. Except in special early circumstances there is virtually no therapy for a completed cerebral infarction (9, 18, 24). Depending upon one's philosophy and the understanding that physiological paralysis may not mean anatomic infarction, there probably is a place for early surgery in symptomatic acute carotid occlusion (4 to 6 hours). Surgery in completed strokes carries a significant increased risk. All surgical and medical therapy is best administered prior to the infarction event. Essentially, therefore, stroke therapy is prophylactic.

7. Although hemodynamic factors are a cause of ischemic cerebral symptoms, emboli (usually platelet aggregates or, on occasion, choles-

terol fragments) are probably the most important cause of transient ischemic attacks (3). According to Fisher, distal emboli have not been proven to occur from the stenotic areas in intracranial vessels.

8. In the arterial system, it is generally agreed that long term anticoagulation is probably ineffective in the prevention of stroke and may carry a greater risk than no therapy at all (2, 15).

9. The dynamic role of platelets in the arterial system has provoked an intense interest in platelet-inhibiting drugs (10, 19). Platelet-inhibiting drugs are now available and fall into three categories:

A. The nonsteroidal anti-inflammatory drugs; for example, aspirin, sulfinpyrazone.

B. The pyrimido-pyrimidine compounds.

C. The tricyclic antidepressants drugs.

Preliminary studies indicate that drugs which inhibit platelet function will be effective in preventing thromboembolic states due to platelet emboli (6). Several studies are underway to test the efficacy of platelet inhibition as a therapeutic answer in the prevention of occlusive cerebral and cardiac syndromes. Should the hypothesis be proven, the benefit to the health of our senior citizens will be inestimable.

10. The present therapy of stroke, providing the patient is in a reasonably good neurological state, requires adequate cerebral arteriography (9, 24).

11. Of all the medical therapies available in the medical treatment of a transient ischemic attack, intravenous heparin is the sole dependable agent. It is best given by constant infusion (18).

12. Carotid endarterectomy has a major role in the preventive treatment of stroke. It can be done with a mortality rate of less than 2% (5, 9, 18, 20, 23, 24).

13. Vertebral endarterectomy, although occasionally indicated, has not statistically been proven to have a high patency rate and to prevent stroke (9).

14. A subclavian steal syndrome, although capable of producing symptoms, is often asymptomatic (4, 7). It is not often followed by cerebral infarction. It can be treated by simple carotid-subclavian bypass surgery (4).

15. The exact indication for surgical therapy with vertebral compression by osteophytes or in the cases of carotid kinks has not been established (9). The exact incidence and documentation of significant thromboembolism distal to small carotid ulceration without major stenosis is unknown.

16. Intracranial bypass surgery; for example, superficial temporal artery or occipital artery middle cerebral or posterior circulation anastomosis, using microsurgical techniques, is now possible with a high

patency rate. Although it is generally accepted that high grade middle cerebral or intracranial carotid stenosis that is symptomatic may be indications for the bypass procedures, they have not been proven to be of lasting benefit in a sufficient number of cases with adequate follow-up (8). However, data are accumulating that are most favorable. Chater has estimated that up to 12,000 cases per year may be available and benefited by these bypass procedures. Presently, cervical carotid-intracranial carotid bypass vein or arterial grafts, although technically possible, are not practical therapeutic measures. Endarterectomy in the intracranial carotid artery has not been of proven value.

17. Since the role of physical and rehabilitative therapy in a significant and completed stroke has not been impressive and because cerebral infarction edema has no proven therapy and may be fatal, neurosurgeons are urged to educate the public about the warning attacks of stroke.

Many of the above statements and assumptions will be discussed and challenged during the scientific sessions of this Congress. However, as a clinical neurosurgeon engaged in stroke therapy, I would like to present a few practical guidelines to the medical and surgical management of extracranial occlusive disease. In the evaluation of a patient with a stroke syndrome, the clinician should establish the temporal profile of the history. The physical examination should include careful auscultation of the cervical and upper thoracic areas but avoid palpation of the cervical carotid arteries (3, 11). The ulcerative plaques that may be contained within these arteries, covered by platelet emboli and/or clot, are extremely susceptible to trauma. Not only have emboli been dislodged, producing an immediate stroke, but, on more than one occasion, both platelet and cholesterol emboli have been seen in the retina after palpation of cervical vessels. A careful cardiac exam is mandatory. The blood pressure in both arms and with position change, if feasible, should be monitored. Additional studies should include a fasting blood sugar and 2-hour postprandial blood sugar level, thyroid studies-usually a T-3 and T-4 determination, an electrocardiogram and, if there is any question about the heart being beset by significant arrhythmias, at least a 12-hour dynamic cardiac monitor determination is indicated. In certain instances, *e.g.*, emboli that are proven or suspected and angiography proves negative, echo cardiography may be very helpful in localizing significant mural thrombi or, on occasion, an atrial myxoma. A chest and skull x-ray and spinal puncture should be routine. When possible, a technetium flow study and brain scan would be extremely helpful in indicating the presence of cerebral infarction or, more importantly as Dr. John Rockett discussed (see Chapter 18), asymmetry of cervical or middle cerebral flow or actual occlusion of a carotid artery in the neck. At least three-vessel cerebral arteriography to include arch aortography

when necessary, providing the patient's condition permits, is considered mandatory. When these factors are determined, a rational medical and/or surgical therapy can proceed.

Auscultation of the cervical vessels has become routine as a result of increasing experience in stroke management. This may present the significant problem of the asymptomatic carotid bruit. Studies are available demonstrating a carotid bruit may be followed in a significant percentage of cases by transient ischemic attacks or strokes (5, 23, 24). Of real concern is the increasing pitch or high pitched bruit which usually indicates a tight stenosis. Providing the patient is otherwise in satisfactory health, it is suggested that ophthalmodynamometry or a technetium flow study should be performed. If the flow study or the retinal artery pressure is decreased on the side of the bruit, cerebral arteriography should be considered (8). If a significant stenotic lesion of 90% or more is visualized, carotid endarterectomy should be considered. The risk of carotid endarterectomy in this individual should not be more than 1% (23, 24). This is less than the risk of further difficulty from that artery.

As Dr. Miller Fisher has said, the patient with the transient cerebral ischemic attack is telling you "Doctor, I am trying to have a stroke." Since there is no way of knowing how many transient ischemic attacks a patient will have before he has a completed cerebral infarction, a patient with an attack deserves immediate attention. Providing the syndrome is clear and particularly if there is an associated appropriate carotid bruit, this patient should probably be managed by obtaining skull and chest x-rays and a spinal puncture and be placed on intravenous heparin. Continuous infusion of heparin is the best way to insure accurate heparinization. It is rare for adequate heparinization to fail to stop transient ischemic attacks (18). This therapy allows a subsequent relative elective evaluation of the stroke syndrome.

A stroke in evolution or early stroke or reversible ischemic neurological deficit is best treated with expediency. Again, angiography is the key to the understanding of the problem. Although it is realized that many vascular surgeons are less than aggressive about this high risk patient, a more appropriate approach would be to immediately render the necessary medical or surgical therapy (5, 24). This aggressive approach can be justified only by obtaining the patient within the first 4 to 6 hours of the event and rapidly determining which therapy is indicated (9). In the case of carotid occlusion with significant neurological deficit, thrombectomy and endarterectomy is recommended. If one operates on this type of patient, the high risk can be justified if the operation is done very early. If thrombectomy produces a good back flow from the internal carotid artery, operative angiography should be done to be certain that all possible clot has been removed before reconstituting flow. Hemorrhagic

cerebral infarction is a definite possibility, although the exact incidence has never been proven (25). Many surgeons have had more than one case who have made dramatic improvements from a near hemiplegic state. On the other hand, in the case of the carotid thrombosis with only transient difficulty, surgery may not be indicated. Since the distal end of the clot may still be active in carotid thrombosis, heparinization for 7 to 10 days to allow the clot to organize and not propagate distally above the carotid bifurcation is recommended. Heparinization for similar time is probably indicated in vertebral thrombosis. This nonsurgical approach is based on the principle that once a vessel that is causing symptoms has become occluded, the clot has become well organized and the patient remains asymptomatic, there is little chance the patient will have difficulty from that vessel again (18). This is not to say that ischemic attacks due to further occlusive disease affecting the collateral blood supply to that hemisphere will fail to occur. Chater estimates with the passage of time, 16% of these patient with carotid thrombosis will develop ipsilateral ischemic symptoms (see Chapter 21).

There are certain circumstances in which surgery should be considered in a patient with a completed infarction. These must be individualized, but one circumstance might be illustrated as follows. A right-handed patient presents with a significant right hemiparesis but with total preservation of speech. Arteriograms reveal a high grade carotid stenosis and a partial embolic occlusion of the middle cerebral artery. The patient might be placed on heparin to prevent further emboli even though there is a risk of a hemorrhagic infarction for approximately a week or 10 days. At this point, he has a hemiparesis but essentially normal speech. He still has the capacity to worsen because another embolus may destroy speech. Additional heparin for another week is followed by arteriography and shows either a persistence or disappearance of the embolus. An acceptable approach to this case would be a left carotid endarterectomy approximately 14 to 21 days after the original completed stroke. This therapy offers preservation of speech and prevents further embolic episodes.

A special problem in surgical vs. medical therapy is often presented by the individual who has bilateral significant carotid stenosis. It is generally agreed if the carotid stenosis is 70% or more, that ultimately bilateral endarterectomy should be performed even on the asymptomatic side (9, 18). This is justified by the low mortality rate of the procedure and the preventive nature of the surgery. The procedures should be done at least 1 week apart. There is some argument in the literature about which side should be done first. Some individuals recommend the most stenotic side should be done first or the side in which cross collateral flow occurs on angiography (9). The patient's symptoms indicate the active

lesion and the symptomatic side should be operated first regardless of the stenosis on the other side. Internal shunts are suggested in bilateral lesions. Prior to operating on the second side, one should document the patency of the operated side either by angiography or by the use of the technetium flow scan. The patency of the vessel can be determined by the dynamic technetium flow scan, but often it will appear to be decreased following surgery. This apparent decrease in flow will not be corroborated by angiography and returns to normal at some time greater than 1 month after surgery. The technique of carotid endarterectomy deserves comment. One must understand and be familiar with vascular surgery principles to perform this operation. This is another subject for subspecialization within neurosurgical groups. Sundt's experience, the best mortality and morbidity available for carotid endarterectomy, illustrates the value of subspecialization (22). To do an operation well, one must perform it frequently. The major problems encountered in carotid endarterectomy are failure to obtain a generous proximal and distal exposure of the affected vessel site and dislodging an embolus from the plaque. Adequate exposure is achieved by making an incision from the region at or just below the ear lobe down along the antero medial border of the sterno cleidomastoid muscle and, when necessary, dissecting up the superficial lobe of the parotid gland and retracting it superiorly. Some surgeons recommend a transversal skin incision. In addition, the patient should be positioned with a sandbag under the operative shoulder with the head turned slightly to the opposite side and the neck somewhat hyperextended. Sharp dissection is mandatory. Gentleness helps prevent dislodging an embolus. Sectioning the ansacervicalis and meticulous dissection of the XIIth nerve allows it to be moved cephalad for a significant distance. The branch of the external carotid artery that usually comes across the top of the hypoglossal nerve can be ligated and divided to allow the nerve to be moved up and insure good distal exposure of the internal carotid artery. The carotid body should be blocked with xylocaine but not disturbed any more than necessary. This is particularly important in the patient with chronic obstructive pulmonary disease (24). Ideally, EEG monitoring should be carried out during this surgery, but it is not essential. General anesthesia is preferred and halothane is the anesthetic of choice (9). Hyperventilation should be avoided. Blood pressure should be elevated slightly. Vascular clamps or ramal tourniquets are used on the distal common carotid and the proximal external carotid. It is worthwhile to use a double loop of No. 0 silk around the distal internal carotid. This allows approximately 0.5 cm. of additional operative space. After blocking the carotid body, the patient is heparinized and, although 50 mg. of heparin are used

commonly, in view of Sundt's experience of finding particulate matter on internal shunts with this dose of heparin, 75 mg are recommended. Additionally, the shunt tubing can be heparinized. The internal carotid artery should be occluded first with subsequent occlusion of the external carotid and common carotid arteries. I prefer the routine use of an internal silastic shunt or Sundt's internal shunt (23). The vessel is opened usually in the common carotid artery with a No. 11 blade knife; Pott's scissors are used to section vertically through the plaque to normal distal intima. A shunt is placed in the internal carotid artery first. It should fit snugly requiring very little compression from the external tourniquet device for security. The shunt is then placed in the common carotid artery being certain to avoid air emboli. Once the shunt is in place, the operation can be done at a comfortable pace. The plaque should be sectioned sharply in the common carotid and peeled out of the external carotid and the internal carotid. At times, a tongue of atheromatous material will extend a significant distance up the posterior medial wall of the internal carotid artery. Almost invariably, the distal end of the plaque will peel out leaving a smooth intimal surface and tacking down of the intima is not carried out routinely. All debris should be removed from the endarterectomized site leaving the vessel wall as smooth as possible. If the internal artery is quite large, simple closure with 5-0 prolene running suture is adequate. However, vein or dacron patches can be used to insure a wide open lumen through the endarterectomized site. The shunt is pulled out prior to final closure of the patch, the internal carotid artery is allowed to back bleed, the external carotid artery allowed to back bleed, and finally the common carotid artery allowed to bleed. The wound is thoroughly irrigated, and final closure is effected by making certain that all of the air is out of the vessel. Then, flow is restored by opening the external, the common, and the internal carotid arteries. Without reversing the heparin, the vessel is carefully palpated and if a thrill is felt, an arteriogram is mandatory. If the arteriogram reveals an intimal flap or any debris, the vessel should be reopened and cleaned. When closure is satisfactory, there should be an excellent pulse without a thrill. Postoperative heparinization is not necessary when these principles are followed. The heparin can be completely or partially reversed at the conclusion of the procedure depending upon the operator's choice.

In summary, there are many unanswered problems in stroke therapy. These comments are presented to stimulate interest and provoke an aggressive approach to stroke therapy by neurosurgeons. There is no doubt that the quality of life can be improved by extracranial and possibly extracranial-intracranial bypass vascular surgery. Prevention of

stroke and extended longevity may be forthcoming by the use of platelet-inhibiting drugs. The main issue, the central plea, is that neurosurgeons lead the way.

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