SURGERY OF THE AUTONOMIC NERVOUS SYSTEM

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WALTER CANNON,[1] in 1929, coined the term "homeostasis" to describe the autonomic mechanism by which the fluid matrix of the mammalian body is maintained in a constant state despite changes in external environment. This is accomplished by the balanced opposition of the sympathetic and parasympathetic divisions of the autonomic nervous system. The sympathetic division provides an emergency protective mechanism which is always ready to go into action to combat any variety of adverse circumstances. When called into action, the sympathetic division uses up the bodily reserves in order to give rise to an increased liberation of body energy and the effects, therefore, are catabolic in character. Some of the more common conditions which arouse it to activity are pain, hemorrhage, infection, asphyxia, extremes in temperature, and any form of intense emotion. The catabolic functions of the sympathetic system are balanced by the anabolic functions of the craniosacral or parasympathetic division which come into ascendancy during periods of rest and recuperation, and are of a conservative character. When one of these systems is excited, the other is inhibited.

However, this normally efficient homeostatic balance may break down occasionally, resulting in pathologic states. When this occurs the surgeon may be called upon to alleviate the situation by interrupting the flow of impulses responsible for the abnormal state. A list of such pathologic conditions follows:

1. Arterial wounds
2. Arterial embolus
3. Arterial thrombosis
4. Arteriosclerosis obliterans
5. Thromboangiitis obliterans
6. Thrombophlebitis
7. Raynaud's disease
8. Hypertension
9. Hyperidrosis
10. Causalgia
11. Peptic ulcer
12. Carotid sinus syndrome

It is apparent that all but two or three of these pathologic states have to do with a disturbance in the circulatory system. This is not surprising inasmuch as maintenance of circulation is the most obvious function of the autonomic nervous system. But what is the explanation for the disturbed physiology which accompanies these pathologic states? I believe that the answers can be found by studying the normal physiologic response to bleeding.

When an experimental animal is bled by means of a cannula placed in an

artery, there results a progressive reduction in the volume of the circulating blood. The blood pressure, however, does not fall immediately as one would expect, but is maintained at its original pressure by a contraction of the vascular bed. This contraction is occasioned by an increased rate of discharge of vasoconstrictor impulses over the sympathetic division. After perhaps 40 per cent of the animal's blood has escaped, the capacity of this sympathetic vasoconstrictor mechanism is exceeded, and a fall in blood pressure occurs. About this time, however, a vasoconstrictor substance appears in the circulating blood.[2] Thus a humoral mechanism comes to the aid of the overtaxed sympathetic division. By enhancing its vasoconstrictor effect, the fall in blood pressure is combated.

In order to reduce the rate of blood loss, nature has arranged that this vasoconstriction which accompanies blood loss is more pronounced in the wounded limb and especially so in the bleeding vessel itself. The vasoconstriction in the involved limb may be so great as to suggest that nature is willing to sacrifice the injured extremity in an effort to preserve the life of the organism.

This vasoconstrictor response to bleeding has been developed by nature because the chief threat to life in animals in the wild state is wounds of blood vessels. Because this is the case it is not surprising to find that this same phylogenetic response of vasoconstriction occurs as a result of other types of vascular insult.

For example, Villaret and Cachera[3] in 1939 demonstrated that the immediate reaction of the pial vessels to solid emboli was widespread vascular spasm. They found that this spasm was just as pronounced in fields where there were no emboli, indicating its reflex nature. These spastic phenomena were intermittent and constantly recurring, and were seen months after embolization. Spasm and dilatation were found to coexist in parts of the same vessel, and changes in calibre might occur over and over again within a few minutes. The dilated portions of vessels contained cyanotic blood, indicating stasis and hypoxia. They observed that the vessels affected by the disturbance in vasomotricity need not belong to the same parent vessel. They also observed that the venous effects were more widespread than the arterial.

Because of the protective mechanisms with which man has surrounded himself, he has lengthened his life to a point where the so-called degenerative diseases are prevalent. The chief of these is arteriosclerosis. When arteriosclerosis results in the occlusion of one of the major vessels in the lower limb, there occurs this phylogenetic response to vascular insult: i. e. vasoconstriction. The limb, therefore, is deprived not only of the volume of blood which would ordinarily pass through the obstructed vessel, but the volume of blood which passes through the collaterals is reduced by the accompanying vasospasm. The same is true also in varying degree in cases of arterial embolus, thrombosis, thromboangiitis obliterans, and thrombophlebitis.

Kuntz[4] has described the anatomic pathways involved in the neurogenic vasoconstriction which occurs in a limb which is the seat of a vascular lesion. He has demonstrated in the blood vessel walls afferent fibers which transverse

the sympathetic trunk and enter the spinal cord through the dorsal roots of the nerves which convey the corresponding efferent fibers. He states that these afferent nerve fibers which reach the extremity through the sympathetic trunk not only conduct impulses which result in painful sensations, but also impulses which reflexly activate the sympathetic nerves to the extremity. Such reflex stimulation tends to increase vasomotor tonus in the extremity and thereby aggravate the pain. Sympathectomy in the treatment of patients in this category not only interrupts the afferent nerve fibers through which the pain is mediated but also abolishes the reflex vasomotor tonus, thus insuring improvement in the circulation of the limb.

In cases of vascular occlusion it would appear that this reflex arc acts, at times, like a reverberating circuit. The afferent painful impulses initiate vasoconstriction; this in turn, increases the pain which then reflexly aggravates the vasoconstriction.[5] The effects of sympathetic nerve block in various pathologic states may persist far longer than one could explain on the effect of procaine, per se. This is perhaps due to the interruption of this reverberating circuit which then may take many hours or days to return to its former pathologic rate of discharge.

Leriche,[6] as long ago as 1917, showed that when excision of the obstructed segment of an artery is carried out, certain extremities often lose their trophic changes and become more comfortable. This operation of arteriectomy is regaining favor with some surgeons.[7] The improvement in vasoconstriction in the affected limb, which sometimes follows this operation, is undoubtedly due to partial interruption of the reflex arc by elimination of some of the afferent fibers in the blood vessel wall. However, there are relatively few who believe that the results of arteriectomy are as satisfactory as those obtained by paravertebral sympathectomy. When one considers how much more completely the reflex arc is interrupted by paravertebral sympathectomy than by arteriectomy, the reasons for the superior results of sympathectomy become evident.

At the present time nearly all operations on the sympathetic system are directed toward the paravertebral portion of the system which comprises the ganglionated cords. Anterior rhizotomy, periarterial sympathectomy, and division of peripheral nerves, have been largely discarded.

Surgical procedures on the autonomic nervous system advocated at the present time are indicated in the accompanying table.

Sympathetic Block

Cervical sympathetic block appears to be of benefit in certain cases of cerebral embolus, cerebral thrombosis, and cerebral trauma.[8] When one wishes to interrupt the sympathetic outflow to the brain, it can be accomplished satisfactorily by injection well cephalad to the stellate ganglion. Here the injection is far simpler and there is no danger of puncturing the pleura, a risk occasionally encountered with a stellate ganglion block.

A procaine block of the stellate ganglion is chiefly employed in order to

Table

PROCAINE BLOCK	
Cervical sympathetic	—Embolus, thrombosis and trauma of the brain.
Stellate ganglion	—Arterial wounds, embolus, thrombosis, causalgia in upper extremity.
Upper thoracic sympathetic (alc.)	—Angina pectoris.
Splanchnic nerve	—Painful conditions within abdomen.
Lumbar sympathetic	—Arterial wounds, embolus, thrombosis, arteriosclerosis obliterans, Buerger's disease, causalgia, thrombophlebitis in the lower extremity.
Caudal (epidural)	—Same as lumbar.
Spinal (subarachnoid)	—Same as lumbar.
PARAVERTEBRAL SYMPATHECTOMY	
Cervical	—Cerebral vascular disease— (effectiveness still to be proved.)
Upper thoracic	—Arterial wounds, embolus, thrombosis, causalgia, Raynaud's disease, hyperidrosis of the upper extremity, angina pectoris.
Supradiaphragmatic (splanchnicectomy)	—Hypertension. Pain in abdomen. (Peet)
Subdiaphragmatic (splanchnicectomy)	—Hypertension. (Adson)
Thoracolumbar	—Hypertension. (Smithwick)
Complete	—Hypertension. (Grimson)
Lumbar	—Arterial wounds, embolus, thrombosis, causalgia, Buerger's disease, arteriosclerosis obliterans of the lower extremity.
VAGUS NERVE RESECTION	—Peptic ulcer.
GLOSSOPHARYNGEAL NERVE RESECTION	—Carotid sinus syndrome.

determine the degree of improvement which one may anticipate from sympathectomy in conditions of the upper extremity. The usual indications are arterial embolus, thrombosis, and causalgia. Alcohol injection of the upper five thoracic sympathetic ganglia has been advocated by White[9] for the relief of angina pectoris. The number of injections required, and the duration of the postinjection pain in these cases, somewhat discourage the widespread acceptance of this technic.

Procaine injection of the splanchnic nerves causes a temporary interruption of the visceral sensory fibers contained in these nerves. This injection is, therefore, sometimes used in order to determine whether one may anticipate relief of intra-abdominal pain by division of the splanchnic nerves.

Procaine block of the lumbar portion of the paravertebral ganglionic chain is used chiefly as a temporizing measure, and in order to determine the benefits

to be anticipated by lumbar sympathectomy in such conditions in the lower extremities as embolus, thrombosis, arteriosclerosis obliterans, Buerger's disease, causalgia, and thrombophlebitis.

Caudal epidural or the lumbar subarachnoid injection of the anesthetic agent is sometimes used instead of a lumbar sympathetic block. This method of administration of the anesthetic agent, provided that it diffuses far enough cephalad, has the advantage that it produces an interruption of the sympathetic impulses to both lower extremities. However, neither of these latter procedures is as selective as a lumbar sympathetic block, inasmuch as they interrupt all of the sensory nerves from the lower extremities and not merely those which traverse the lumbar sympathetic chain.

Operative Procedures

Excision of the cervical sympathetic chain is being employed in cases of cerebral vascular disease. The effectiveness of this procedure, however, is yet to be proved.

When sympathetic denervation of the upper extremity is indicated because of diminished circulation due to arterial wounds, embolus, or thrombosis, or in cases of causalgia, Raynaud's disease, and hyperidrosis, the procedure most commonly employed is a modification of Smithwick's upper thoracic preganglionic sympathectomy.[10] If sympathectomy is to be performed for the relief of angina pectoris, a more extensive procedure is indicated, such as the excision of the stellate and the four upper thoracic sympathetic ganglia as advocated by Lindgren and Olivecrona.[11]

For the relief of painful conditions within the abdomen, the supradiaphragmatic sympathectomy, sometimes called splanchnicectomy devised by Peet, is preferred.[12] The effectiveness of this procedure in the pain of pancreatitis is described by deTakats.[13] In this case the aim of the surgeon is to interrupt the visceral afferent fibers which traverse the sympathetic system. The interruption of the sympathetic fibers is merely incidental to the accomplishment of this object.

In the treatment of hypertension the four most commonly employed operations are the supradiaphragmatic sympathectomy advocated by Peet, the subdiaphragmatic sympathectomy advocated by Adson, the thoracolumbar sympathectomy advocated by Smithwick[10] and the so-called complete sympathectomy described by Grimson.[14] Considering the morbidity involved in the more extensive procedures, it is my opinion that the supradiaphragmatic sympathectomy of Peet is probably preferable. However, the effectiveness of some of the newer sympathetic blocking agents will probably relegate the surgery of hypertension into discard.

At the present time the most commonly employed operative procedure on the sympathetic nervous system is lumbar sympathectomy. It is indicated in cases of deficient circulation in the lower extremity caused by arterial wounds, embolus, thrombosis, Buerger's disease, arteriosclerosis, and in cases of traumatic causalgia. An occasional case of Raynaud's disease may affect the lower ex-

tremities to the extent that lumbar sympathectomy is indicated. The benefit following lumbar sympathectomy is due not only to the interruption of the efferent stream of sympathetic impulses but also, in part, to the interruption of the afferent painful pulses which traverse the lumbar sympathetic chain and serve to accelerate the rate of discharge of vasoconstrictor impulses. The frequency of vascular disease in the lower as compared with the upper extremities is due to the erect posture which man has assumed and for which nature has not as yet made a satisfactory adjustment.

There are various theories as to why the syndrome of causalgia is frequently relieved by sympathectomy. The explanation I prefer is that offered by Doupe, Cullen, and Chance[15] who believe that afferent discharges over the sympathetic pathways traverse the "artificial synapse" at the site of the nerve injury, and in this way produce impulses in afferent nerves resulting in a sensation of pain. Their work was based on the earlier experiments of Granit, Leksell, and Skoglund,[16] in 1944, who demonstrated by study of the nerve action currents that an artificial synapse is produced in a mixed nerve by injury or pressure. It is, therefore, easy to see how a continuous flow of efferent sympathetic impulses going down a mixed nerve could become short circuited at the point of injury and jump across to the naked axis cylinders of the afferent nerves with the production of a pain impulse.

Operations for the relief of conditions produced by overactivity of the parasympathetic nervous system are vagus nerve resection for peptic ulcer, as advocated by Dragstedt,[17] and resection of the carotid sinus nerve or, more effectively, by resection of the glossopharyngeal nerve intracranially, as advocated by Bronson Ray[18] for the relief of carotid sinus syncope.

Surgery of the sympathetic nervous system has progressed through many phases and it seems likely that the day will come when surgery will no longer be required in the management of these physiologic disturbances.

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