The role of mechanical factors in the pathogenesis of short-term and prolonged spasm of the cerebral arteries

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The authors describe fibrous structures they name "chordae" that stabilize the position of arteries in cerebral subarachnoid spaces. The innervation of these structures and their relation to the innervation apparatus of the arterial wall is discussed. Animal experiments and human autopsy material were used to study the role of the stabilizing structures in the pathogenesis of arterial spasm following the rupture of saccular aneurysms. Mechanical stimuli produced short-term but not prolonged arterial spasm.

KEY WORDS cerebrovascular spasm · cerebrospinal fluid · cerebral arteries · subarachnoid space · stabilizing structures · chordae · rupture of aneurysm

JOHNSON, et al., Kapp, et al., and others have suggested that bands of connective tissue between the arterial wall and the arachnoid have a role in the production of spasm of cerebral arteries following rupture of saccular aneurysms. Different investigators have applied a variety of names such as “arachnoid bands,” “fibrous trabeculi,” or “avascular fibers.” We call these structures “chordae.” The concept is that accumulation of blood in the subarachnoid space separates the arteries in the basal cisterns from the adjacent arachnoid and so stretches these fibers. This mechanical irritation in turn affects the arterial wall and thus causes spasm of regional arteries.

Anatomical Studies of Chordae

Chordae Stabilizing the Position Of Arteries in CSF

General. The chordae that connect the arterial wall with the arachnoid are merely a small part of a whole system of poorly understood fibrous structures that stabilize the position of arteries in cerebrospinal fluid (CSF). We have called these purely collagenous structures “chordae” because of their resemblance to the well-known chordae tendinae of the heart valves. The chordae in the CSF are surrounded by endothelial sheaths. Phase contrast microscopy and micromanipulation demonstrated two types of chordae, namely, those capable
or incapable of elongation. In the former, all collagen bundles are arranged longitudinally in a regular wave-like pattern. When such a chorda is stretched, the “spare plicas” of the collagen bundles are spread, and the chorda becomes longer. On the other hand, chordae incapable of elongation contain a rectilinear core of collagen bundles which is surrounded by multiple concentric layers of fine circular collagen fibers. No spare plicas are present, and when such a chorda is stretched, the high degree of elasticity contributed by the collagen prevents elongation and leads rather to kinking and torsion. The fibrous structures that stabilize the position of the arteries in the CSF are composed of quantitatively and spatially varying combinations of both types of chordae. This creates differing degrees of mobility of the arteries connected with the chordae. Thus, the chordae that stabilize major arteries in the basal cisterns, and their distal branches in the channels over the convexity of the hemispheres, permit easy local adaptation to respiratory and pulsatile deformations of the arteries.

We have separated the chordae into three main variations, which are listed diagrammatically in Fig. 1.

Type I Chordae. The position of an artery with reference to the walls of a CSF cavity is stabilized by Type I chordae (Fig. 1 A-C). Fig. 1 A shows a cross section of a CSF channel with an artery located in its lumen. Chordae suspend the artery from the walls of the channel. The inner ends of the chordae are interlaced in the arterial adventitia; the outer ends are fixed to the
wires of the channel. There is a variation of this type in which the chordae form a kind of hammock in which the artery rests (Fig. 1 B); all of the ends of the chordae are fixed in the walls of the channel. Another variation was found for the branches of the inferior posterior cerebellar artery in the cisterna magna (Fig. 1 C). Here the artery appears to be tied to the surface of the cerebellum by short and interanastomosing chordae shaped like brackets; the absence of chordae along the vein located near this artery is noteworthy.

**Type 2 Chordae.** The configuration of the arterial trunk proper is stabilized by Type 2 chordae (Fig. 1D). Here the chordae resemble a bowstring with auxiliary fixation points on the concave side of the artery; apparently they stabilize or fix the shape of the curve in a given part of the artery.

**Type 3 Chordae.** The position of the arterial branches is stabilized by Type 3 chordae (Fig. 1 E-I). There are many queer variations from straight chordae (Fig. 1 E) to a peculiar chorda in the shape of a rein with a loop at the end through which the arterial branch passes (Fig. 1 I). The mobility of an arterial branch stabilized by two chordae (Fig. 1 G) obviously is smaller than when only one chorda is present (Fig. 1 E).

The relatively longer segment of the middle cerebral artery with its branches in the Sylvian fissure deserves special attention. Here the chordae form a united well-developed system (Fig. 1 J) that stabilizes the spatial interrelations of the whole arterial tree. When the position of an arterial branch was changed during micro-manipulations *in situ*, we could observe the strain on relevant chordae, the spreading of spare plicas in collagen bundles, and the subsequent return to their original position. The thinnest chordae of about 25 \( \mu \) in diameter were found in the channels located in the depth of the pia mater close to the cortex of the cerebral hemispheres. These chordae stabilize the position of small arterial branches from which the radial arteries of the brain arise.

Figures 2 through 11 constitute a photographic atlas of actual film preparations showing the various kinds of chordae diagramatically represented in Fig. 1.

**Innervation of Chordae**

"Film" Preparation of Adventitia and Chordae. Innervation of the chordae was studied by a special technique that preserved the natural connections of the chordae with the arterial adventitia. A cerebral artery removed *en bloc* was impregnated with...
silver by the Campos or Gross-Bielschowsky method, cut longitudinally and mounted on glass shaped like a plate; the intimal side was applied to the glass. After dehydration the arterial wall was covered with sufficient thin layers of 2% celloidin solution to form a transparent film. When the film was removed from the glass with forceps all of the adventitia came with it, together with the chordae and other structures, without impairing their natural topographic interrelations. The film was then stained with Erlich hematoxylin and mounted in Canadian balsam.

Fig. 3. Left: “Hammock” type of chordal structure is located in the lumen of a channel that opens into a cistern. Right: Higher power view of same.

Fig. 4. Left: Drawing of a film preparation showing interanastomosing bracket-shaped chordae along a branch of the inferior posterior cerebellar artery in the cisterna magna. Note the lack of chordae along the vein. Arrow points to the site of the actual photograph (right) from which the drawing was developed.
Results. The chordae have their own innervation; silver impregnation reveals receptors and nerve fibers and cells. The nerve fibers, both myelinated and unmyelinated, run along the chordae and become interlaced in the bundles of the external nervous plexus of the arterial adventitia. Among the various types of receptors seen, the commonest are those similar to nerves-tendon spindles (Fig. 8 left). Receptors are located on the surface of the middle broad part of the chordae. The receptor is formed by one or two myelinated fibers which sometimes approach the broad part of the chorda from opposite directions, as for example, from the side of the CSF cavity wall or from the side of the arterial adventitia. Losing myelin, the fibers form terminals ending in numerous leaves, buttons, and loops closely applied to the surface of the chorda. Sometimes nerve cells of a pseudo-unipolar type are found in the chordae (Fig. 8 right). In such cases the common axon may be seen to divide into two fibers diverging along the chorda in opposite directions.

Apart from their own innervation, the chordae establish close spatial relationships with the nerve elements of the arterial adventitia. At the point of fixation to the arterial wall the collagen bundles of the chordae spread in a fan-shaped manner and, interlacing in the adventitia, penetrate and envelop the bundles of adventitial nervous plexuses; collagen bundles also enter the zones of adventitial receptors. This observation of intimate connection of the chordae with the nervous apparatus of the arterial wall is supported by the fact that contemporary electron microscopic and histochemical data have indicated that the nerve elements of the arterial wall occur...
FIG. 6. Left: Chordae from the main arterial trunk stabilizing the position of the arterial branches by fixation to the trunk. Right: A small arterial branch is supported by a loop of chorda. This branch arises from the large arterial trunk that occupies most of the background.

FIG. 7. Complex chordal structure at the site of its fixation to basilar artery adventitia.
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Only in the adventitia, and do not penetrate the media and intima.\textsuperscript{10,13,14,18,24,26–29} There are also close connections between the chordae and the so-called "paravascular" nerves of the cerebral arteries that were described in isolated arteries by Tsvetkov\textsuperscript{25} and Fang.\textsuperscript{16} In our film preparations, in which the arteries are not isolated but are studied microscopically while \textit{in situ} in the CSF space, the peculiarities of these nerves are especially clear. They are rather large nerve trunks that in some sections complete-

Fig. 8. \textit{Left:} Drawing of film preparation showing the receptor on the surface of the broader part of a chorda; note resemblance to a nerve-tendon spindle. A bundle of nerve fibers of the external nervous plexus of arterial adventitia is pictured in the background. Silver impregnation after Campos. \textit{Right:} Drawing of pseudounipolar type nerve cell in the chorda. Impregnation after Gross-Bielschowsky.

Fig. 9. Type 2 chordae. Microscopic photograph of film preparation showing unstained chordae with an axillary core and windings of semitransparent circular fibers. Paravascular nerve trunks, in contrast to the chordae, stain a deep blue. Sudan black.
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FIG. 10. Drawing of film preparation showing spatial interrelations of chordae and paravascular nerve trunks on the surface of the basilar artery. The whole background of the picture is formed by arterial adventitia in which wavy collagen bundles create a characteristic striated pattern. Four cut chordae are seen in the middle of the picture interlacing as they enter the arterial adventitia through queerly curved endings. Thin threads are seen to deviate from the chordae and fix themselves to the epineurium of the paravascular nervous trunks. Some bundles of nerve fibers leave the paravascular nerve trunks and penetrate the adventitia.

ly leave the adventitia and run freely in the CSF at a distance of 1 to 3 mm from the arterial wall. In their CSF course they are supplied by epineurium and are completely surrounded by an endothelial sheath. When stained with Sudan black (Lison-Dagnelli) or with Schiff stain (Feulgen and Voit), the chordae and the paravascular nerves can be reliably differentiated (Fig. 9). Under such conditions, thin threads are seen to separate from the chorda as it approaches the artery; these enter the epineurium of the paravascular trunks. In other words, the chordae stabilize not only the position of the arteries in the CSF, but also that of the paravascular nerve trunks connected with the arteries (Fig. 10).

Comparative Anatomical Studies. All the data presented so far concern the arrangement and innervation of the stabilizing structures in man, in whom these structures are especially well developed. In view of the numerous publications on experimental spasm of the cerebral arteries, it seemed important to study comparable structures in experimental animals. The pattern at the base of the brain is different in dogs and cats. In the area of the anterior and middle cerebral arteries in these animals the arterial wall adheres intimately to the arachnoid. Some parts of the arteries of the circle of Willis are connected with the arachnoid by a thick network of collagen bundles resembling felt surrounding the arteries. Chordae are only found in the area of the basilar and vertebral arteries, and even here are not so well developed as in man. In dogs, chordae with spare plicas prevail, while in cats the chordae have a circular winding. In both species the basilar and vertebral arteries are connected by chordae and a vascular network as well as arachnoid. On the convexity of dog and cat brains the position of the arteries in the channels is also
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Fig. 11. Scheme for spreading of the bloodstream from a ruptured aneurysm through the basal cisterns and CSF channels arising from them. 1 = aneurysm of the anterior communicating artery; 2 = basal cisterns; 3 = CSF channels; 4 = arteries in the lumina of the channels; 5 = chordae stabilizing the position of the arteries; 6 = system of subarachnoid cells. Arrows indicate the direction of the spreading bloodstream.

Discussion

Function of Chordae

The problem of arterial pulsation in a closed skull remained unsolved despite its stabilization by chordae. Although the para-vascular nerves in these animals are much thinner than in man, they are located outside the adventitia in the CSF and are closely adjacent to the chordae.
three centuries of recognition. Recent physiological studies, especially the data of Moscalenko, who employed the highly sensitive method of electroplethysmography, do provide convincing evidence. Our laboratory observations of the chordae were deliberately conducted through the open skulls of dogs and cats, a condition in which this pulsation is sharply increased. When the arachnoid over the hemispheric convexity is exposed, the CSF channels with their enclosed arteries can be clearly seen. The arteries branch within the channels, so that any given channel section may contain several arterial branches. The white chordae that suspend the artery from the walls of the channel or connect different arterial branches are easily identified through the transparent arachnoid. Neurosurgeons are familiar with physiological changes of the arterial diameter and the bending, torsion, or displacement of the arterial branches. Sometimes, in a short segment, the neighboring branches converge and even wind one around the other. The amplitude of these arterial movements rises significantly during forced respiration. Immediately after the cessation of the deformation the arterial branches may return completely to their initial normal shape and position. The elastic properties of the arterial wall undoubtedly contribute to this arterial adaptability. Our study of dynamic changes in the chordae of living animals shows that these structures also provide stabilization in the normal position of the whole arterial branching system in the CSF cavities, limit physiological deformation, and help return the system to normal.

These observations were especially vivid along the base of the brain where the stabilizing structures are most highly developed. Following occipital bone resection in cats the dura could be removed in such a way as to leave the underlying arachnoid intact. In dogs the dura is so firmly adherent to the arachnoid that preservation of the latter is impractical. The physiological deformations of the basilar and vertebral arteries, their branches, and their arterial chordae were clearly seen through the arachnoid distended by the CSF in the cisterns. In this case the changes of the interposition of the arterial branches and their shifts with reference to the walls of the CSF cavities were also observed. Thanks to the reflection of light by the collagen chordae bundles, the spare plicas of these bundles could be seen spreading during the straining of the chordae and returning to normal as the tension was relaxed.

It was peculiar that only some of the chordae were under strain during the deformation of an artery, while the rest remained relaxed.

Thus we now know that arteries do not float freely in the CSF, but shift only to a degree permitted by the stabilizing structures. The existence of such structures along the arteries and their absence along the veins provide important morphological arguments in favor of the modern concept of the pulsation of cerebral arteries in the closed skull.

**Innervation of Chordae**

We can only hypothesize regarding the significance of the innervation apparatus serving these fibrous structures and the related arteries. In the cisterns and branching channels the chordae are placed transversely to the CSF flow, and thus experience changing CSF pressure gradients. The chordae immediately elongate or shrink in response to these stimuli. Since the receptors found on the chordae are, like the nerve-tendon spindles, typical "mechanicoreceptors" in their structure, we must assume that deformation of the chordae stimulates the receptors located on them. Despite the morphologically proven connection between the chordal nerve fibers and the adventitial nerve plexuses, the role of the former in the vasomotor innervation of the arterial wall still remains obscure. At present it seems possible but not proven that the nerve cells of the chordae, like the nerve cells of the arterial wall, participate in local regulation of the size of the arterial lumen.

**Short-Term Vasospasm**

As we have previously demonstrated, the blood from a ruptured aneurysm spreads under high pressure to the cisterns, and for a long distance through channels surrounding the arteries (Fig. 11). The rupture of the aneurysm wall as such is hardly the cause of...
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arterial spasm since we know that the thinned “dome” of the aneurysm, where the rupture usually occurs, contains few of the muscle and nerve elements that might contribute to the development of arterial spasm. We therefore assume that the hemodynamic impact of the bloodstream causes spasm by its effect on the relatively normal walls of arteries located near the aneurysm rather than on the aneurysm itself. The primary targets of this impact are the inflammatory arachnoidal adhesions that connect the aneurysm with the adjacent arterial segments. The chordae and paravascular nerve trunks we have described are buried in these adhesions. Gillingham has reported the considerable power of the blood jet from a ruptured aneurysm that tears the surrounding arachnoidal adhesions. Rupture of the adhesions must be accompanied by severe mechanical irritation and disruption of the chordae and paravascular nerve trunks involved. Zlotnik has attributed the development of spasm during surgical manipulation of the adhesions surrounding an aneurysm to mechanical irritation of the chordae crossing the cisterns and attaching to paravascular channels (Fig. 11). The chordae respond by undergoing strain, deformation, and rupture.

To study the role of the chordae in the development of arterial spasm, feline and canine experiments were conducted with direct mechanical irritation of the chordae stabilizing the position of the basilar artery. Under stereoscopic microscope control (∗50) a thin needle was introduced into the subarachnoid space; this action produced no arterial spasm. However, during micro-manipulation of the needle among a group of chordae, or on a single chorda, without touching the arterial wall, we invariably observed the development of arterial spasm. The diameter of the blood jet decreased by 40% to 60%, and at the same time, the thickened whitish arterial walls became clearly visible. During mechanical irritation of a single chorda we noted the peculiar picture of an “asymmetrical” arterial spasm, i.e., thickening of only part of the circumference of the arterial wall in the zone of this chorda’s fixation. Similar results were obtained in cases in which the chordae were irritated by the tip of a vibrator. These experiments justify the postulate that when a certain intensity of deformation and straining of the chordae occurs, short-term spasm of the corresponding arteries always develops. This spasm, caused by mechanical irritation of the chordae, seldom lasts longer than 20 minutes.

It is also clear that a sufficiently powerful bloodstream flowing out of a ruptured aneurysm may cause spasm of the adjacent arterial segments by direct mechanical irritation of their walls. We directed a thin jet of normal saline solution maintained at body temperature and pressurized approximately to the level of canine carotid artery pressure (2200 mm H₂O) against the walls of exposed basilar and vertebral arteries in dogs. Arterial spasm was observed in only three of the eight animals and even these showed no more than 20% to 30% constriction. This was invariably less than the spasm produced by mechanical irritation of the chordae; moreover, the spasm caused by the hydrodynamic effect was of no more than 5 min duration.

Other natural causes of short-term spasm following rupture of an aneurysm include the tension of the arterial wall itself, displacement of the artery by a hematoma, and torsion of the artery near a blood clot.

Artificial mechanical irritation of normal arteries under a variety of experimental and operative conditions usually produces a short-term local arterial spasm.

Summarizing the above data, we conclude that under both experimental and natural conditions, mechanical stimuli are capable of producing only short-term spasm. It should be admitted that short-term acute arterial spasm developing immediately after the rupture of an aneurysm is also conditioned by chemical factors, which also act briefly (the Echlin factor, serotonin, and other spasmogenic blood substances). An analysis of the effect of these substances was not considered pertinent to our investigation.

Prolonged Vasospasm

Mechanical stimuli seem to have nothing to do with the still mysterious pathogenesis.
of chronic spasm lasting many days; chronic cerebral vasospasm, however, is of major clinical significance.\textsuperscript{1,2,6}

In a recent experimental study on monkeys, Simeone, \textit{et al.},\textsuperscript{3}\textsuperscript{a} punctured a major artery at the base of the brain and observed angiographically the development of a severe diffuse arterial spasm that persisted for 4 days. They concluded that such a penetrating injury of the arterial wall can by itself produce prolonged arterial spasm. Still, as the authors indicated, the arterial puncture was accompanied by some degree of subarachnoid hemorrhage (extravasation of the contrast medium from the site of the puncture, as seen in angiograms and autopsy data). This means that the effect of the spasmogenic substances of the blood was not excluded. Moreover, an arterial puncture can hardly be compared with rupture of an aneurysm whose pathologically altered walls differ greatly from the walls of a normal artery.

Landau and Ransohoff\textsuperscript{23} also believed that in monkeys mechanical irritation alone could produce prolonged arterial spasm; the injury of major arteries at the base of the brain by means of needle punctures or cross-clamping resulted in an angiographically demonstrated spasm that persisted for 5 days. However, the authors themselves noted that the irritation was not purely mechanical; both means of arterial damage were often accompanied by subarachnoid hemorrhage. Fortuna and La Torre\textsuperscript{17} in another angiographic study divided a branch of the basilar artery in rabbits; the artery developed a prolonged spasm which the authors interpreted as a manifestation of the peculiarities of the spasmogenic mechanical injury of the arterial wall. The importance currently attached to the mechanical factor in the pathogenesis of the prolonged spasm was indicated by Suwanwela and Suwanwela,\textsuperscript{34} who studied the angiographic development of spasm in man following skull-brain injuries. No arterial spasm was found on the angiograms of the majority of patients with traumatic subarachnoid hemorrhages.

On the other hand, Roy, \textit{et al.},\textsuperscript{32} in their monkey experiments with repeated angiography failed to produce a prolonged arterial spasm after puncture of the intracranial internal carotid artery; nor did spasm develop after repeated puncture of this artery, despite a 2-week period of observation.

We believe the unconvincing nature of these angiographic studies is connected with such experimental factors as the difficulty of a nonsanguineous frontotemporal approach to the major basal arteries of the brain, failure to differentiate in these experiments between the effect of mechanical and chemical stimuli, and incomplete opacification of many small basal arteries.

In this connection, the elegant experiments of Echlin\textsuperscript{11} are remarkable. Using a nonsanguineous method he exposed the basilar and vertebral arteries of monkeys and punctured the basilar artery with a needle which he then left in place; 1\textsuperscript{1/2} min later severe local arterial spasm developed at the puncture site, and disappeared almost completely in 20 min. In other words, it was a short-term spasm.

Our animal experiments on the role of mechanical irritation in the pathogenesis of prolonged arterial spasm were conducted in cats and dogs. We also exposed the basilar and vertebral arteries by a nonsanguineous method and punctured the media and adventitia of the basilar artery tangentially without penetration of its lumen. This method excluded any possibility of a subarachnoid hemorrhage. The results of the experiments fully supported Echlin's data. At the site of puncture of the artery a local short-term spasm developed but lasted no more than 30 min. Another series of experiments was conducted on the branches of the middle cerebral artery located on the convexity of the hemisphere. We had previously demonstrated that in contrast to the major basal arteries these vessels show little if any spasm in reaction to autogenous blood on the adventitia.\textsuperscript{4} The puncture of such an artery and the subsequent removal of the needle resulted in a brief subarachnoid hemorrhage. At the puncture site a severe local arterial spasm developed, which was evidently caused by the mechanical factor of the puncture. The spasm was short-lived and disappeared some 20 to 30 min later. The third series of experiments was conducted on small branches arising from the basilar and vertebral arteries at
the base of the brain, and from the middle cerebral artery over the convexity. When these branches were intentionally torn from the arterial trunks, significant subarachnoid hemorrhage often developed, and at the site of the rupture a severe arterial spasm was observed. But this spasm was also a short-lived one, lasting as a rule not more than 1½ hours. In none of the other experiments in these three series, even with direct visual observation for up to 9 hours, did we note transformation of a short-term into a prolonged arterial spasm.

We thus arrived at the conclusion that mechanical irritation cannot be considered a direct cause of prolonged arterial spasm. The suggestion has been made that some tissue decay substance is formed in response to mechanical irritation of the arterial wall, and later participates directly or indirectly in the chemical stimulation of arterial spasm. This requires further investigation.

Summary

The position of arteries bathed by cerebrospinal fluid (CSF) is stabilized by fibrous structures composed of "chordae." These structures control the position of the artery against the walls of the CSF cavity, the configuration of the arterial trunk itself, and the position of branches arising from an arterial trunk within the CSF space. In the process of physiological deformations (pulsatile, respiratory, or muscular) the arteries shift in the CSF within the spatial limits permitted by the stabilizing structures. Surrounding the chordae are nerve fibers, receptors similar to nerve-tendon spindles, and pseudounipolar nerve cells. The chordae are also closely related to the nerve elements of the arterial adventitia and the so-called "paravascular" nerve trunks that run freely in the CSF in some segments. In canine and feline experiments, when the chordae that stabilize the position of the basilar artery were irritated mechanically, spasm of this artery was produced. It can be assumed that the hydrodynamic impact of the blood jet streaming from a ruptured aneurysm affects primarily the inflammatory adhesions that connect the aneurysm with the adjacent arterial segments; as shown histologically, the chordae and paravascular nerve trunks are located in the depth of these adhesions. Spreading beyond the limits of the adhesions the blood jet produces a similar mechanical irritation of the chordae that cross the lumina of the cisterns and channels. Thus, under both natural and experimental conditions, mechanical irritation was capable of producing short-term but not prolonged arterial spasm of cerebral arteries.

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