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SOME RESEARCHES ON THE PERI-ARTERIAL SYMPATHETICS*

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THE sympathetic nervous plexuses included in the external layer of blood-vessels seem to possess a real autonomy. The systematic study of the phenomena which follow the exciting of average-sized arteries, that is, of prevailing muscular structure, reveals the existence of a very characteristic physiological reaction which never fails in normal circumstances.

A knowledge of the constituents of this reaction is quite interesting from the triple point of view of physiology, pathology and therapeutics; it sets new problems in physiology; it permits us to explain certain pathological phenomena; finally, it authorizes some promising therapeutic attempts.

I feel the best way I can express my thanks for the great honor the American Surgical Association has done me in inviting me to take part in their work is to present to it the results of my researches on this subject, these last few years.

I. When you remove the sheath of an artery, the brachial artery for instance, you see, just at the moment its external layer is pinched, the vessel contracts, its pulsation stops at once and its size diminishes. If you excise the cellular layer the diminution will progressively reach the third or the fourth of the normal size of the artery. The segments on both sides maintain their normal size provided the operation has not injured them.

This arterial contraction usually causes the pulse to disappear, but it does not altogether interrupt the circulation.

If the artery is cut a thin thread of blood is seen inside; on the other hand, if during the experiment capillaries are examined by Weiss's method, you will see, at the moment the arterial contraction takes place, the capillary loop diminish regularly in its whole length, pale but remaining visible. If the artery is tied the loop is almost indistinguishable, though a little blood still passes (Fig. 1).

The arterial contraction is the primary element of the characteristic physiological reaction against excitation (Fig. 2).

SECONDARY SIGNS.—In the following hours pulsation is imperceptible or very feeble; the operated limb is colder than the other; there is a difference

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of 3° or 4° C. After several hours, varying from three to fifteen, secondary signs appear which form the next element of the characteristic reaction.

They never fail when the excision of the nervous layer is really performed; if they are wanting, it is due to the incompleteness of the nerve operation. Out of sixty-four operations only one failed; in this case, one of my first, the artery was enclosed in fibrous tissue and did not contract during dissection; thus I had not touched its nervous sheath. The secondary signs are the following:

1. An elevation of local temperature reaching 2° and even 3° C., central temperature not being modified. The patient has a subjective sensation of heat. One of my patients complained the day after operation of "a hell fire" in his hand.

2. An elevation of the arterial pressure, which may reach 4 c.c. of mercury according to the healthy side. Claude Bernard had already noted the same figures when measuring arterial pressure after section of the sympathetic in the neck.

FIG 1.—(1) Aspect of a normal capillary loop in a finger, examined by Weiss' method; (2) diminution of size when the brachial peri-arterial nerve is excited; (3) almost complete disappearance after ligation of the brachial artery.

3. An increasing amplitude of oscillations, shown by the sphygmomanometer.

These statements have been verified on my patients by my friend, Doctor Heitz, one of the most eminent cardiologists of France.

The vasodilator reaction is transitory, after peri-arterial sympathectomy it becomes attenuated from the fifth to the sixth day and disappears after three or four weeks.

Such is the characteristic physiological syndrome of the peri-arterial sympathetics against excitation.

II. Pathologically, this excitation may be provoked as well on visceral arteries as on arteries of limbs, and this by direct traumatic causes or indirect infectious or toxic causes.

Whatever may be the cause, as soon as the peri-arterial sympathetic plexus is injured the physiological reaction appears, in exactly the same way the oculopupillar reaction follows the injury of the cervical sympathetic plexus.

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At the level of the viscera we know absolutely nothing of these reactions; we may suppose that ischaemia and prolonged vasodilatation with consecutive elevated blood-pressure may disturb the ductless glands for instance, the thyroid or pancreas, but we have no positive experience concerning the subject. The only experimental fact I know is the following: In a case of diffuse oedematous thyroid hypertrophy a unilateral high perithyroidal sympathectomy caused an extreme diminution of size of the corresponding lobe in the next few days. That is a little thing but it allows one to believe there is a very interesting field of future research.

FIG. 2.—(1) Sudden lowering of the arterial pressure immediately after excitation of the peri-arterial sympathetic; (2) rise of the arterial pressure following the preceding phases.

Finally it is only at the level of limbs that we begin to distinguish the phenomena resulting from an injury of the peri-arterial sympathetic plexus, and their analysis is not easy.

To expose them as clearly as possible one may isolate two groups: In the first group the characteristic physiological reaction is pure, with two striking aspects; in the second case, reaction is disturbed and gives various troubles.

First Type. One may choose two characteristic examples, "stupeur des artères" and Raynaud's disease. I will not insist on the well-known syndrome of "stupeur artérielle." It is an active secondary spasm due to sud-

den excitation of the external arterial layer; a woundless traumatism may produce it as well as a projectile. We have noticed it after violent shock of the humerus, the arm being limp and lifeless, the fingers colorless; one might have thought the brachial artery was crushed. There was really no injury and these phenomena were temporary.

Against Kuttner's opinion I still believe that this contraction is of nervous origin instead of muscular origin, and this for reasons I do not want to develop here.

I want only to make you notice that, in certain cases, the contracture may be so intense as to lead to gangrene from insufficient circulation. This led during the war to unnecessary amputations.

Raynaud's symptoms are typical of vasomotor sympathetic disease. In certain cases of the peri-arterial sympathetics of the whole limb seem to be excited, for the vascular contraction involves even the capillaries, producing deformations that may be seen on the living limb.

This may be observed surgically when the sympathetical irritation is manifest; for instance, in the case of cervical rib or in certain subclavian obliterations (Fig. 4). In these cases the crisis has the exact figure of the physiological reaction with its two characteristic phases, painful ischæmia and consecutive vasodilatation.

Second Type. In many circumstances the initial cause is less known and the physiological reaction is disturbed; either by contracture of too long duration or by abnormally persisting dilatation.

In all cases it produces considerable biological disturbances in the sub-jacent tissues, creating thus various associations of motor, sensitive, vasomotor, glandular and trophic symptoms, or even signs of local necrosis.

Concerning sensibility there may be no systematized hypoæsthesia, but principally painful causalgic phenomena. These troubles are not provoked by an injury of sensitive nerves but begin at the level of the tactile apparatus. The Meissner's, Paccini's and Ruffini's corpuscles are surrounded by a rich vascular network, insuring the perfection of sensations.

Supposing the vascularization disturbed by inflammation, the nervous apparatus suffers at first contact. The sympathetic injury producing peripheral vasodilatation has the identical effects: thus may be explained the acute pains in Weir Mitchell's causalgia and certain painful stumps.

By a less-known process, injury of the peri-arterial sympathetics may provoke motor troubles, either stiffness and contracture, a kind of hypertony localized to certain muscular groups, or a rapid atrophy.

One has noted in "stupeur arterielle" immediate muscular rigidity. The same contracture was also frequently noticed after war wounds and described under the name of reflex troubles by Babinski and Froment.

In these cases when acting on the peri-arterial sympathetics one has seen motor troubles diminish or even disappear as if the contraction of stripped muscles were dependent on the sympatheticus (Fig. 6).

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One may believe muscular symptoms to be but consequences of defective circulation and that sympathectomy acted in modifying it; but one must not forget that the histologists admit for the muscles both a myelinic and amyelinic innervation. The influence of the sympathetics on the muscular

FIG. 3.—Figures showing the capillary loop in a finger and its different sizes before and after Raynaud's disease. (1) Normal; (2 and 3) contracture irrégulière; (4) surdilatation après la cuire.

tonus was shown by De Boer, and the modern neurologists seem to believe that the tonus of muscles at rest is conditioned in invertebrates by the sympathetic system. The presence of motor phenomena is thus easily explained.

In association one frequently notices changes in the sudorific function, profuse sweating or absolute dryness of the skin, which form a constant symptom of the peripheric feature. As in causalgia, one frequently sees

trophic troubles concerning nails, hair or skin and even spontaneous blisters.

Sometimes repeated pressure produces sloughs on limbs whose sympathetic functions are disturbed by a traumatism or amputation.

Other vasomotor phenomena are cyanosis, local blue cedema or large white cedema reaching the origin of the limb without any signs of phlebitis or arterial obliteration. Pains and muscular atrophy may accompany these

symptoms as well after deep contusions of the tip of the limb as in small traumatisms.

All these apparently different symptoms have one common point: they are but the manifestation of a circulatory change, vessels being not injured, none of the apparatus concerning them is touched, nor the sensory, the muscular systems, or glands or skin. And as all are more or less connected, there is evidently a common origin.

This may not be found in the cerebrospinal nervous system as there is no systematized topography. It is neither in the vascular apparatus which is anatomically healthy; operations on the vasomotor system seem to prove that their origin is to be found in the sympathetic innervation. In some cases

there is anatomopathological proof: The artery holds more than usually to the common sheath of the vessels by sclerous adhesions, and the external layer of the artery is more vascularized. The artery presents a particular aspect, being striped in a parallel way by very fine vessels. I have found these lesions with paroxysmal pains in chronic arteritis, in painful stumps and in trophic troubles. But one must acknowledge that in most cases one cannot find any anatomical change explaining the vasomotor ataxy, for our knowledge on the anatomopathological features is still very imperfect.

Another explanation of the syndrome might be given: Sometimes when there is a defective vasomotor innervation the causal injury (bruise, fracture, scar) is often distant from the vessels, at the periphery, in a richly furnished zone of sensitive innervation and the vasomotor disturbances seem due to an ortho- or antidromic reflex, starting from the injured point, the way back being the peri-arterial sympathetics.

This is explained by the fact that at the beginning of the accidents the sympathetic irritation may be sufficiently intense to bring forth spasm and fatal ischæmia in limited parts of the tissues. Thus are produced the local gangrene of Raynaud's disease or after certain prolonged pressure Volkmann's syndrome symptomatic of local muscular necrosis.

Is it not the proof that modified circulation is the real cause of all the phenomena studied above?

III. This leads one to believe that logically the treatment of those vasomotor or trophic troubles must aim to modify peripheric circulation by peri-arterial sympathectomy. The technic is to isolate the artery on 8 to 10 cm., to fix the vascular sheath after dividing, to hold one part with a forceps and dissect off the cellular tissue either with knife or cannular sound until there has been effected complete denudation of the vessel, which becomes greatly reduced in size. This brings no injury to the arterial wall. I verified this fact twice at four and eight months' interval.

I have performed this operation sixty-four times: In eleven cases of causalgia or equivalent syndromes; two cases of painful stumps; nineteen cases of post-traumatic contractures; four cases of large post-traumatic œdemas; one case of trophœdema; four cases of ischæmic sequels; one case of trophic sloughs on a stump; ten cases of trophic sloughs after nervous section; one case of trophic slough of the heel after medullary injury; one case of varicose eczema; one case of trophic troubles after frostbite; one case of spasmodic paralysis; three cases of an attempt to modify tension of cerebrospinal fluid; two cases of Jackson's epilepsy; one case of goiter; one case of intermittent claudication; one case of erythromelalgia.

I obtained remarkable successes and had complete failures. Operation failed in the case of intermittent claudication; in one case of trophic troubles after frostbite, in one case of spasmodic paralysis, and in certain cases of painful syndromes like erythromelalgia.

These failures are easily explained by the fact that the syndrome's nature is still unknown. Sympathectomy was performed, but as an attempt after numerous therapeutical trials. These failures, however, must not discourage, because their analysis may permit one to class in different groups seemingly alike facts and to enlighten some syndromes still obscure. This discussion shows the impossibility of delimiting yet exactly the indications of sympathectomy. I only want to give the results of five years' personal experience.

1. *In painful phenomena* sympathectomy is often very efficacious. In causalgia after war wounds it gave me in nine cases two complete failures, two satisfying improvements, and five excellent results, two of them being noted for several years. Platon has published a series of twelve cases, all seen again after more or less long intervals. He obtained 75 per cent. of successes. In certain painful crises preceding gangrene caused by obliterant endarteritis with or without intermittent claudication it has given the same good results as in painful acroparæsthesic syndromes consecutive to bruise of finger, to wounds of hand, palm or footsole. Sympathectomy is thus worth trying in these cases.

It was twice performed for Raynaud's disease and gave a good result.

The result was less constant in painful stumps where I had one success and one failure. It seems to be efficient where the neuralgia of the stump

is associated with vasomotor crises, muscular contracture or convulsive cramps. The neurologist, Clovis Vincent, related recently a striking example: In a case of painful stump, both deep general narcosis and the section of the main nerves failed to attenuate the stiffness and the convulsive cramp. Immediately after sympathectomy the muscles relaxed, and forty days after the pains had not reappeared.

2. *Concerning muscular phenomena* only the hypertonic symptom is influenced by sympathectomy. Heitz and myself have seen it in contractions consecutive to war wounds. All eighteen patients were much improved; the day after operation contracture ceased and voluntary movements became possible. In some very serious cases, only sympathectomy permitted the motor reëducation. I do not believe one can improve by sympathectomy what is wrongly called Volkmann's ischæmic paralysis. Whatever may be its starting-point, the syndrome is the consequence of a muscle's focal necrosis; it is a definitive lesion that nothing can modify.

In three personal cases of trophœdema a rapid diminution of the œdema was obtained.

3. *In trophic troubles* leading to ulcers, sympathectomy is very efficacious: twelve cases out of thirteen were followed by rapid healing, but relapse is possible if the cause of the trophic trouble has not been removed, and the cause is not always removed by the sympathectomy.

This is the case in perforating ulcers which never were operated successfully. Therefore after my experience I should not advise it. Sympathectomy also failed in a case of numerous ulcerations consecutive to frostbite. The initial lesions, neuritis or arteritis, provoked relapse of the ulcers.

4. In cases of trophic symptoms after nerve sections results were excellent. In ten patients I obtained rapid healing of the ulcers by sympathectomy. However, after observing seventeen cases, I believe sympathectomy should be replaced here by the removal of the neuroma of the sectioned nerve, which is the starting-point of vasomotor reflexes causing the trophic trouble. The removal of the neuroma must be followed by the reconstitution of the nerve's continuity either by suture or by grafts, avoiding thus the relapse of the neuroma. I performed seven grafts with complete satisfaction and now prefer this method to sympathectomy in spite of its satisfactory results.

What is the real action of sympathectomy? How does it realize healing of the most desperate ulcers? This seems to me due only to local circulatory hyperactivity noted always after sympathectomy in the following hours, vasodilatation being apparently one of the most active causes in the tissue's growth; certain local giantisms observed during growth depend probably on constant vasodilatation. After sympathectomy, vasodilatation accompanies local elevation of temperature and of pressure, both favoring healing. This fact is experimentally proved in the following way: Having produced vasodilatation in a rabbit's ear by removal of the superior cervical ganglion, one makes on both ears a wound of equal size. The sympathetized side is

FIG. 4.—The forceps catches a fold of the arterial external layer and holds it out of the wound.
 (1) In both figures the thickness of the external layer has been exaggerated to elucidate the demonstration. Drawings by Dr. P. Bonnet, associate professor of surgery in Lyons.

FIG 5.—The knife opens this fold and softly decorticates the artery.

FIG. 6.A.—Contracture of the fingers after injury of the forearm.

FIG. 6.B.—Contracture of the fingers after injury of the forearm—periarterial sympathectomy. Result after three days.

FIG. 7.—Contracture of the fingers after injury of the forearm—peri-arterial sympathectomy. Result after six days.

FIG. 8.A.—Trophic ulcer of the first toe after section of the sciatic nerve.

FIG. 8.B.—Trophic ulcer of the first toe after section of the sciatic nerve.
Recovery eight days after peri-arterial sympathectomy.

FIG. 9.A.—Spontaneous ulcer of stump with vasomotor troubles.

FIG. 9.B.—Spontaneous ulcer of stump with vasomotor troubles. Healed by peri-arterial sympathectomy.

more abundantly bleeding and more intensively red colored. Four days later its wound is covered with granulations, epidermis begins to grow, while on the opposite side the wound remains gray-colored without any epidermic border. On the eighth day there is complete healing on the sympathetomized side, while on the other side healing extends only over half the wound. A fortnight later the scar of the vasodilated side is more regular and supple, of better quality than the other and shows no retraction.

Peri-arterial sympathectomy has identical effects. Healing of trophic troubles is thus a question of vasodilatation, and therefore peri-arterial sympathectomy must be tried in these cases. The characteristic of sympathectomy is to be more vascular than a nerve operation. For this reason its consecutive vascular reactions might perhaps be employed in many circumstances. Producing an immediate ischæmia, lasting eight or ten hours, one can perform it to insure preventive hæmostasis. I did it successfully in three brain operations. On the other hand, the post-operative vasodilatation being lasting, it could be utilized to help the insufficient circulation, for instance, in obliterant endarteritis, or to prepare the reëstablishment of cerebral circulation previously to ligature of the common carotid. Therefore in spite of the hypothetic character of it I must believe the question ought to be studied concerning the viscera and glands. I wonder if the possibility of producing locally active hyperæmia might not give us a new power of action on organs when these are becoming insufficient? Only repeated experimental researches may reveal to us what those suggestions are leading to—they are not yet begun; I offer them to your appreciation.