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Abstracts of the Seventh Annual Stonwin Medical Conference—Endovascular Interventional Neuroradiology

**Sponsored by the Harry Winston Research Foundation, Inc., Held at Stonwin, Scarsdale, NY,
July 10–12, 1989**

The concept of the Stonwin Medical Conference originated with Harry Winston and his wife, Edna. Their support of medical and scientific technological development was well recognized. These efforts were continued by their sons Ronald and Bruce through the formation of the Harry Winston Research Foundation, Inc. A collaboration with the Department of Neurosurgery of the New York Neurological Institute under the directorship of Bennett M. Stein, provided a firm academic atmosphere. In this setting, the first conference was held July 14–15, 1983. It was an immediate success. Subsequently, guests from Europe, the Soviet Union, Japan, and the People's Republic of China contributed enormously to the subjects under discussion. In 1986, an agreement was signed to promote scientific and medical collaboration between the N. N. Burdenko Neurosurgical Institute under the directorship of A. N. Konovalov, the Department of Neurosurgery of the New York Neurological Institute, and the Harry Winston Research Foundation. An exchange program for students and fellows in neurosurgery began in the spring of 1988 and is expected to continue without interruption.

This year's topic—Endovascular Interventional Radiology—was proposed by Professor Konovalov. The group assembled for the conference included the majority of experts engaged in challenging the frontiers of interventional radiology. We were proud to have Juan M. Taveras as our guest of honor and moderator. The conference and its discussions were unprecedented in demonstrating the success and validity of this newest technology and brought to the forefront the problems that must be faced and overcome to ensure future development in this field.

Brief abstracts of the talks are presented here. A complete text with detailed discussions and illustrations will be published by Plenum Press in 1990.

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Interventional neuroradiology. Taveras JM (Massachusetts General Hospital, Harvard Medical School, Boston, MA 02114).

Neurologic surgery is a relatively young subspecialty of general surgery. Aside from emergency evacuations of epidural and subdural hematomas, which go back to ancient history, no attempts to remove lesions such as neoplasms of the brain and spinal cord were publicized until 1879 and 1884, for the brain, and 1887, for the spinal cord. This led many general surgeons, as many as 500, to publish

reports on brain operations between 1886 and 1896, but the total of reporting surgeons in the succeeding 10 years dropped to about 80, probably because of the high complication rate associated with neurologic surgery and the inability to locate a lesion accurately before surgery. Harvey Cushing advocated the development of neurologic surgery as a subspecialty of general surgery. In 1905, in an address entitled "The Special Field of Neurological Surgery," he stated, "It seems clear that in order to advance surgical measures, specialization or better concentration of thought and energies along

given lines is necessary." The result of these efforts by Cushing and others was that neurosurgeons became adept at not only clinical neurology and surgery as applied to the CNS but also neuropathology and radiological diagnosis. Neurological surgery began to develop rapidly after adequate methods for localizing the lesions became available, and the first of these were ventriculography and pneumoencephalography described by Dandy.

Among radiologists, expertise in the interpretation of neurodiagnostic images developed extremely slowly, and thus interpretation of radiographs was carried out mostly by neurosurgeons. This lasted for about 20 to 30 years until programs for training radiologists in the performance and interpretation of these diagnostic procedures began in the late 1950s. Once specially trained persons were available in neuroradiology, the need was recognized, and neurosurgical departments in the United States began searching for trained neuroradiologists and demanding that a service be developed in the various hospitals to deal with the special needs for competent diagnosis of the lesions of the CNS.

Today, we are dealing with an extension of the technical approaches used in diagnostic neuroradiology, particularly in reference to the vascular system, to carry out the treatment of some vascular lesions. These therapeutic measures, for the most part, use intravascular navigation techniques to achieve the occlusion of arteriovenous malformations, arteriovenous fistulas, and some saccular aneurysms and to reduce the vascularity of extremely vascular neoplasms. It has become apparent that this is a valuable method to assist the neurological surgeon in the management of vascular lesions that are totally inoperable or difficult to operate on and sometimes to eradicate totally some lesions such as arteriovenous fistulas without the need for open surgery. Thus, the need has been recognized, and, again, it is the neurological surgeons who are taking the lead in demanding that specially trained persons be made available at the various centers where these vascular lesions of the brain and spinal cord usually are managed.

It is important that we strive to train these subspecialists as thoroughly as possible so that they can become useful, competent members of the teams of professionals who manage these lesions. For this reason, it is suggested that the neuroradiologist be trained traditionally, starting with general radiology and continuing with neuroradiology. The neuroradiological part of the training should be modified to provide additional training in the interventional procedures as well as in neurosurgery. Trainees entering the subspecialty from neurosurgery should complete the training necessary to qualify for neurosurgical board examinations while continuing to pursue a program that will carry them through basic radiology, neuroradiology, and interventional neuroradiology. Concerning the name of this subspecialty, perhaps interventional neuroradiology or surgical neuroradiology is the best term to use. Either of the two is clear.

I see continuing and rapid development in this important new field, and I have no reason to suspect that its growth will not continue.

The interrelationships between neurosurgeons and radiologists in interventional technology. Leeds NE (Beth Israel Medical Center, New York, NY 10003).

This conference has demonstrated the great potential of the endovascular approach to aneurysms. The significant impact of Dr. Shcheglov on the use of balloon catheters in intracranial aneurysms, as well as his impact on the training of several North American interventional neuroradiologists, was apparent from the numerous reports extolling his approach and technical skills.

Endovascular treatment of vascular lesions in the neck, at the base of skull, or within the brain requires a team approach with collaboration of neuroradiologist, interventional neuroradiologist, and neurosurgeon. Appropriate standards and training are required to achieve this goal; the patient is the beneficiary of the best therapeutic technique.

The American Society of Neuroradiology (ASNR) formed an inter-society collaboration committee to work with our clinical colleagues

in the neuroscience community. At the first meeting in Chicago, in May 1988, the neurosurgeons emphasized the need for training in endovascular procedures in view of the advances being made. Anne Osborn, then president of the ASNR, formed a committee, under my chairmanship, that included Alex Berenstein, Grant Hieshima, and Stephen Kieffer. In collaboration with the European Society of Interventional Neuroradiology, we developed a 3-year program for training neuroradiologists or neurosurgeons to perform interventional procedures. At the neurosurgical meeting in Washington, April 2, 1989, the training programs were discussed with the American Association of Neurological Surgeons Task Force on Neuroradiology, chaired by Robert G. Ojemann. Neurosurgical support was given to the training requirements for neurosurgeons to perform endovascular therapy. The successful collaboration between neurosurgery and neuroradiology was stressed, and the result has been and will continue to be a striving for the best diagnoses and patient care as a result of joint efforts and collaboration.

It is hoped that this meeting will foster a collaboration of those present at least to provide useful data on the treatment of aneurysms, arteriovenous malformations, and other vascular lesions. The purpose of data collection is to compare results and learn the best methods of therapy while avoiding unsuccessful therapy or pitfalls.

Flow patterns and the localization of vascular disease in the circulation. Karino T, Goldsmith HL (McGill University Medical Clinic, Montreal General Hospital, McGill University, Montreal, Canada).

Fluid mechanical factors play an important role in the localization of sites of atherosclerosis, the focal deposition of platelets that results in thrombosis, and the formation of aneurysms and dilatation of the vessel wall. Localization is confirmed mainly to regions of geometric irregularity where vessels branch, curve, and change diameter and where blood is subjected to sudden changes in velocity and direction. In such regions, flow is disturbed, and separation of streamlines from the wall and the formation of eddies are likely to occur.

To determine the flow patterns and fluid mechanical stresses at such sites, we have carried out a series of studies, initially in glass models of stenoses and branching arteries and later in isolated transparent segments of arteries and veins in dogs and humans postmortem. The streamlines, fluid velocities, and wall shear stresses were determined by using flow visualization techniques, recording and analyzing the motions of individual particles in suspensions of microspheres and blood cells on 16-mm cine film. The detrimental effects of an arterial stenosis on blood cells were studied in the annular vortex formed downstream of a sudden tubular expansion. We found that the vortex is a region of stagnant flow in which aggregates of red cells and platelets spontaneously form and are trapped, whereas single cells escape and rejoin the mainstream, thereby lowering the hematocrit. Collisions of cells with the wall and their subsequent adhesion also are enhanced. Similar flow patterns were observed in a venous valve in the saphenous vein of the dog where platelet aggregates are known to form the nidus of a large red thrombus in the valve pocket. We also studied the detailed characteristics of recirculation flows in glass models of T-junctions, comparing flow patterns with those obtained in arterial T-junctions of the dog abdominal aorta. The geometry of the aortic T-junctions was such as to minimize the flow disturbances in the aorta and branches. In the human carotid bifurcation, however, a large standing recirculation zone consisting of complex spiral secondary flows was shown to exist in the carotid sinus even at flow rates well below physiologic values in normal subjects.

More complex flow patterns with recirculation zones and spiral secondary flows were observed in the circle of Willis at the junction of the internal carotid and the posterior communicating arteries, at the basilar artery bifurcation, and at the anterior communicating artery junctions. Here, we also saw a selective localization of saccular aneurysms at particular branching sites that appears to be associated with the direct impingement of fluid elements onto and around the flow divider from the central core of the inflow vessel.

The activation of hemostatic mechanisms in interventional neuroradiology. Rand JH (The Mount Sinai School of Medicine, New York, NY 10029).

Among the aims of interventional neuroradiology is the deliberate induction of thrombosis in vasculature having a susceptibility to bleeding or rupture. Injury to the vascular endothelium results in a sequence of events leading to the development of hemostatic plugs or intravascular thrombi. Whether these thrombi consist mainly of platelets or of fibrin probably depends on a number of factors, including local flow conditions, depth of injury, the types of connective tissue materials exposed, endogenous anticoagulant and platelet-inhibiting mechanisms, and the patient's pharmacologic status with respect to anticoagulant medications and platelet-function inhibitors. Platelet reactivity with the injured vascular surface depends on platelet activation by agonists that are released or exposed by the blood vessel wall and involves the participation of platelet membrane-associated glycoproteins. These membrane glycoproteins serve as binding sites for adhesive glycoproteins that in turn mediate binding to extracellular matrix materials or bind directly to extracellular matrix. The formation of fibrin is thought to depend largely on the release of tissue factor, which together with factor VII activates factor IX; activated factor IX together with factor VIII then activates factor X. The tissue factor-factor VII complex also activates factor X directly without the participation of factors IX and VIII. It recently has been discovered that the normal vascular intima lacks tissue factor antigen, but that adventitia is endowed richly with this material. Endogenous anticoagulant mechanisms include the antithrombin system, the protein C-protein S system, and the plasmin activation system.

Detailed knowledge of how these various interrelated systems function will assist in designing interventional strategies that promote the formation of localized thrombi and minimize the complications resulting from either the unwanted extension or the unwanted dissolution of therapeutic thrombi.

Endovascular treatment of traumatic carotid-cavernous fistulas. Serbinenko FA, Lysachev AG, Nekipelov EF, Kljuchnikov SI, Smirnov NA, Smirnov VE, Sutchkov AA (N. N. Burdenko Neurosurgical Institute, AMS, Moscow, USSR).

At the N. N. Burdenko Neurosurgical Institute, 1689 patients with different cerebrovascular diseases have been treated by the endovascular balloon-catheter method since this technique was first introduced in 1969. The method was used most often for treatment of arteriovenous fistulas. Traumatic carotid-cavernous fistulas were seen in 630 patients, and total exclusion of the fistula was accomplished in 98.4%. Additional intracranial clipping of the internal carotid artery was required in 11 patients (1.6%). The internal carotid artery was preserved in 478 patients (75.8%), and occlusion of the carotid-cavernous fistula and the internal carotid artery was performed in 141 (22.6%). Reconstruction of the internal carotid artery often was obtained with partial stealing. The complication rate of endovascular treatment was 12.1%. Complications included dysfunction of oculomotor nerves, transient ischemic attacks, asymptomatic empty-balloon emboli of cortical branches, and persistent pain. Mortality after surgery was 0.8% (five patients). To date, the endovascular balloon-catheter method is optimal for treatment of traumatic carotid-cavernous fistulas.

Indications for the superior ophthalmic vein approach in the management of carotid-cavernous fistulas. Debrun GM (The Johns Hopkins Medical Institutions, Baltimore, MD 21205).

In a series of 100 traumatic carotid-cavernous fistulas, two patients were treated with a balloon introduced through surgical exposure of the superior ophthalmic vein and detached in the cavernous sinus. In one case, the patient had surgical trapping of the internal carotid artery, and therefore no access to the cavernous sinus via the usual endarterial route was available. In the second case, a small trigeminal

artery that filled the cavernous sinus had been injured, and no balloon could enter this tiny vessel. The inferior petrosal sinus could not be used in these two cases for access to the cavernous sinus. The two patients had an anatomic cure with excellent cosmetic results.

From a series of 47 spontaneous carotid-cavernous fistulas of the dural type, five patients were treated by using the superior ophthalmic vein approach after failure of one or several embolizations of all external carotid branches supplying the cavernous sinus. Four of these had no access to the cavernous sinus through the inferior petrosal sinus, and approach via the inferior petrosal sinus failed in the last patient. Four of these five patients had an anatomic cure after one balloon was filled with iodine contrast material and detached in the cavernous sinus. In one of these five, the small size of the angular vein and the tortuosities of the origin of the superior ophthalmic vein stopped us from advancing the balloon catheter into the cavernous sinus. However, it was easy to reach the sinus with a tracker catheter inserted through the superior ophthalmic vein. Platinum coils without filaments were deposited into the cavernous sinus. The patient improved clinically, but had no anatomic cure of the fistula, proving the poor thrombogenicity of platinum coils alone.

Puncture of a large arterialized superior ophthalmic vein has been considered dangerous. I think that surgical exposure of this vein by an experienced team of neuroophthalmologist, surgeon, and vascular neurosurgeon allows a meticulous dissection of the vein and good control of any bleeding by using two vessel loops and by making a small opening in the vein with immediate advancement of the balloon catheter. The procedure must be done in the operating room, and digital subtraction real-time television monitoring must be used. We have not used intraoperative cerebral angiography in any of these cases. The reason is obvious: It would be time-consuming and difficult in the operating room to catheterize all the arteries that usually supply these dural fistulas.

In summary, the superior ophthalmic vein approach is indicated when (1) the usual intraarterial route fails, or no access to the internal carotid artery is available; (2) approach via the inferior petrosal sinus fails, or the inferior petrosal sinus is not patent; or (3) embolization of the external carotid branches has failed to cure the dural fistula. I think that surgical approach via the superior ophthalmic vein is safe and easy and gives excellent cosmetic results. I wonder if it should not be done before embolization of the external carotid branches, which has some potential hazards and is not always successful.

Impact of present technology on intravascular treatment of brain arteriovenous malformations and fistulas. Vinuela F, Dion J, Duckwiler G, Jordan M, Bentson J (University of California, Los Angeles, Medical Center, Los Angeles, CA 90024-1721).

Brain arteriovenous malformations may be associated with a substantial surgical morbidity and mortality, depending on their size and location. The development of supraselective catheterization of individual feeders of the malformation followed by the injection of Amytal (amobarbital) has been complemented with concomitant clinical neurologic evaluation and computerized electroencephalographic brain mapping. In patients with arteriovenous malformations near or at the level of the sensory, motor, or speech cortex, intraoperative electrocorticography with the patient awake may be performed before surgical removal of the malformation.

The correlation between the anatomic information (supraselective angiography, CT, and MR) and the functional evaluation has shown that brain arteriovenous malformations labeled untreatable can be embolized and removed surgically when a shift of brain function in eloquent areas is demonstrated.

The use of supraselective catheterization with new microcatheters and the use of PER embolization functional evaluation of brain have had a significant impact on morbidity and mortality associated with intravascular embolization of brain arteriovenous malformations. Our neurologic morbidity for embolization of arteriovenous malformations has fallen from 18% to 6.5%, and the mortality has decreased from 4.3% to 2%. The percentage of obliteration of the nidus superior to

75% is rising from 35% to 70%. When surgery was done after embolization, removal of the residual malformation was associated with a significant decrease in the time spent in the operating room and in the amount of blood lost.

Several materials were used for the embolization of brain arteriovenous malformations. In 40 patients, a mixture of Avitene, polyvinyl alcohol, and 30% ethanol was used. Histopathologic analysis of the resected malformations showed that the mixture produced a mild-to-moderate intravascular inflammatory response without evidence of angioneurosis or severe inflammation of the surrounding brain parenchyma. This mixture appears to be suitable for those cases in which embolization will be followed by surgical resection of residual arteriovenous malformation.

Cerebral arteriovenous malformations. Berenstein A (New York University Medical Center, New York, NY 10016).

Endovascular embolization of brain arteriovenous malformations has gained acceptance as an important technique in the management of patients who have this problem. Embolization techniques have evolved from the surgical introduction of embolic agents via cut-down of the carotid artery to the use of percutaneous catheter techniques and particulate embolic agents, namely, Silastic spheres and polyvinyl alcohol or liquid tissue adhesives. Currently, the more accepted mode of endovascular embolization of cerebral arteriovenous malformations is superselective catheterization and the injection of liquid embolic agents such as isobutyl-2-cyanoacrylate or *n*-butyl-cyanoacrylate. This is true primarily if embolization will be the only treatment or will be combined with radiosurgery.

Preoperative embolization is used as an adjunct in patients who have lesions that are otherwise nonresectable because of size or location, or it may be done as the first stage of treatment. In these instances, use of Silastic spheres or detachable balloons may be a simple and effective approach. Complete occlusion of brain arteriovenous malformations is usually not possible with particles or balloons.

Palliative embolization may be performed for the control of headaches and for relief of neurologic deficits related to ischemia of the surrounding areas of the brain. The ischemia may be related to an arterial steal or to venous hypertension, which may decrease tissue perfusion further. Occasionally, redistribution of flow toward ischemic brain areas can stop or reduce some preexisting neurologic deficits. Also, it may be of benefit in patients with uncontrollable seizure disorder, who usually have severe venous hypertension and in whom a good endovascular cast can improve the neurologic condition significantly.

In those patients who have a hemorrhage, the goal of treatment should be complete obliteration. However, currently, this may not be possible in a large number of patients referred for endovascular treatment, who usually have the more complex type of vascular malformations. Complete anatomic obliteration by endovascular techniques is thought to occur in 15–20% of cases. However, in small lesions or single-pedicle malformations that are in strategic areas of the brain, complete obliteration can occur in 70–80% of cases. Embolization also can reduce some lesions to a small enough size so that an adjunctive treatment such as radiosurgery may be effective in completely obliterating these lesions.

In those patients whose preoperative assessment clearly indicates that complete obliteration will not be possible, an attempt is made to select pedicles that are involved in the high-flow fistulas or that harbor an arterial or venous aneurysm in their course. If a venous ectasia or area of constrained venous outflow is filling primarily from one territory, and this territory can be reached, partial embolization is acceptable, as this area may represent the weaker area of potential hemorrhage. The location of the malformation also may have a prognostic implication. Long-term follow-up on these patients eventually will show the role of complete or partial embolization of brain arteriovenous malformations and its protection against recurrent hemorrhage or disabling complications.

Successful and safe embolization with isobutyl-2-cyanoacrylate or *n*-butyl-cyanoacrylate depends on superselective catheterization just proximal to the nidus of the arteriovenous malformation and beyond normal arteries. Accurate delivery of the embolizing agent to the nidus requires experience. Currently, the results of various centers that perform significant numbers of cerebral embolizations point to a morbidity of between 5% and 7%, including visual impairment, and a mortality of 3–6%; an additional 10% of patients may have transient neurologic deficit in the early stages after the intervention.

We have treated 25 patients with midline arteriovenous malformations draining into the vein of Galen. The results seem quite favorable when this group is compared with patients who were untreated or treated surgically. The main disadvantage of the cyanoacrylates is that currently they are used on an experimental basis. The ideal agent for cerebral embolization is not yet available. Ideally, the best agent would be one that can be injected and, if it enters the wrong location, can be dissolved and a new cast obtained. The agents must be biocompatible, producing permanent occlusion, even if an incomplete cast has been obtained. They should have low viscosity, be radiopaque, and not adhere to the catheter tip.

To make endovascular cerebral embolization safer, pharmacologic or physiologic testing can be done. This includes the use of 50–75 mg of sodium Amytal (amobarbital) injected superselectively into a pedicle alone or in conjunction with somatosensory evoked potentials.

Endovascular treatment of arteriovenous malformations of the brain. Lysachev AG, Serbinenko FA (N. N. Burdenko Neurosurgical Institute, AMS, Moscow, USSR).

At the N. N. Burdenko Neurosurgical Institute, 318 patients with arteriovenous malformations were operated on (37.4% of all patients operated on). Endovascular treatment was performed primarily in patients who had malformations that were inoperable because of size, location, or associated neurologic conditions. Two methods were used: balloon occlusion of afferent vessels of the malformation (208 patients) and intravascular thrombosis with a cyanoacrylate produced in the Soviet Union (110 patients). Free embolization of branches of the internal carotid artery and proton irradiation were used also. Total exclusion of the malformation by means of the balloon catheter was obtained in several cases. Total thrombosis with cyanoacrylate was obtained in 12.7% of cases; partial thrombosis (70–80%) was obtained in 20%. The remaining part of the malformation could be treated with irradiation. Rupture of vessels by the balloon occurred in seven patients, persistent neurologic deficit in 6.6%, and postoperative death in 2.2%. Clinical follow-up of three groups of patients (without operation, balloon occlusion, and intravascular thrombosis) indicated that endovascular treatment was effective in patients who had inoperable arteriovenous malformations. Intravascular thrombosis of arteriovenous malformations is a promising endovascular method.

Endovascular therapy of cerebral arteriovenous malformations: London, Ontario, experience. Fox AJ (University Hospital, London, Ontario, Canada).

Since 1976, more than 150 patients with brain arteriovenous malformations have been treated by embolization. Early on, embolic material included plastic beads and Gelfoam-Pantopaque mixtures. Those patients treated with beads and no surgery invariably had small artery-to-artery collateral feeding networks on follow-up angiography. In a series of about 120 patients treated with acrylic glue, complete obliteration of the malformation was achieved in six cases; all six had small malformations with one or two feeders. A variety of medium to large arteriovenous malformations in various areas were treated by glue; the outcomes varied. Many patients had had embolization as a presurgical stage, and this enabled complete resection for many, which might have been impossible without embolization. Among other patients who did not have surgical resection, some showed refilling of nidus on long-term follow-up angiography, sug-

gesting that glue did not always block the nidus. In other cases, further thrombosis of the malformation occurred or no change in the result. With glue embolization, mortality was 4.5%, and moderate-to-severe morbidity was 10%. Recently, we have used a combination of Avitene, Ivalon, ethanol mixture, and acrylic glue, mostly as preoperative procedures, and we have had many fewer complications. Embolization is an excellent way of enabling surgical resection for difficult brain arteriovenous malformations. The best cases for complete obliteration by embolization alone are small malformations that are easily resectable.

Microsurgical treatment of deep-seated cerebral arteriovenous malformations. Filatov UM, Eliava SS, Sazanova OB, Moscovichyute LE, Serova NK, Belousova OB (N. N. Burdenko Neurosurgical Institute, AMS, Moscow, USSR).

The problem of surgical treatment of patients with cerebral arteriovenous malformations is one of the most pressing in vascular neurosurgery. According to data provided by several authors, the risk of fatality after the first hemorrhage from supratentorial arteriovenous malformations varies from 10% to 35%. Thereafter, the risk of recurrent hemorrhage is 20%, and morbidity is noted in about 40% of those patients who survive. The danger of hemorrhage from arteriovenous malformations located in the deep structures of the brain, including the corpus callosum, hippocampal gyrus, caudate nucleus, and thalamus, substantially increases because of the proximity of the ventricular system and the functionally important subcortical structures. Published reports that describe the surgical treatment of patients with deep-seated arteriovenous malformations have been based on limited series of observations. Thus, many questions about the treatment of deep-seated cerebral arteriovenous malformations remain unanswered.

The present report is based on analyses of 103 patients who had deep-seated arteriovenous malformations. All of these patients underwent a complex protocol of studies, including thorough neurologic, neurophysiologic, and electrophysiologic studies both pre- and postoperatively. Cerebral angiography and CT scanning of the brain were used to establish the final diagnosis. Tables 1–4 show the location and size of the surgically removed deep-seated malformations and the postoperative mortality.

Tables 5 and 6 show the neurologic morbidity in patients with hippocampal and corpus callosum malformations treated surgically. Analysis of the dynamics of neurologic signs and symptoms showed that in 26 (67%) of the cases an increase in focal neurologic deficits occurred postoperatively (e.g., homonymous hemianopia, memory disturbances). Analysis of the dynamics of neurological symptomatology showed that in 11 cases (27.5%) an increase in focal neurologic symptoms was noted at the time of discharge (homonymous hemianopia, anomia). Two patients died (4.7%). The cause of death in one was occlusion of both anterior cerebral arteries; in the other, anesthetic-related complications.

Thus, precisely defining indications for the surgical treatment of patients, use of microsurgical methods as well as accumulated experience in surgical treatment establishes the groundwork for the successful treatment of deep-seated cerebral arteriovenous malformations.

TABLE 1: Arteriovenous Malformations of the Thalamus (n = 12)

Size	No. Excised
Large	0
Medium	4
Small	8
Postoperative mortality = 0	

TABLE 2: Arteriovenous Malformations on the Caudate Nucleus (n = 12)

Size	No. Excised
Large	0
Medium	5
Small	7
Postoperative mortality = 0	

TABLE 3: Arteriovenous Malformations of the Hippocampal Area

Location	No. of Cases	Size		
		Small	Medium	Large
Anterior 1/3	6	1	3	2
Middle 1/3	13	3	7	3
Posterior 1/3	20	6	12	2
Total	39 (70%)	10	22	7

TABLE 4: Arteriovenous Malformations of the Corpus Callosum

Location	No. of Cases	Size		
		Small	Medium	Large
Anterior 1/3	8	0	8	0
Middle 1/3	11*	3	7	1
Posterior 1/3	21*	8	11	2
Total	40	11	26	3

* Indicates cases with partial excision of malformation.

TABLE 5: Arteriovenous Malformations of the Hippocampal Area: Analysis of the Dynamics of Pre- and Postoperative Neurologic Signs at the Time of Discharge

Location	No. of Patients	Postoperative Deficits	
		No	Yes
Anterior 1/3	6	2	4
Middle 1/3	13	4	9
Posterior 1/3	20	7	13
Total			26 (67%)

TABLE 6: Arteriovenous Malformations of the Corpus Callosum: Analysis of the Dynamics of Pre- and Postoperative Symptoms and Signs at the Time of Discharge

Location	No. of Patients	Postoperative Deficits		Mortality
		No	Yes	
Anterior 1/3	8	7	0	1
Middle 1/3	11	9	2	0
Posterior 1/3	21	11	9	1
Total				2 (4.7%)

Dural arteriovenous malformations. Berenstein A (New York University Medical Center, New York, NY 10016).

Ten to fifteen percent of all intracranial arteriovenous malformations involve the dura exclusively. They consist of a network of small, multiple fistulas, which most frequently involve the regions of the cavernous sinus, sigmoid sinus, tentorium, falx, and anterior cerebral fossa.

A dural arteriovenous malformation is an acquired lesion that follows the pathologic recanalization of thrombosed major sinuses. This has been well documented for dural vascular malformations of the sigmoid sinus and for those of the spinal cord. In the cavernous area, the major signs and symptoms are related to abnormal venous drainage of the ophthalmic venous system, which may be the result of secondary glaucoma, visual disturbances, and cavernous sinus

distension, producing dysfunction of ocular motor nerves. Approximately 30–40% of these lesions will resolve spontaneously, and interventional therapy is not indicated. Relatively urgent treatment may be indicated in cases in which the increased ocular pressure is uncontrolled or rapid deterioration of visual acuity is evident.

Most dural vascular malformations of the sigmoid sinus are clinically innocuous. The major complaint may be an objective tinnitus, which may become quite disturbing. Those malformations draining into cortical veins may result in an intracerebral hemorrhage, or they may manifest themselves in the form of seizures and require treatment. Some other dural malformations of the posterior fossa are associated primarily with significant thrombosis that compromises venous drainage of the normal brain; patients may have increased intracranial pressure, progressive papilledema, and dementia. Occasionally, this may be irreversible.

Embolization has become the treatment of choice for dural arteriovenous malformations in the cavernous sinus. In our experience with more than 50 patients, signs and symptoms disappeared in 94% of cases. The lesion was cured anatomically in the majority of cases. Delayed or secondary thrombosis is more likely to occur after incomplete embolization of cavernous sinus lesions even when branches of the internal carotid artery (C4 and C5) are involved. Whereas in more than 40 patients with dural arteriovenous malformations in the posterior fossa that required treatment, improvement in terms of signs and symptoms was seen in more than 90% after embolization, total anatomic obliteration of the lesion could be accomplished in only 70%. The complication rate in the treatment of dural arteriovenous malformations is about 1% and may include damage to the cranial nerve or unwanted cerebral embolization.

Embolization of these malformations requires a good understanding of the vascular anatomy, as the lesions frequently are at bordering territories and involve intra- and extracerebral anastomoses. Embolization can be done by using particulate material or liquid embolic agents, such as the fast-polymerizing acrylics isobutyl-2-cyanoacrylate and *n*-butyl-cyanoacrylate.

Spinal dural arteriovenous malformations. Berenstein A (New York University Medical Center, New York, NY 10016).

In 1977, Kendall and Logue reported on 10 cases of spinal dural arteriovenous malformations, and in 1980, Merland and coworkers did an excellent review of 13 cases and called these lesions "intra-spinal extramedullary arteriovenous fistulas draining into medullary veins." Only with the recognition of the true pathologic nature of spinal dural arteriovenous or meningeal fistulas has much of the confusion related to spinal cord arteriovenous malformations been resolved.

These lesions are abnormal arteriovenous shunts, usually single arteriovenous fistulas, that are not within the cord at all but in the surface of the dura, usually at the level of the intervertebral foramen. The arterial supply usually arises from a radicular artery in the region of the intervertebral foramen. In most, a single extradural pedicle gives rise to a small (sometimes microscopic) shunt that is within the dura mater itself. From this, a highly tortuous single draining vein emerges. This, in turn, pierces the dura some millimeters either above or below the accompanying nerve root and passes to the coronary venous plexus, producing venous hypertension of the medullary veins. The draining veins are in general quite tortuous and dilated, and they frequently drain cephalad and caudad.

The typical clinical presentation of spinal dural arteriovenous malformations is slow, progressive mixed motor and sensory myelopathy. Leg weakness is commonly the initial sign; this is followed by sensory changes. Radicular or back pain may be present at the time of diagnosis. These signs and symptoms are slowly progressive with or without remission. In our experience and in all reported cases, no evidence of hemorrhage has been found.

Early diagnosis and treatment offer the best chance for recovery. Myelographic examination shows serpentine vascular filling defects in the subarachnoid space and on the surface of the spinal cord. When the myelogram is analyzed carefully, it often is possible to predict the level of the lesion, inasmuch as a single dilated draining

vein enters the intradural space with the nerve root. The spinal cord itself is usually normal in size but may be atrophied. Absence of widening of the spinal cord generally occurs.

Angiographic findings consist of a small, sometimes microscopic, nidus in the dura, with a single dilated and tortuous intradural draining vein that frequently matches what is seen on the myelogram. Ascending venous drainage is seen more often than descending drainage and may reach the intracranial dural sinuses. The feeding artery originates from the intercostal or lumbar arteries after they reach the lateral surface of the vertebral body, and it is not dilated.

Endovascular occlusion of this recently recognized lesion has become the primary treatment of choice. The goal is to occlude the feeding artery, the nidus, and the intradural part of the draining vein. The best embolic agent for these lesions is a liquid embolic material to produce a complete cast, such as isobutyl-2-cyanoacrylate or *n*-butyl-cyanoacrylate. If the nidus is occluded, the shunt may reopen through the vast collateral circulation between the intercostal and/or lumbar arteries, and this is more likely to occur with particulated embolic materials such as polyvinyl alcohol or dura mater.

If embolization fails or if the feeding vessel also gives rise to the anterior spinal artery, surgical resection of part of the dura mater, including the proximal segment of the draining vein, can be curative. Clipping of a feeding artery would not eliminate the shunt.

Embolization of spinal cord arteriovenous malformations. Berenstein A (New York University Medical Center, New York, NY 10016).

The goal of endovascular treatment of spinal cord arteriovenous malformations is occlusion of the nidus of a vascular malformation or the neovascularity of a tumor, preserving vascular supply to normal territory. In order to achieve this goal, the embolic material should be injected as close as possible to the nidus or neovascularity, and the material should be resistant to reabsorption or recanalization. In the last 20 years, several embolic materials and catheter systems have been developed for various applications.

Spinal cord arteriovenous malformations can be divided into three groups: true spinal cord arteriovenous malformations, spinal dural arteriovenous fistulas, and metameric arteriovenous malformations. The prevalence of spinal cord vascular malformation in relationship to the various types of space-occupying lesions ranges from 2% to 4%.

Spinal cord arteriovenous malformations are supplied by the anterior and/or posterior spinal arteries. The malformations may be located totally in the spinal cord, partially in the spinal cord, or totally on the surface of the cord. The mean age (years) at presentation is the mid-20s. However, in more than 50% of patients, the signs and symptoms are present before the age of 16 years. The most common clinical presentation is hemorrhage, subarachnoid or in the spinal cord itself (hematomyelia). Other presenting neurologic signs and symptoms are root or back pain (15–20%), motor weakness (33%), and numbness. Impotence and bowel and bladder dysfunction are often associated signs.

Spinal deformity, kyphosis, and scoliosis may be seen in patients who have spinal cord arteriovenous malformations. Once hemorrhage occurs, the recurrence rate within the first month is nearly 10%; and within 1 year, 40%. Mortality directly related to hemorrhage from the malformation is at least 17.6%.

These malformations can occur at any level of the spinal cord; however, more often they appear in the cervical area (cervical enlargement) and near the conus medullaris. In principle, the malformation has multiple feeders, with high-flow arteriovenous shunting, via the anterior and/or posterior spinal arteries. The feeding radiculomedullary arteries are enlarged in caliber and may have a tortuous course, unlike the straight course of the normal spinal arteries.

Depending on the situation of the nidus, either anterior or posterior spinal arteries will dominate the supply. The size of the nidus and presence of an arteriovenous fistula govern the caliber of the feeders. Aneurysmal dilatation (pseudoectasia) of the feeding artery at the nidus or at the draining veins may occur, which causes widening of the spinal cord, mimicking a tumor and displacing the spinal arteries. The draining veins are usually multiple and dilated, often extending

the whole length of the spinal cord. The anterior and posterior spinal veins can be identified easily on the lateral view of the angiogram. Anastomoses exist between the anterior and posterior spinal veins, frequently perispinally (coronary venous plexus). In cervical arteriovenous malformations, the veins may drain intracranially into the mesencephalic veins, which can cause intracranial subarachnoid hemorrhage.

The development of microsurgical techniques and precise presurgical evaluation, including high-quality spinal angiography, have improved the results of the surgical excision of spinal cord arteriovenous malformations. However, morbidity is still high in patients who have had excision of deeply situated intramedullary malformations. Endovascular embolization has become a highly competitive mode of treatment in the last 15 years. Embolization of deep-seated arteriovenous malformations can be curative with less morbidity than that associated with surgical excision. Preoperative embolization facilitates favorable surgical results. In some instances, partial embolization reduces or stabilizes neurologic deficits.

The goal of embolization is complete occlusion of the nidus of the malformation and feeding arteries, preserving the vascular supply to the normal spinal cord. Historically, pellets of Gelfoam (gelatin sponge) and Silastic spheres were choices of embolic materials. The Gelfoam pieces are difficult to direct precisely into the malformation, and they will be reabsorbed, dissolved in 1–2 weeks; thus recanalization of the occluded segment generally occurs.

In the last 10 years, isobutyl-2-cyanoacrylate and *n*-butyl-cyanoacrylate have been accepted as two choices of embolic materials that can produce a complete cast of the nidus without recanalization. The safety of embolization with these materials is increased when the new softer microcatheters that can be introduced even into the anterior spinal artery close to the nidus are used. In order to evaluate the functional importance of a particular vessel, a provocative test with 50–75 mg of sodium Amytal (amobarbital) is performed before the embolic material is injected. The test is correlated clinically and with monitoring of sensory evoked potentials.

When surgical excision is planned, preoperative embolization can be carried out relatively safely. Polyvinyl alcohol foam of various sizes can be injected by flow direction. This material is small enough to penetrate and occlude the nidus.

Transcranial Doppler studies in arteriovenous malformations. Mohr JP, Petty GW, Massaro AR (The Neurological Institute of New York, New York, NY 10032).

Transcranial Doppler sonography has been used in our institution to measure the velocity of blood in the intracranial arteries in arteriovenous malformations before and after embolization and surgery. Qualitative studies of findings obtained with transcranial Doppler sonography have been described previously. In the arteries feeding a malformation, the mean velocity is typically higher than normal, and the pulsatility index is lower. Using these features, we performed transcranial Doppler sonography before and after treatment of arteriovenous malformations in 15 patients. We compared the hemodynamic changes in feeding and nonfeeding arteries that occur after embolization and surgical resection.

The changes in the mean velocity and the pulsatility index were analyzed in 19 feeding arteries that were embolized or ligated as a part of surgical resection of the malformation. The average percentage decrease in velocity was 39.7%, and the mean decrease was 46.5 cm/sec ($p < .001$, two-tailed paired *t* test). The mean decreases in velocity were somewhat greater for the surgically treated arteries than for embolized arteries, but these differences were not statistically significant. Increases in the pulsatility index were greater for the surgically treated arteries than for embolized arteries, but these differences also were not statistically significant.

Our study has confirmed the previous assumption that embolization produces hemodynamic changes in arteriovenous malformations that are similar to those that occur after surgical resection. Our data showed that embolization had a therapeutically important effect on the embolized feeding artery, one similar to that seen in feeding arteries after surgical resection. It may be important that such effects

are not readily seen on angiography, a technique not as sensitive to changes in flow velocity. Our data also indicate that surgical resection produces hemodynamic changes in feeding arteries consistent with elimination of the shunt. After embolization, it was possible to show that the flow to the malformation by vessels not embolized increased, as inferred by increasing velocity and declining pulsatility index in these vessels. Such changes were evident almost from the first and did not change in the few instances of follow-up studies done months later. Thus far, for angiographically visible malformations, some abnormality in velocity or pulsatility index has been observed in transcranial Doppler studies of feeding arteries.

These data show that embolization results in hemodynamic changes that are qualitatively similar to the changes that occur after surgical resection of arteriovenous malformations. Transcranial Doppler sonography is a reliable and convenient noninvasive method of monitoring the hemodynamic effects of treatment of these malformations.

Determining the effects of treatment: transcranial Doppler studies in arteriovenous malformations. Mohr JP (The Neurological Institute of New York, New York, NY 10032).

Efforts to estimate the impact of intervention on the natural course of disease finally has reached even arteriovenous malformations. Here, however, the attempt to estimate the effects of treatment is confounded by the small data base; the great difficulty of obtaining data on the natural history; a history that spans decades of risk; the nonuniformity of the disorder, which may be a family of conditions rather than a single one; and finally and not least, the fact that the outcomes of surgery and interventional radiology appear to depend heavily on the skills of the management team more than do many other conditions.

Until the 1930s, arteriovenous malformations scarcely were identified at all autemortem. In the last few years, improvements in neuroradiologic imaging have increased the rate of discovery. Because most of the reported cases come from large centers with special interest in surgical treatment, data on the natural history of the disorder are unclear. The largest series report of the prevalence of arteriovenous malformations is the 549 cases (8.6%) among 6368 subarachnoid hemorrhages. Because subarachnoid hemorrhage accounts for roughly 10% of strokes, arteriovenous malformations cause approximately 1% of all strokes. These figures are reflected in another population-based prospective study of stroke carried out by me and my colleagues in southern Alabama in the United States. In an eligible population of 100,000 patients studied over a period of 3 years, nine arteriovenous malformations occurred among 494 new cases of stroke, an incidence of 1.8%.

Few studies have dealt with the natural history of unruptured malformations. The few reported contain a mixture of asymptomatic and symptomatic cases with various presentations, with nonuniform decisions on who has surgery and who does not. Estimations of the risk of initial hemorrhage vary from 1% to as high as 4% per annum. For rebleeding, the estimation is as high as 17% in the first year, settling down to 3% after 10 years, with a 29% risk of death by 20 years after the diagnosis. Little is known about possible changes in the malformation over time and the effect such changes have on the prognosis. Nor is it known which forms of arteriovenous malformations are static anomalies and which grow and whether the risk of hemorrhage is related to such events.

Estimating the efficacy of embolization or surgery will be difficult to test in a clinical trial. Fox, Pelz, and Vinuela attempted to determine whether a clinical trial of the efficacy of embolization is feasible. Assuming an annual risk of first hemorrhage of 1–2% and an annual rate of rehemorrhage of 3–4%, they contrasted their experience at Western Ontario with 111 cases and found a mortality rate of 4.5% and permanent morbidity of 12.6%. By assuming a combined morbidity-mortality of 10% with treatment and a hemorrhage risk of 4% and by assuming that treatment reduces bleeding rate by 50%, they calculated that 5250 cases followed for a mean of 10 years would be needed to conduct a trial to compare treatment with natural history, a practical impossibility. Although other groups might disa-

gree as to the risks of embolization, each group arguing that the risk is lower in their institution, the constant changes in technique make it difficult to compare results between centers and even in the same center over time. The material used for embolization has evolved over the years from pieces of fascia and dura to barium-impregnated Gelfoam (gelatin sponge), barium-impregnated Silastic spheres, polyvinyl alcohol powders, several forms of polymerizing substances such as silicone and bucrylate, and, most recently, detachable balloons, platinum coils, and even newer glues that remain flexible. Add to this the possibility that the patient may have had one of several forms of radiation and then surgical intervention, and it requires little imagination to determine that it will be almost impossible to compare a given form of treatment with another form.

The issue is not the smaller lesions that are obliterated. It is generally agreed, but untested, that obliteration of the malformation rids the patient of the major risks of death from the lesion. The issue is, instead, the common larger malformations that resist obliteration by embolization and radiation and are associated with high morbidity and with technical difficulty in their total removal.

A proposal was put forward at the meeting to devise a uniform method of data collection to form a data bank to determine at least the spectrum of the cases being treated. It is hoped that such a data bank would permit early detection of complications of the newer forms of treatment. If such a data bank can show some uniformity among cases, then the feasibility of a clinical trial can be tested.

Endosaccular detachable balloon catheter treatment of cerebral saccular aneurysms. Scheglov VI (Neurosurgical Institute of Kiev, USSR).

Endovascular technology is developing and undergoing refinement rapidly. The experience of 725 endovascular procedures on saccular aneurysms during the past 14 years at the Neurosurgical Institute of Kiev has shown that the operation is relatively simple and the associated trauma is minimal. Yet, the system of occlusion is both complex and demanding, requiring the determination of appropriate balloon shapes for given aneurysms, the "training" of each balloon such that its entry into the aneurysm is facilitated, and the use of one or two additional balloons concomitantly to generate turbulent flow artificially and permit balloon entry into the aneurysm. Strict monitoring of the patient is mandatory during and after each procedure.

Operations are divided into reconstructive procedures during which the aneurysms were occluded and the parent vessels preserved (561 cases, 91%) and deconstructive procedures in which the parent vessel, at the level of the aneurysm was occluded along with the aneurysm (56 cases, 9%).

Most of our operations were performed on ruptured aneurysms at least 3 weeks after the ictus. We have operated on approximately 17 patients with acute aneurysmal rupture without mortality. Acute-phase occlusions require special preparations, including specially designed balloons, soaking the latex in thrombin and oil, occluding the aneurysmal cavity without dangerously increasing intraluminal pressure, and leaving the catheter in place for at least 24 hr. After occlusion of the aneurysm has been verified, we do not hesitate to use induced hypertension to overcome the effects of vasospasm or anticoagulation to mitigate against symptoms thought to result from embolic or thrombotic events.

At the Neurosurgical Institute of Kiev, all aneurysms are managed by the endovascular technique regardless of their location. Giant aneurysms larger than 5 cm are usually managed with a combined approach of endovascular occlusion via two to three balloons and direct surgical intervention. Aneurysms smaller than 0.5 cm are not treated by endovascular methods.

Our results based on the total of 617 patients indicate that outcome was good in 467 or 80% and fair in 117 or 20%. Thirty-three patients died after the operation, representing a mortality of 5.4%. Of those deaths, nine (1.7%) occurred after operations on 519 patients in fair condition, and 24 (24.5%) occurred after operations on 98 patients

in poor condition. Four patients died 6 months to 4 years later of unrelated illnesses.

Tables 1-8 illuminate the data accumulated during our experience and are based on the total of 617 patients.

TABLE 1: Aneurysm Location and Type of Operation

Location of Aneurysm	Total Cases	Reconstructive Operations	Deconstructive Operations
Internal carotid	301	266	35
Cavernous	52	43	9
Ophthalmic	49	42	7
Posterior commun.	172	153	19
Bifurcation	28	28	0
Anterior commun.	207	195	12
Middle cerebral	64	61	3
M ₁	5	5	0
M ₂ and M ₃	59	56	3
Vertebrobasilar	17	15	2
Multiple aneurysms	28	24	4
Total	617	561 (91%)	56 (9%)

TABLE 2: Aneurysms That Also Could Be Treated by Direct Surgical Methods with Comparable Results

Total number	409
Reconstructive operations	376
Deconstructive operations	33
Died after operation	22
Of 338 patients in fair condition	6 (1.7%)
Of 71 patients in poor condition	16 (22%)

TABLE 3: Giant Aneurysms

Total number	69 (11% of series)
Reconstructive operations	54
Deconstructive operations	15
Died after operation	6
Aneurysm diameter, 2.5 cm	60 (1 died)
Aneurysm diameter, 5.0 cm	9 (5 died)

TABLE 4: Ophthalmic and Intracavernous Aneurysms

Ophthalmic	49
Reconstructive	42
Deconstructive	7
Died after operation	2
Intracavernous	52
Reconstructive	43
Deconstructive	9
Died after operation	2

TABLE 5: Vertebrobasilar Aneurysms

Total number	17
Reconstructive	15
Deconstructive	2
Died after operation	1
Complications	2

TABLE 6: Multiple Aneurysms

Total number	28
Reconstructive	24
Deconstructive	4
Died after operation	4
Of 20 patients in fair condition	1
Of 8 patients in poor condition	3
25 patients with two aneurysms, and one patient each with three, four, and five aneurysms, respectively	

TABLE 7: Small Aneurysms (≤ 0.5 cm)

Total number	17
Reconstructive	5
Deconstructive	12
Died after operation	0

TABLE 8: False Traumatic and Carotid-Cavernous Aneurysms

Total number	15
Reconstructive	14
Deconstructive	1
Died after operation	0
Characteristic features: profuse nasal bleeding	

Endosaccular treatment of berry aneurysms by the endovascular approach: analysis of 40 cases. Moret J, Picard L (Fondation A. De Rothschild, Paris, France, and Hopital St. Julien, Nancy, France).

After developing a special modification of our catheter that allows us to eliminate the problem of catheter dead space when polymerizing substances are used, we began to deal with the endosaccular treatment of berry aneurysms. Most of the aneurysms were also suitable for surgical clipping, making this preliminary study a potential source of material for comparison with direct neurosurgical treatment as regards results and complications.

Forty aneurysms in 38 patients were treated by endovascular techniques in this study; two patients had aneurysms in two separate locations. In four cases, it was not possible to treat the aneurysms either because atherosclerotic changes contraindicated catheterization of the parent vessel at the aortic arch or because the neck of the aneurysm was small or the parent vessel was stenotic, which precluded positioning the balloon in the aneurysmal sac. Therefore, 36 berry aneurysms were occluded by placement of an endosaccular balloon. The anatomic sites of the aneurysms were as follows: four intracavernous, seven caroticoophthalmic, five posterior communicating, five carotid bifurcation, 10 basilar artery (tip or trunk), three middle cerebral artery, four anterior communicating artery, one posterior cerebral artery, and one posterior inferior cerebellar artery.

Six patients were treated in the acute phase after bleeding. In that group, we had five anatomic cures. One patient died as the aneurysm was occluded; a combination of vasospasm and progressive thrombosis of the basilar tip occurred. In one case, we did not succeed in catheterizing the parent vessel. Thirteen patients were treated after the acute phase, between the third and eighth weeks after bleeding. In that group, we had 12 anatomic cures. In one case, we did not succeed in catheterizing the neck of the aneurysm. Two complications occurred in that group: one hemianopsia and one partially regressing hemiplegia. In 21 patients who had an aneurysm that had been discovered incidentally, we obtained 19 anatomic cures. In two cases, we did not succeed in reaching the aneurysm. In one of the patients, hemiplegia occurred after rupture of the balloon and migration of the polymerizing substance into the middle cerebral artery. On long-term follow-up (6 to 12 months), four aneurysms have partially

recanalized and required a second balloon treatment. Three of the four aneurysms were developing from the basilar tip.

The analysis of results and complications of this series is promising with regard to the technique. It seems correct to say at this time that aneurysms of the basilar tip and those at the caroticoophthalmic junction should be treated by the endovascular approach as the first choice of management.

Endovascular treatment of arterial aneurysms. Kononov AN, Serbinenko FA, Filatov JM, Lasarev VA, Tchurilov MV, Nekipelov EF, Sazonova OB, Serova NK (N. N. Burdenko Neurosurgical Institute, AMS, Moscow, USSR).

Endovascular treatment of arterial aneurysms by means of a balloon catheter has been performed at the N. N. Burdenko Neurosurgical Institute since 1970. Different types of endovascular procedures have been developed: reconstructive (occlusion of aneurysmal neck or sac), deconstructive (proximal occlusion or occlusion at the aneurysmal level), and combined (bypass, dissection of giant aneurysms, exclusion from circulation, and so forth). From 1970 to June 1989, 267 patients were operated on (14% of all patients with arterial aneurysms who were operated on during that period). In most cases, they had giant aneurysms that were in difficult sites (cavernous, caroticoophthalmic, supraclinoid) (162 cases) or false traumatic cavernous aneurysms with massive epistaxis that required urgent intervention (44 cases). In 62.7% of cases, the aneurysm was excluded from circulation with the cerebral vessel with good results. In cases of cerebral circulatory insufficiency, endovascular operations were combined with bypass surgery. Combined operations were performed in 37 patients (1.9%). Total exclusion from circulation was obtained in 91%. The main complication was thromboembolism of cerebral vessels (7.9%). Postoperative mortality was 7.5%.

Endovascular operation is an effective method of treatment of giant aneurysms, aneurysms located in a difficult site, and false aneurysms with massive epistaxis that require urgent intervention.

Aneurysms. Berenstein A (New York University Medical Center, New York, NY 10016).

Aneurysms are one of the most exciting frontiers where surgical neuroangiography may offer an alternative in management. Currently, it is especially valuable in those instances in which contemporary surgical techniques are unavailable or, if available, are associated with significant risks.

Balloon technology can be divided into those procedures that result in a reconstructive operation in which the parent vessel is preserved or those cases in which the parent vessel cannot be preserved and the artery is occluded either proximally or by balloon trapping. Ongoing investigations have led to the use of detachable balloon-catheter systems as an alternative to vascular clamping and ligation for the promotion of thrombosis for symptomatic, giant, unclippable intracranial aneurysms. Aneurysms of the internal carotid and vertebral arteries have been thrombosed by balloon occlusion just proximal and distal to the neck (trapping). Cerebral ischemia may result from rapid surgical ligation or balloon occlusion of the common or internal carotid arteries in 20–30% of cases, and the outcome cannot be predicted accurately by angiography. Tests aimed at more accurately predicting tolerance to carotid occlusion, including the determination of regional cerebral blood flow, are still under development. Slow-vessel closure with a Selverstone clamp, under systemic heparinization, may allow reopening of the vessel if neurologic clamping is of questionable value because no significant drop in perfusion pressure occurs until the final closure. Therefore, tolerance testing is desirable before occlusion of a vessel with clamps or balloons as extracranial/intracranial bypass procedures may be performed and may reduce immediate ischemic complications after closure of the carotid artery.

The choice of balloon occlusion or ligation is not settled, although balloon occlusion does avoid a surgical incision and allows a trapping

procedure. By preventing retrograde filling of the aneurysmal lumen, the trapping procedure may provide more secure occlusion of an aneurysm than proximal ligation does. Just before permanent occlusion via a balloon is done, a tolerance test can be performed with a balloon catheter. Inflation of the balloon to occlude an artery temporarily that subsequently will be closed permanently is maintained for 20 min under systemic heparinization.

Balloon occlusion is performed under neuroleptic analgesia; the patient is awake to allow neurologic testing during the tolerance test and the occlusion. Balloons are used for arterial closure, as inflation often is maintained for months to years. After closure, blood pressure is maintained to ensure adequate cerebral perfusion. Clinical tolerance to occlusion of the carotid artery was reported as excellent when angiography showed good cross-filling of the ipsilateral middle cerebral artery from the anterior or posterior communicating arteries during temporary balloon occlusion of the ipsilateral carotid artery.

In view of the risks involved, the procedure still must be considered investigational. However, as experience with using it accumulates, the advantages over the sometimes unpredictable results of carotid ligation may outweigh the risks, and balloon occlusion may be a practical alternative for obliteration of an aneurysm.

Treatment of intracranial aneurysms and arteriovenous malformations with preshaped thrombogenic coils. Hilal SK (The Neurological Institute of New York, New York, NY 10032).

We recently have used thrombogenic coils for the treatment of intracranial aneurysms and arteriovenous malformations in 198 patients. Six patients had aneurysms, and 192 had vascular malformations. The coils were made either of platinum wire coated with Dacron fibers or of platinum wire coated with copper and Dacron fibers (Cook Inc., Bloomington, IN). A double-helix construction also was used in which one platinum wire and one copper wire were both wound in a single coil. The multimetallic construction of platinum and copper was more thrombogenic and also produced a galvanic effect that enhanced thrombogenicity.

For treatment of aneurysm, the coils were preshaped in a figure eight configuration before insertion so that they expanded immediately on release in the aneurysm. The coils were delivered selectively inside the lumen of the aneurysm by using a "Tracker" microcatheter. A technique was used that allows the strengthening of the neck of the aneurysm, with some of the coils projecting in the main artery as prongs. After endothelialization, these prongs add support to the neck of the aneurysm. The technique offers the advantage that the coils conform to the shape of the aneurysm and that the main lumen of the parent artery remains open even in cases in which the neck of the aneurysm is large. The problem of balloon dislodgment into the main artery thus is avoided. We treated three aneurysms of the intracavernous carotid artery, one of the middle cerebral artery, one of the basilar artery tip, and one of the vertebral artery. We achieved obliteration of four aneurysms and nearly complete obliteration of two. There were no stray coils. The method appears to offer a safe alternative to the balloon technique, particularly for irregular aneurysms that have a wide neck.

For arteriovenous malformations, the coils used are straight segments 3–4 mm long. They are injected into a vascular malformation to produce an "interstitial platinum cast" of the malformation when the angioarchitecture of the lesion is coarse. In these cases, rapid shunting precludes contact time sufficient for a liquid embolic material to produce thrombosis. The liquid embolic material is injected after the flow through the malformation has been slowed down sufficiently with the coils. When the vascular malformation has a fine racemose architecture, the liquid embolic material is injected first, and then the supplying vessels are obliterated with the coils to prevent recanalization.

Angioplasty of arterial vasospasm. Hieshima GB, Higashida RT, Halbach VV (University of California, San Francisco, San Francisco, CA 94143).

Intracranial arterial vasospasm after subarachnoid hemorrhage is associated with a significant rate of morbidity and mortality. We have

investigated transluminal angioplasty as a treatment of intracranial arterial vasospasm. The procedure is performed via a transfemoral approach, and a silicone microballoon is used. A total of 27 vessels in 13 patients were treated by transluminal angioplasty. The vessels included vertebral, basilar, posterior cerebral, cavernous and supraclinoid internal carotid, and middle and anterior cerebral arteries.

In all patients except one, all vasospastic vessels that were dilated remained dilated. The exception was a patient who had a recurrent subarachnoid hemorrhage and recurrent vasospasm. Three patients did not have signs and symptoms related to vasospasm. In them, the angioplasty was performed to pass through the zone of narrowing to reach an intracranial aneurysm that was treated by occlusion via a detachable balloon. Five patients had dramatic and rapid reversal of neurologic deficit after angioplasty. Three patients showed no clinical change after dilatation of the vasospastic area. Two patients had complications related to transluminal angioplasty: One had rupture of the cavernous carotid artery when a Latex angioplasty balloon was being used. The second had successful dilatation of the area of vasospasm without significant reversal of signs and symptoms associated with this territory. Approximately 14 hr after the angioplasty, the patient had a hemorrhagic infarction involving the perforator artery territory of the zone of vasospasm. This infarction was correlated with previous studies on reperfusion infarcts and may give evidence that angioplasty of the parent artery can result in improvement in perfusion through perforator arteries within this vascular territory.

Follow-up angiography was performed in four patients within 24 hr of treatment and at 1-week or 1-month intervals. This has shown continued patency of the dilated vessels and return to normal caliber in all cases except one, a patient who had recurrent subarachnoid hemorrhage and recurrent vasospasm. This technique may have greater value in a series of patients in whom early clipping of the aneurysm is followed by vasospasm. This vasospasm can be managed aggressively medically. If medical management fails to arrest the vasospasm and improve the clinical state of the patients, then transluminal angioplasty may be of significant value. The pioneering work of Dr. Zubkov at the Leningrad Institute on angioplasty of vasospasm may provide further insight into the indications and contraindications of this method.

Endovascular therapy of cerebral aneurysms: London, Ontario, experience. Fox AJ (University Hospital, London, Ontario, Canada).

Since 1978, approximately 110 patients with brain aneurysms have been treated by using detachable balloons. The standard protocol of occluding the parent artery by means of a detachable balloon after an occlusion test in the awake patient, under systemic heparinization, has been performed in more than 90 patients. All who had petrous or cavernous aneurysms had complete isolation of the aneurysm. Of 26 patients with supraclinoid aneurysms, 16 had complete thrombosis of the aneurysm despite the occlusion not trapping the aneurysm. Another 10 patients needed further surgery to achieve complete thrombosis of the aneurysm. Similarly, in three of six basilar aneurysms, complete thrombosis was achieved by means of bilateral vertebral occlusion despite the lack of trapping, and in three cases, additional surgery was required. Ischemic events occurred hours to days after treatment in 10% of cases. One third of these had strokes; the other two thirds had minor transient ischemic events. One of the three stroke patients had some residual foot weakness (1.1% permanent deficit). About one third of the carotid cases had bypass surgery.

In the group of 20 or so patients treated by detaching balloons within the aneurysm, a variety of technical problems and clinical complications occurred. All cases were difficult unclippable aneurysms, almost all giant aneurysms, and most with wide necks. The main problem was regrowth of aneurysm from a residual neck remnant, a situation known from surgical clipping. Comparing this with the excellent large successful series done by Shcheglov in Kiev suggests that the best cases for complete obliteration by intraaneurysmal balloon are aneurysms with small necks, the type of aneurysms treated by standard neurosurgical clipping. In the Kiev series, giant aneurysms and aneurysms with large necks appeared to be associated with greater risks.

A neurosurgical point of view on the interventional neuroradiologic approach to cerebral aneurysms. Solomon RA (College of Physicians and Surgeons of Columbia University, New York, NY 10032).

The last decade has seen the rapid development of interventional neuroradiologic approaches for the treatment of intracranial aneurysms. Microcatheter techniques are becoming increasingly refined, but the level of morbidity and mortality associated with balloon occlusion of aneurysms is just beginning to be addressed in a scientific fashion.

The neurosurgical treatment of intracranial aneurysms has been refined to a high level, and in most cases, surgical treatment of aneurysms is associated with a morbidity and mortality rate that is less than 5%. The neurosurgeon can take advantage of techniques not available to the interventional neuroradiologist. The surgeon can obtain both proximal and distal control of the artery that contains the aneurysm by using temporary clips. Intraoperative rupture and giant aneurysms thereby can be handled with some facility. By means of the microscope, perforating arteries or distal branches of the main artery can be seen and their lumens preserved.

Definitive clipping of an intracranial aneurysm is a proved technique, and regrowth of an aneurysm from the site of a previous clipping is extremely rare. All reported cases of regrowth of an aneurysm were in patients in whom either a residual neck of the aneurysm was visualized during postoperative angiography or no control angiogram was performed. If no residual neck can be visualized on a postoperative angiogram, delayed rebleeding or formation of an aneurysm at the same site approaches zero.

Endovascular treatment of intracranial aneurysms is certainly an exciting field. However, development of this technique must proceed slowly and in conjunction with experienced neurosurgeons. Currently, application of interventional radiologic techniques to treatment of intracranial aneurysms should be limited to patients who have had surgical exploration done by a competent surgeon who has experience in treating aneurysms. In some instances, the experienced surgeon will know in advance that clipping will be impossible, and these patients could be referred directly to the interventional neuroradiologist. Large intracavernous aneurysms that do not project into the subarachnoid space seem to be appropriate aneurysms for treatment by the interventional neuroradiologist inasmuch as the natural history of these lesions does not warrant the risk of morbidity and mortality associated with direct surgical approaches. At our present level of sophistication, intracranial surgery in the medically stable patient is superior to endovascular techniques for the treatment of all intracranial aneurysms that are amenable to direct clipping. As time progresses and more experience is gained with the use of intravascular techniques, the indications for endovascular treatment of aneurysms can be liberalized as experience dictates.

Embolization of intracranial vascular disorders. Wang C, Wu Z (Beijing Neurosurgical Institute, Beijing, People's Republic of China).

Eighteen cases of traumatic carotid-cavernous fistulas (14 men and four women, 21–52 years old) were treated by using our self-made detachable balloon. The locations of the fistulas were shown by angiography. Five were in the C3 segment, seven in the C4 segment, and five in C3–C4 segments. One patient had a bilateral fistula; the left part was in C3–C4 segments, and the right was in the C3 segment. The conventional Seldinger technique was used in all of our procedures, including femoral and carotid approaches. In order to make the balloon radiopaque, a stainless-steel wire was placed in the lumen of the balloon preoperatively. When occlusion of the fistula occurred, the patient's intracranial noise disappeared immediately. We usually used 60% Conray (iothalamate meglumine) to inflate the balloon. The volume of contrast material injected was determined by the size and appearance of the balloon. In this group, two or three balloons were needed to close the fistula in most cases. If the balloon could not enter the fistula, the first balloon was put at the fistula site, and a second balloon was placed proximal to the fistula. The carotid artery should be preserved as far as possible even though in most cases no complications occurred after trapping of the fistula. If the internal carotid artery cannot be preserved, it is necessary to occlude

it temporarily for more than 30 min. At the same time, the neurologic functions of the patient are monitored carefully before the balloon is detached. Fifteen of 18 fistulas were embolized successfully. The internal carotid artery was kept patent in nine cases. The bruit disappeared immediately after embolization in nine cases, within 1 week in three cases, and in 2 months in two cases.

Twelve patients with intracranial arteriovenous malformations were treated with isobutyl-2-cyanoacrylate embolization by means of the so-called sandwich injection technique. In total, 19 surgical angiographic-embolization treatments were performed. Although occlusion was complete in only one case, five patients improved clinically, and another two recovered fully. This suggests that the microballoon-catheter technique is useful in the treatment of dural arteriovenous malformations and in the preoperative treatment of arteriovenous malformations in Rolandic or speech areas or deep in the brain as long as the malformations have large arterial feeders. Isobutyl-2-cyanoacrylate is not the most satisfactory agent for embolization of cerebral arteriovenous malformations, but its use can result in an improvement in patients' clinical status. In order to prevent the microballoon catheter from adhering to the vessel wall during embolizing procedures, injection of an excessive amount of the cyanoacrylate should be avoided. The sandwich (or continuous column) technique should be used, and the microballoon catheter should be withdrawn rapidly immediately after the embolizing material is injected and before the retrograde column of the cyanoacrylate approaches the tip of the microballoon catheter.

Interventional neuroradiology: the Massachusetts General Hospital experience. Pile-Spellman J (Massachusetts General Hospital, Boston, MA 02114).

During the last 2 years, nearly 350 interventional neuroradiologic procedures have been performed in slightly more than 200 patients. The diseases treated included bleeding, tumors, craniofacial arteriovenous malformations, aneurysms, vasospasm, and stenotic cerebral vascular disease; most of these patients had brain arteriovenous malformations. During this period, a shift has occurred, from treating for heroic and palliative indications to increasing adjuvant and definitive treatment.

These changes have been brought about by advances in materials, methods, and our understanding of the disease. These in turn have made it possible to (1) localize the pathologic process more focally, (2) identify normal structures, and (3) effectively ablate abnormal structures. The first has been done with the help of more selective catheterization that uses variable stiffness microcatheters and with a greater use of more direct vascular access, such as the transcarotid or direct percutaneous approach. Additionally, greater understanding of the nature and findings and effect of secondary high-flow angiopathy has allowed us to focus on the underlying disease. Selective temporary tolerance testing by using Amytal (amobarbital), Xylocaine (lidocaine), or temporary occlusion with close tailored neurologic examination or electrophysiologic monitoring of the patient is performed on vessels before the vessels are occluded permanently. This has allowed greater confidence in the occlusion of noncritical vessels, has allowed occlusion of vessels in which such a procedure otherwise would have been expected to create a deficit, and has stayed occlusion of presumably noncritical vessels that would lead to significant deficits. Additionally, it has provided important physiologic information to the surgeon in those cases in which the occlusion or ablation is to be done surgically. The safe and effective ablation of the vessel has been made possible with the help of new tissue adhesives, including *n*-butyl-cyanoacrylate and cryoprecipitate-thrombin mixture.

Our first experience with *n*-butyl-cyanoacrylate (Avacryl) in 28 patients has been extremely encouraging. Technically, the material has many of the beneficial properties of isobutyl-2-cyanoacrylate such as low viscosity and permanence of occlusion. However, because it does not have the dangerous properties of isobutyl-2-cyanoacrylate, such as a high tensile strength, Avacryl is significantly safer to use. This allows greater flexibility in the way the material is delivered.

Cryoprecipitate-thrombin mixtures offer a biologically compatible, easily manageable embolic agent. Our first laboratory studies showed

that (1) the coagulation time and clot density could be controlled by the amount of thrombin added; (2) nonionic contrast agents increased clotting time, whereas ionic agents decreased it; and (3) relatively low concentrations of thrombin could be used that avoided the problems of thrombin excess. We have used this agent in 12 patients, primarily via direct puncture but also transarterially and transvenously. It is well tolerated by the tissues, despite the fact that it causes thrombosis and fibrosis. Direct injection of this material into the vascular lesion can be useful both as a palliative and an adjunctive procedure. The occlusion does not appear to be nearly as dense and permanent as those caused by the acrylates. The high viscosity of the material is a disadvantage in that it cannot be used through present calibrated leak systems. The risk of giving blood products can be alleviated or mitigated by using autologous blood products or products from designated donors. We lack sufficient follow-up to know this agent's role in endovascular treatment, but we are encouraged, particularly in its use for facial arteriovenous malformations.

Lastly, our laboratory studies on the effects of angioplasty on vasospasm have suggested that the highly muscular arteries are paralyzed and the endothelium defunctionalized after angioplasty. The results also suggested that the highly muscular and relatively nonelastic cerebral vessels do not tolerate overdilation as well as the highly elastic systemic vessels that also have the added protection of a thick adventitia.

The concept and use of lasers for treatment of occlusive cerebrovascular disease. Murphy-Chutorian D (Eclipse Surgical Technologies, Inc., Palo Alto, CA 94303).

The object of this presentation is to describe briefly directed laser treatment of peripheral vascular disease and to discuss the limitations that must be overcome to treat cerebrovascular occlusive disease. Also, a new laser delivery system that was designed to overcome many of these obstacles (the Eclipse LaserPlus, patent pending) will be mentioned.

The first percutaneous laser angioplasty trial was performed by Bob Ginsburg at Stanford in 1983. It was concluded that the laser was an excellent cutting tool. However, a laser can remove normal healthy tissue as easily as it removes atherosclerotic plaque. Therefore, it is easy to perforate the wall of the artery with the laser beam. All attempts to control the laser energy by visualization (angiography or fluoroscopy) have been associated with perforations. Two fundamental problems with this approach are reaction time and resolution.

Reaction time is the time required by the physician to squeeze the trigger to start laser firing. Because the lasers and hot tips are so efficient at removing tissue, 0.1 or 0.2 sec is sufficient to cause perforations. The fastest human reaction time required to recognize visually the need to stop firing the laser and to release the trigger is probably at least 0.3 sec. One solution to this disparity is to create an automatic feedback system to accurately control firing of the laser. By using a computer firing mechanism, the laser can be controlled on the order of a few milliseconds.

The thickness or volume of tissue that is directly in front of the fiber and that can be identified correctly is a measurement of resolution. This measurement is critical because the media of vessels is quite thin (0.1 mm) in the cerebral arteries. If the detection or visualization system cannot discern the margin of the media underlying the obstructive plaque or thrombus, then perforation or dissection is likely to occur.

The MCM SmartLaser is the only directed laser system that has been used in the clinical treatment of peripheral vascular disease. It addresses the issue of controlling the laser output by using spectroscopic

copy and a computer to provide automatic, feedback-control firing of the laser. The decision to fire or not fire is made by an algorithm, based on tissue fluorescence, that is stored in the computer. The laser does not fire at intima, media, or blood. All other tissue signals within the artery are considered to be indications of disease, and the computer will allow the system to fire. Improvements to the system over the past 2 years allow actual recognition of different plaque types. Little patient-to-patient variability occurs in spectroscopic signals from normal vessels.

This system was treated in various experiments before clinical trials started. In one, atherosclerotic arteries obtained at autopsy were used, and an optical fiber was placed in the most disadvantageous position, that is, perpendicular to the arterial wall. Then an attempt was made to perforate the artery with the laser. The diagnostic system accurately detected the media, and perforation by the laser was prevented. In the initial series of 70 percutaneous cases, the SmartLaser created a narrow channel in a totally occluded leg artery in 57 (81%). Balloon angioplasty was required to achieve definitive recanalization in all cases. Patency rates at 1 year were approximately 80–85%.

In conclusion, the SmartLaser® appears to open small channels successfully in total occlusions to allow subsequent definitive balloon angioplasty. However, the directed laser requires improvements in delivery systems to achieve definitive recanalization of peripheral arteries. In the cerebral vasculature, percutaneous laser devices must prevent embolic debris, navigate tortuous vascular pathways, and treat the lesion so that balloon angioplasty is not needed.

A new catheter from Eclipse Surgical Technologies, Inc., called the LaserPlus, was designed to treat cerebral arteries without causing emboli. The device uses a new, solid-state mid-infrared laser source. One advantage of the delivery system is a mechanism for widening channels through obstructions while extracting the debris from the circulation. In addition, the device uses low levels of laser energy to decrease the occurrence of thermal damage or vasospasm in the cerebral arteries. Finally, the LaserPlus catheter is trackable over standard 0.014- to 0.018-in. (0.036–0.046 cm) guidewires. Preclinical trials of the LaserPlus delivery system currently are under way.

Proposed standardized scale for clinical neurologic outcome of patients undergoing embolization procedures. Fox AJ (University Hospital, London, Ontario, Canada).

A standard method is suggested for recording information on the neurologic outcome of patients with brain arteriovenous malformations and aneurysms who have embolization procedures. Recent presentations by different groups suggest that different criteria are used to catalog results. For example, a devastated patient who did not get worse from embolization might be classified as an excellent result. Long-term follow-up of such patients is appropriate, and there is a need to document complications of both the treatment and the disease itself. Before treatment, the clinical status of patients varies. They can have clinical worsening from either the disease or its treatment and after initial recovery, can decline in the long term, especially if embolization is somewhat incomplete. A "functional" status scale of patients in follow-up can be used to assess the degree of difficulty patients are having on their own, and this can be scored by a research assistant. In addition, a "stroke severity scale" can be used to evaluate the severity of specific events of ischemia or hemorrhagic stroke. This system, borrowed from the North American Symptomatic Carotid Endarterectomy Trial, can allow various cooperative groups to perform a standard evaluation on patients, especially for long-term follow-up.