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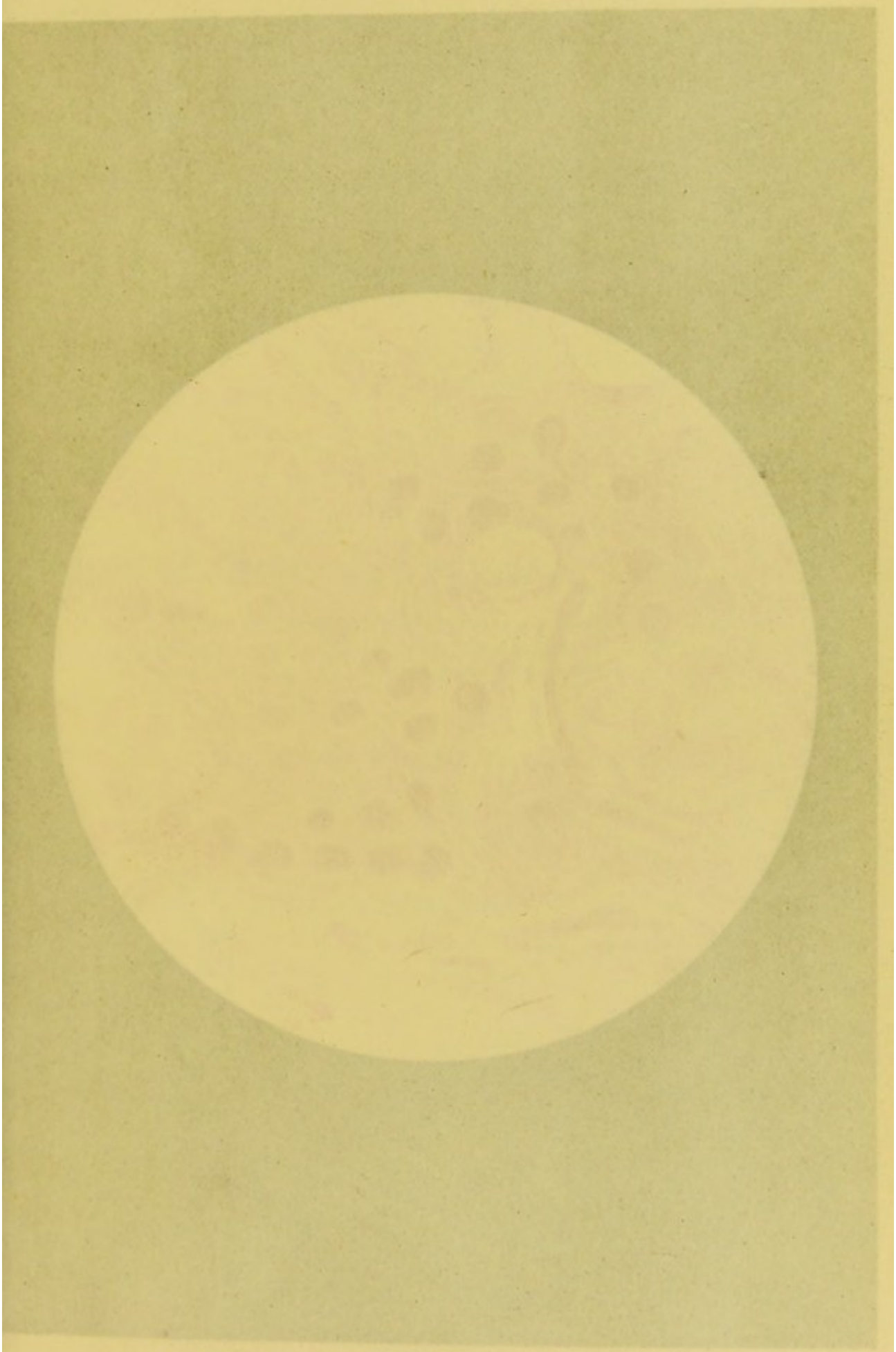
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THE

INFLUENCE OF THE SYMPATHETIC





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THE

INFLUENCE OF THE SYMPATHETIC ON DISEASE

BY

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PATHOLOGICAL ANATOMY AT THE BRISTOL MEDICAL SCHOOL

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WITH ILLUSTRATIONS

LONDON
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1885

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In memoriam Patris dilectissimi

Ἰδοὺ, ταῦτα μέρη ὁδοῦ αὐτοῦ, καὶ ἐπὶ ἰκμάδα λόγου ἀκουσόμεθα ἐν αὐτῷ.

Job xxvi, 14.

PREFACE.

When, for the Bradshaw Lecture at the College of Physicians in 1882, I chose as my subject 'The Influence of the Sympathetic on Disease,' it was scarcely possible to do more than touch on the subject.

The present book is an extension of the same line of thought; but, for the convenience of readers, it has been thought advisable to add some pages on the anatomy and physiology of the sympathetic system. It will be readily seen that these chapters are by no means exhaustive, but they are perhaps necessary for reference.

The workers in this department are so numerous, that it is difficult even to realise the extent of one's obligations to them; but I may say that, some years ago, the Sympathetic System was the subject of the Astley Cooper prize at Guy's Hospital. Although the prize essay has never been published, yet, by the kind permission of the author, Mr. George Arthur Woods, of Southport, and the courtesy of

the authorities of Guy's, I have been favoured by a perusal of it.

For much help, especially with regard to the authorities on the subject, I am indebted to Dr. Chapin's Fiske Fund Prize Essay (Rhode Island Medical Society).

I have to thank numerous friends and colleagues for cases, or for the use of plates.

The three most important points that the consideration of the sympathetic seems to me to illustrate, are—(1) The marvellous effect of reflex action in health and disease in connection with this system. (2) The exquisite mutual dependence of the various portions of the nervous system—cerebral, spinal, and sympathetic—on each other. (3) The fact that, in spite of this mutual dependence, the sympathetic, under conditions of lesion of brain or cord, is enabled within certain limits to act independently.

E. L. F.

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INFLUENCE OF THE SYMPATHETIC ON DISEASE.

CHAPTER I.

THE ANATOMY OF THE SYMPATHETIC.

Anatomy

It seems at least doubtful whether the anatomy of the whole sympathetic system of nerves is thoroughly known. It is easy to trace the great gangliated cords, lying on each side of the vertebral column, with the nerves proceeding from them, connected with the cranial nerves either directly or through the medium of one of the various ganglia in association with these cranial nerves, and united in their whole course with spinal nerves. It is easy to map out the large plexuses, lying more or less in the central line in front of the bodies of the vertebræ, and sending branches to viscera and vessels. Nor does much remain to be discovered as to the arrangement of the ganglia of the viscera themselves, which possess a certain amount of independent action, and yet are in close relation with the sympathetic ganglia and plexuses in their neighbourhood. But, owing to the extreme

ANATOMY

tenuity of a sympathetic nerve-filament, it has hitherto been found impossible to decide whether the great cerebro-spinal centres contain sympathetic ganglia, existing in the brain or spinal cord, though not of them. Experiment and pathology seem to show that such centres, for at any rate vasomotor action, exist in the medulla oblongata, and probably in each section of the spinal cord, even if not in higher centres still. It is conceivable that in all parts of the cortex of the brain, cells or a layer of cells may rule vascular tone, and may thereby act as a starting-point for the most usual manifestations of emotion. It has, however, been impossible to go beyond conjecture on this point. Some physiologists would fain consider the chain of Clarke's cells in the cord to be such sympathetic ganglia. The objection to this view is that this group of cells does not exist all through the cord. The whole question of the independence of the sympathetic system hangs upon the answer to this question. It will be seen that in limited spheres such independence of action obtains; but in a very large proportion of instances the action of the sympathetic is so correlated with the spinal cord, that the former is nearly inert without the nerve supply derived from the latter. It is impossible to deny that the sympathetic is a branched roadway from the cerebro-spinal system, into which overflow paths of innervation from all points of the great nerve-centres, to spread to all sides of the periphery. But is it nothing else? And if it depends so closely on the brain and cord, does it do so from a connection with some portion of this

system, or because in these great centres are imbedded ultimate sympathetic ganglia in close association with the gangliated cords, and united also with each other by delicate connections. If it be allowable to hazard a conjecture, founded more upon pathological and clinical observation than upon anatomical proof, the gangliated cords that run the length of the trunk of the body are united with some of the cells of the anterior horns, and probably with the cells of the second and third dimensions. Anatomy

Seeing the impossibility of tracing a fibre of Remak in the tissues of the brain or spinal cord, the existence of vasomotor centres in these organs, proved, as it is, by vasomotor disturbances on lesion of certain districts of the cerebro-spinal centres, affords by no means positive proof of the sympathetic ganglia being directly fed and influenced by them. The sympathetic connection between the medulla oblongata and the two chief centres in the cervical cord, the vasomotor centre for the head and face, and the oculo-pupillary, is proved by experiment and pathology. Such centres may lie in the cord and yet not be of it; and the whole system will consist not only of the well-known chains of ganglia, of the nerves and plexuses of the internal organs, of a vasomotor apparatus for the whole body, but of important ganglia that are situated, probably for purposes of correlation, within the structure of the great centres of the cerebro-spinal system.

The twofold connection of the sympathetic ganglia with the spinal cord by means of the double set of

ANATOMY

Superior cervical ganglion

fibres from each ganglion to the spinal nerve above it; the fact that of these fibres one, medullated, seems to have its exit from the cord with the anterior root of the spinal nerve, the other, non-medullated, to enter the spinal cord and to be distributed to its vessels; the connection of each ganglion with one below it by similar medullated and non-medullated fibres, all speak on the one hand of the close dependence of this great chain of ganglia with the cerebro-spinal centres, whilst on the other they point under certain circumstances to their independent action.

It seems certain that the three cervical ganglia are composed of a larger number than would answer to those corresponding to each vertebra in the dorsal and lumbar spinal column.

From the intimate connection of the superior cervical ganglion with those of the head and face, and with the cranial nerves, it is one of the most important in the whole body. Connected by filaments with the middle cervical ganglion lying below it, and united with the spinal cord in the usual manner, except that it is associated with the four first spinal nerves, it divides superiorly into two main branches, one that anastomoses with the glossopharyngeal, the pneumogastric, and the hypoglossal nerves; the other larger, the carotid branch, which practically appears as the origin of the cranial sympathetic. It forms the carotid plexus round the internal carotid artery; it gives off branches to form the cavernous plexus at the level of the cavernous sinus, and it surrounds by its meshes all the ramifications of the internal carotid.

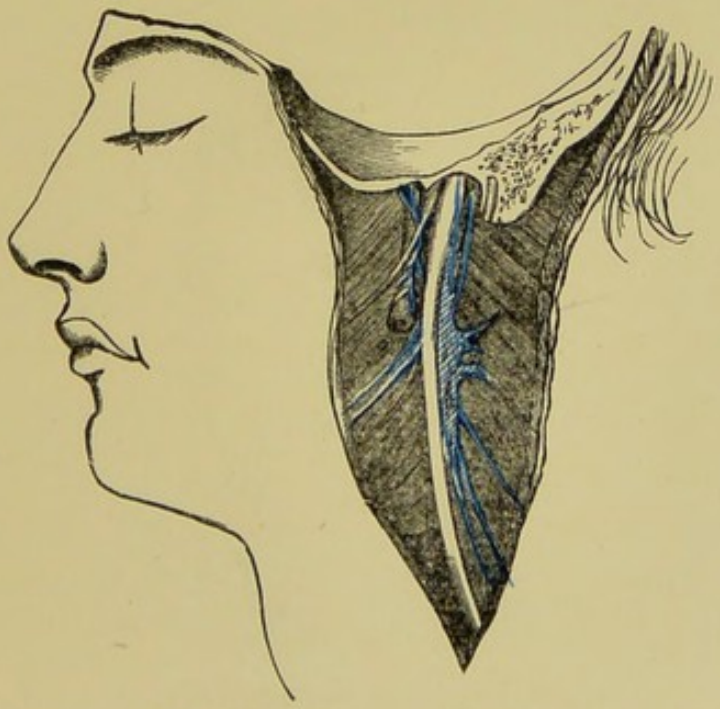
PLATE I.

A



Connection of Superior Cervical Ganglion with Ganglion of the Root of Vagus.

B



Connection of Superior Cervical Ganglion with Ganglion of Trunk of Vagus.

The position of this ganglion behind the internal carotid artery, and opposite to the second and third cervical vertebræ, favours this arrangement. The carotid plexus sends one branch to the membrana tympani, which unites with the nerve of Jacobson (the tympanic branch of the glossopharyngeal), and with it goes to the otic ganglion. A second branch is destined to form part of the Vidian nerve, and to go to Meckel's ganglion. A third branch, sometimes single, sometimes composed of several filaments, joins the sixth nerve, and expands with it on the external rectus muscle of the eye. Another branch goes to form a plexus on the middle meningeal artery, from which filaments extend to the otic ganglion and the gangliform enlargement of the facial nerve. A terminal branch along the anterior communicating artery goes to form the small ganglion of Ribes, in which the sympathetics of each side are united.

The cavernous plexus gives branches to the mucous membrane of the sphenoidal sinus, to the dura mater of the basilar groove, the pituitary body, the third, fourth, and sixth nerves, and to the Gasserian ganglion of the fifth nerve, and sends a pretty large branch to the ophthalmic or lenticular ganglion. This constitutes a small centre, directly connected with the third and fifth nerves, and forms, with the spheno-palatine and the otic ganglia, the ganglionic part of the cranial sympathetic, or rather, the three principal quarters of the peripheral centre of the cranial region.

From the ophthalmic ganglion, the ciliary nerves

ANATOMY

Superior cervical ganglion

form in the ciliary muscle a ganglionic plexus, called the orbicular ganglion, from which branches run to the cornea, the ciliary muscle, and the iris.

The cavernous plexus also sends branches to the carotid artery. Filaments from the superior cervical ganglion, moreover, unite with branches from the pneumogastric, the glossopharyngeal, and the hypoglossal nerves to form the pharyngeal plexus.

This superior cervical ganglion gives off also, either as one or two branches, the upper cardiac nerves, joining on the left side the superficial cardiac plexus, on the right side helping to form the superior cardiac plexus. These unite with other branches of the sympathetic, notably with some from the thyroid branch of the middle cervical ganglion, as also with fibres from the pneumogastric, the external and recurrent laryngeal nerves. From this nerve branches are distributed to the thyroid body, accompanying the inferior thyroid artery. Other branches to blood-vessels spring also from the superior cervical ganglion.

The connections of this ganglion with the cranial ganglia are therefore manifold. All the cranial arteries also possess small vasomotor ganglia.

1. The petrous ganglion of the glossopharyngeal nerve is situated in a depression in the lower border of the petrous portion of the temporal bone. It is formed of all the fibres of the glossopharyngeal, and is connected with the pneumogastric by a branch united to one of its branches, and by another which goes to the upper ganglion of the pneumogastric.

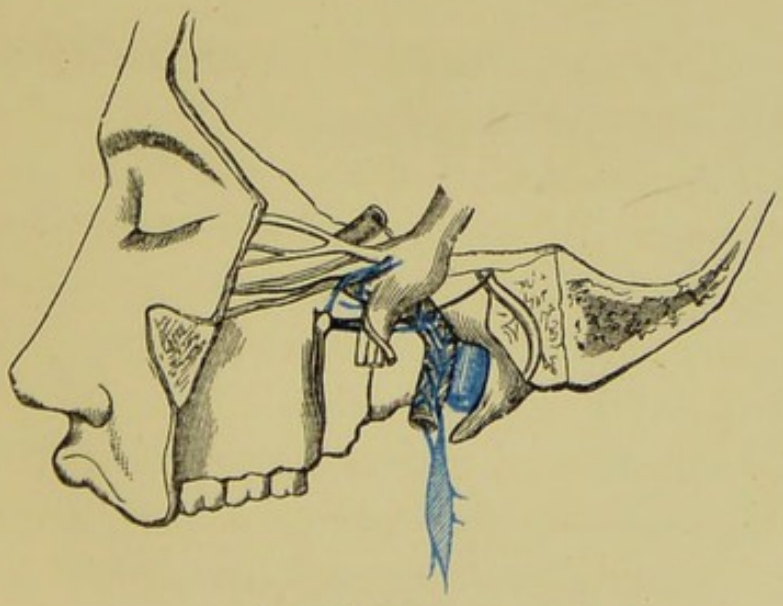
PLATE II.

A



Connection of Superior Cervical Ganglion with Petrous Ganglion of the Glosso-pharyngeal.

B



Gasserian Ganglion.

Superior cervical ganglion

It is connected with the sympathetic by a branch that comes to it from the superior cervical ganglion. It gives off the nerve of Jacobson, which divides into six filaments, with one of which, the third, the sympathetic is again united in the carotid canal.

2. The otic ganglion (Arnold's) of the fifth nerve. Situated immediately below the foramen ovale, on the inner surface of the inferior maxillary nerve, and close to the origin of the internal pterygoid nerve. It is connected with the inferior maxillary division of the fifth nerve, and its internal pterygoid branch —with the auriculo-temporal branch of the fifth nerve, with the glossopharyngeal and facial nerves through the small petrosal nerve from the tympanic plexus — and with the sympathetic by filaments from the plexus surrounding the middle meningeal artery.

3. The Gasserian or semilunar ganglion of the fifth nerve. Formed by fibres of the sensory root. It receives on its inner side filaments from the carotid plexus of the sympathetic.

4. The ophthalmic, lenticular, or ciliary ganglion of the first division of the fifth nerve lies at the back of the orbit between the optic nerve and the external rectus muscle, generally on the outer side of the ophthalmic artery. It has three roots: one, the long one, from the nasal branch of the ophthalmic nerve, a branch of the fifth, which is sometimes joined by filaments from the cavernous plexus of the sympathetic; a second, the short one, from a branch of the third nerve, for the inferior oblique muscle; a

ANATOMY

Superior cervical ganglion

third, the sympathetic root, from the cavernous plexus of the sympathetic.

5. The spheno-palatine, or Meckel's ganglion, on the second division of the fifth nerve, deeply placed in the spheno-maxillary fossa, close to the spheno-palatine foramen. It has three roots: 1. Motor, from the facial nerve through the Vidian. 2. Sensory, from the spheno-palatine branches of the fifth nerve. 3. Sympathetic, from the carotid plexus, through the Vidian nerve.

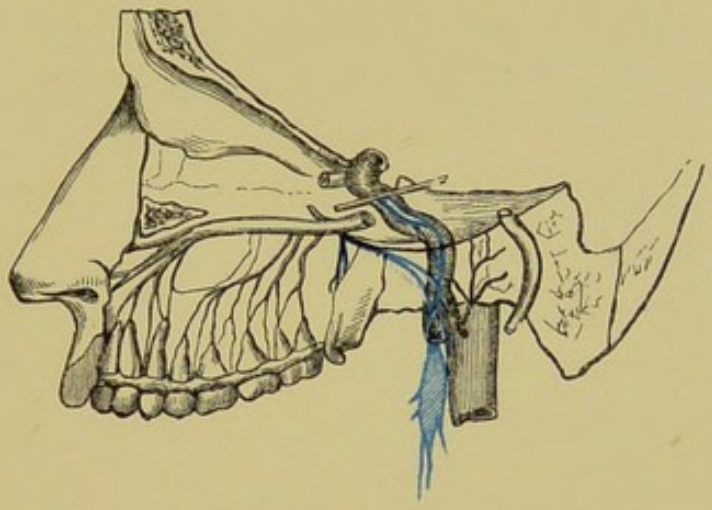
6. The submaxillary ganglion, on the third division of the fifth nerve, is situated above the deep portion of the submaxillary gland, near the posterior portion of the mylo-hyoid muscle. It has three roots: 1. Motor, from the chorda tympani, a branch of the facial nerve. 2. Sensory, from filaments from the gustatory nerve, a branch of the fifth. 3. Sympathetic, from filaments from the nervi molles, the sympathetic plexus round the facial artery.

7. The gangliform enlargement on the facial nerve is situated opposite the hiatus Fallopii. It communicates by the large petrosal nerve with Meckel's ganglion; by filaments from the small petrosal (a branch of the eighth nerve) with the otic ganglion; by the external petrosal with the sympathetic filaments accompanying the middle meningeal artery.

8. The jugular ganglion of the pneumogastric, the ganglion of the root of the pneumogastric, is connected with the spinal accessory nerve, with the petrous ganglion of the glossopharyngeal, with the facial nerve by means of its auricular branch, with

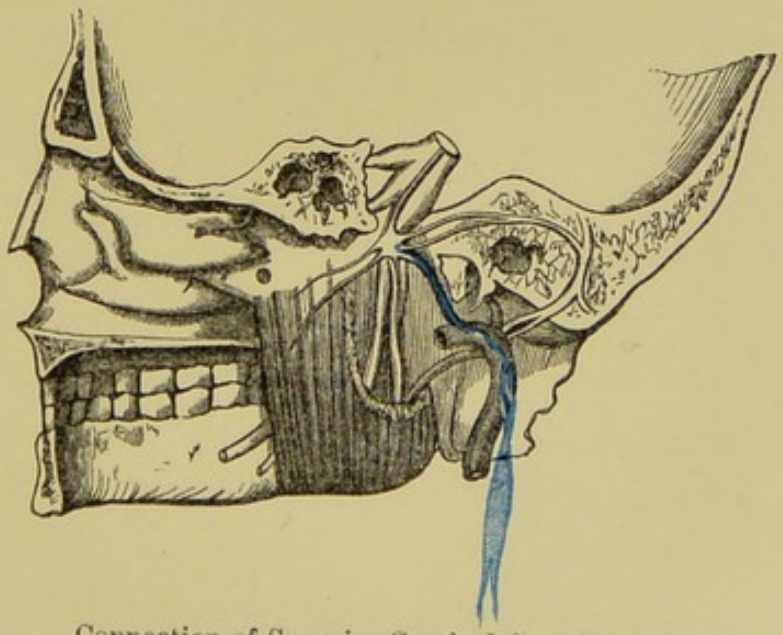
PLATE III.

A



Connection of Superior Cervical Ganglion with Meckel's Ganglion.

B



Connection of Superior Cervical Ganglion with Submaxillary Ganglion.

the sympathetic by an ascending filament from the superior cervical ganglion.

9. The ganglion of the trunk of the pneumogastric is connected with the hypoglossal nerve, with the superior ganglion of the sympathetic, and with the loop between the first and second cervical nerves.

The middle cervical ganglion is situated close to the inferior thyroid artery, and may be on it. If the superior cervical ganglion may be considered as made up of four ganglia, the middle may be looked at as composed of two, and it is connected with the fifth and sixth cervical nerves. It has two main branches: 1. Thyroid, which divides into two, the first supplying the thyroid gland and joining the recurrent laryngeal and the external laryngeal nerves; the other supplying the thyroid artery, and communicating with the upper cardiac nerve. 2. The middle cardiac nerve, which in the neck communicates with the recurrent laryngeal and the upper cardiac nerves; in the chest it is associated with filaments from the recurrent laryngeal nerve, and terminates in the deep cardiac plexus, often on the left side uniting with the lower cardiac nerve. The middle cervical ganglion communicates also by fibres with the ganglion above and below it.

The inferior cervical ganglion is situated between the neck of the first rib and the transverse process of the last cervical vertebra, beneath the vertebral artery. It is connected with two and sometimes with three lowest cervical nerves, and may be considered

Anatomy

The cervical ganglia

a single ganglion, like one of the thoracic chain. Its important branch is the lower cardiac nerve, which communicates with the middle cardiac and the recurrent laryngeal, and terminates in the deep cardiac plexus. It also sends branches round the subclavian artery, and a more important branch along the vertebral artery; and this branch communicates with the sixth, seventh, and eighth pair of nerves, so bringing the phenomena of tinnitus and vertigo into relation with lesions of very various parts of the system.

The connection, therefore, of the sympathetic with the cranial nerves is as follows:

With the first nerve, only indirectly through the nasal branch of the ophthalmic nerve (of the first division of the fifth), which nerve is joined by filaments from the cavernous plexus of the sympathetic.

With the second nerve, the optic, perhaps by a branch from Meckel's ganglion, perhaps by a small filament from the ciliary nerve; from the lenticular ganglion, penetrating the optic nerve with the arteria centralis retinæ.

With the third nerve, the motor oculi, by filaments from the cavernous plexus; with the fourth nerve, the pathetic, by filaments from the cavernous plexus in the outer wall of the cavernous sinus.

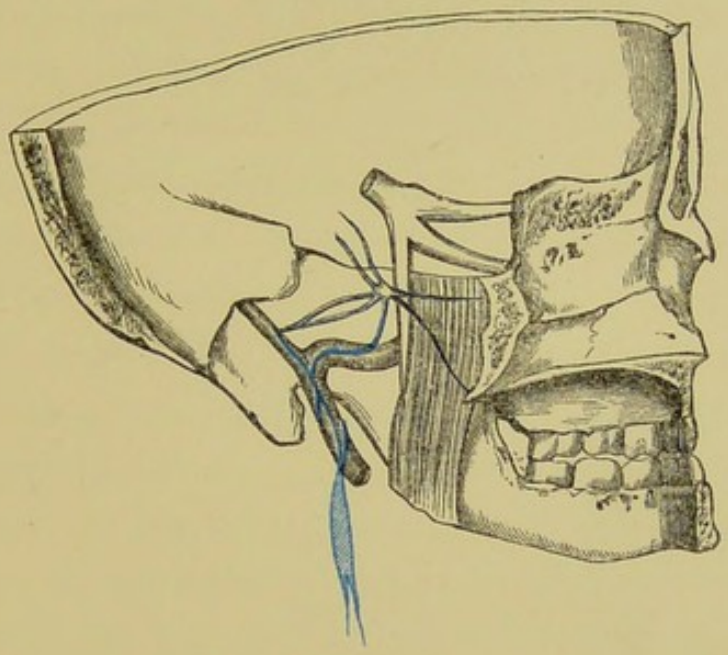
With the fifth nerve, the trifacial.

1. The Gasserian ganglion, by filaments from the carotid plexus.

2. The ophthalmic nerve, by filaments from the cavernous plexus, the ganglionic branch of the nasal

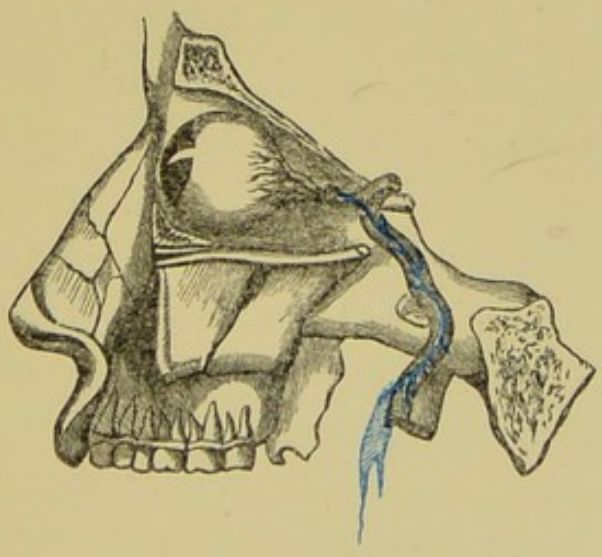
PLATE IV.

A



Otic Ganglion.

B



Lenticular Ganglion.

branch of the ophthalmic nerve by filaments from the cavernous plexus.

3. Meckel's ganglion, by filaments from the carotid plexus through the Vidian nerve.

4. Ophthalmic ganglion, by filaments from the cavernous plexus.

5. Otic ganglion, by filaments from the plexus round the middle meningeal artery.

6. Sub-maxillary ganglion, by filaments from the plexus round the facial artery.

With the sixth nerve, the abducens, in the cavernous sinus, by filaments from the carotid and cavernous plexuses.

With the seventh, the facial; in the aqueductus Fallopii, by the external petrous connected with filaments accompanying the middle meningeal artery; and at its exit from the stylo-mastoid foramen, by filaments from the carotid plexus.

With the seventh, the auditory, it is doubtful whether there is any direct connection; but perhaps sympathetic fibres join it from the internal auditory branch of the vertebral artery.

With the eighth, the glossopharyngeal. The petrous ganglion of the glossopharyngeal is joined by a branch from the superior cervical ganglion; the carotid branches of the tympanic branch are joined by filaments from the carotid plexus.

With the eighth, the pneumogastric.

The ganglion of the root, by a branch from the superior cervical ganglion; the ganglion of the trunk, by a branch from the superior cervical

Anatomy
The cervical ganglia

ganglion; the pharyngeal branch, by filaments of the pharyngeal plexus.

The external laryngeal branch, by a communication with the superior cardiac nerve; the recurrent laryngeal branch, by cardiac branches of the sympathetic; the superior cervical cardiac branches, by cardiac branches of the sympathetic; the anterior pulmonary branch, by filaments from the sympathetic; the posterior pulmonary branch, by filaments from third and fourth thoracic ganglia; the gastric branches, by fibres from the solar plexus.

With the eighth, the spinal accessory—at the ganglion of the root of the pneumogastric.

With the ninth nerve, the hypoglossal, opposite the atlas, by branches derived from superior cervical ganglion.

The duties of the cervical ganglia may be summarised by saying, that all three ganglia in some sense rule the heart; that all in various degrees supply vasomotor power to the vessels of the spinal cord; that the superior cervical ganglion, by its connection with the lenticular ganglion, has much to do with the movements of the iris; by its association with other cranial nerves, has some part in the secretion of saliva, of tears, of nasal and pharyngeal mucus. This ganglion, too, supplies vasomotors to the external carotid and its branches, thus innervating the vessels of the face, scalp and ears; to the internal carotid and its branches within the skull, innervating the dura mater, the vessels of the anterior and middle

The cervical ganglia

brain, both basal ganglia and cortex, the latter through the vessels of the pia mater. By its branches to the pharyngeal plexus it supplies the pharynx and upper part of the œsophagus. Whether this superior cervical ganglion is the only vasomotor centre for these portions of the brain, or only the chief one, is not quite certain. But after ablation of the superior cervical ganglion, vasomotor influence may gradually be supplied by nerves from the cervical plexus, by fibres from the pons, medulla oblongata, and upper part of the cord. These act but little when the cervical sympathetic is intact, but come into action when it is destroyed. It is probable, but not actually proven, that all the nerves whose nuclei lie in the bulb contain sympathetic filaments.

The middle cervical ganglion supplies vasomotors to the thyroid gland, the larynx and part of the trachea.

The inferior cervical ganglion supplies vasomotors to the vertebral and basilar arteries and their branches.

The close connection between the branches from the superior cervical ganglion and the two ganglia of the pneumogastric, with one of which, too, the jugular ganglion, the spinal accessory nerve is united, shows the anatomical difficulty in separating the physiological functions of the sympathetic and the vagus. The space in which the vagus is not connected with the sympathetic is so small, and so difficult of access, that this fact alone accounts for the great variation in views as to the distinctive

Anatomy

Cardiac innervation.

functions of these nerves in the stimulation or inhibition of the cardiac movements, more especially as probably at its very origin the pneumogastric carries some sympathetic fibres.

The six sympathetic cardiac nerves interlace with the cardiac nerves from the vagus, and form the cardiac plexuses. Of these, the superficial cardiac plexus, situated beneath the arch of the aorta, and in front of the right branch of the pulmonary artery, is composed of the lower branch of the left, and sometimes of the right pneumogastric nerve, with only one sympathetic branch, the first cardiac branch of the left side. This plexus includes in its meshes a ganglion, the ganglion of Wrisberg. It sends branches to the anterior coronary plexus, and on the left side supplies filaments to the left anterior pulmonary plexus.

A larger, and far more important plexus, the deep cardiac plexus, is composed of all the cardiac nerves of the sympathetic and the pneumogastric, except those that go to form the superficial cardiac plexus. This deep plexus is situated between the arch of the aorta and the end of the trachea. It is made up of all the cardiac branches from the cervical ganglia of the sympathetic (except the superior cardiac nerve of the left side) and all the branches to the heart of the vagus and the recurrent laryngeal (except the lower cardiac nerve of the left side). On the left side branches are sent off to unite with the superficial cardiac plexus, but the main number of branches go to form the posterior coronary plexus. On each side the deep cardiac plexus sends filaments to the

anterior pulmonary plexus; on the right side branches join filaments from the superficial cardiac plexus to form the anterior coronary plexus; some filaments terminate in the posterior coronary plexus, and some are distributed to the right auricle of the heart. The anterior coronary plexus is situated along the course of the anterior coronary artery. It is composed, as stated above, of filaments mainly from the superficial cardiac plexus, receiving also a few fibres from the right side of the deep cardiac plexus. The posterior coronary plexus is distributed along the course of the posterior coronary artery and the back of the heart. It derives its nerve supply mainly from the left side of the deep cardiac plexus, but is also united to some fibres from the right side also.

Besides these plexuses the heart is furnished with numerous nervous filaments and ganglia, lying mainly beneath the lining membrane of the heart, and forming a nerve supply that, under certain circumstances, seems able to act as an independent system. The nerves and ganglia of which this system is composed are practically extensions of the sympathetic. The ventricles are more rich in nerves than the auricles, the left ventricle more richly supplied than the right. The ganglia exist in greatest number between the auricles and ventricles, and in the septum of the heart; in smallest number, even if they are found at all, at the apex of the organ. What their function is, is not fully known. Whether these ganglia act as co-ordinating centres for the opposite influence of the vagus and the sympathetic cardiac nerves,

Anatomy

Cardiac innervation

whether they have much to do in ordering the cardiac rhythm, whether they are merely centres of reflex arcs for the heart itself, it seems certain that their chief function is to keep up nerve supply to the heart under injury to the cervical ganglia or to the vagus. The whole existence of the individual depending on the action of the heart, it is not unreasonable to expect that, besides the elaborate contrivance for nervous supply from the medulla oblongata and the sympathetic, there should be some further arrangement by which the action of the heart, and thereby the life of the individual, might be secured, for a time at least, even under conditions of serious nerve inhibition.

The lungs are singularly little connected with the sympathetic. It is indeed doubtful whether the circulation of the pulmonary artery is influenced at all by vaso-constrictors. From the superficial cardiac plexus, however, branches are sent to the anterior pulmonary plexus of the left side. From the deep cardiac plexus fibres are supplied to the anterior pulmonary plexus of each side. From the upper portion of the thoracic part of the gangliated cord, the third and fourth thoracic ganglia, the posterior pulmonary plexus derives some of its nerve supply. The distribution of the nerves from these plexuses seems confined to the vessels of the bronchial tubes. Small ganglia are found upon them.

In the thorax, on each side of the spinal column, lying over the heads of the ribs, and covered by the

The thoracic ganglia

pleura, is placed a ganglion corresponding to each dorsal vertebra, connected with the spinal nerve of the same region by two branches, one a branch of the nerve itself, the other composed of sympathetic filaments. The disturbances that occur in the circulation of the spinal column on the destruction of this sympathetic branch render it more than probable that it has its origin in the ganglion, although the oculo-pupillary phenomena, and the vasomotor, that arise on section of various regions of the upper cord, prove that each segment of the cord may contain a sympathetic centre. The first thoracic ganglion is small, and is sometimes united with the inferior cervical ganglion. Each ganglion is connected by one or more branches with the ganglion above and below it; the second and third send branches to the thoracic aorta, the vertebræ, the œsophagus; the fourth ganglion sends important branches to the posterior pulmonary plexus. The lower thoracic ganglia are the centres of origin for the splanchnic nerves. Of these, the great splanchnic, the most important sympathetic nerve in the body, is generally formed by roots from the fifth, sixth, seventh, eighth and ninth thoracic ganglia. Its origin varies in different individuals. The tenth ganglion may send roots to it, and in very rare cases it may derive fibres from all the upper ten dorsal ganglia, even from the first. It follows an obliquely descending course over the bodies of the dorsal vertebræ, and after perforating the diaphragm terminates in the semilunar ganglion, sending off numerous branches from this ganglion to

ANATOMY

The splanchnic nerves

which reference will be made. In its course through the chest it receives roots, but gives off no branches. Spinal nerves preponderate in its composition.

The small splanchnic nerve usually arises from the tenth and eleventh thoracic ganglia, and communicating with the great splanchnic in its course through the chest, perforates the diaphragm and terminates in the cœliac plexus.

The smallest splanchnic nerve arises from the twelfth thoracic ganglion, and sometimes sends a branch to the small splanchnic nerve lying above it. It also perforates the diaphragm and terminates in the renal plexus, and perhaps the lower part of the cœliac.

The great plexuses of the abdomen and the pelvis are so connected, that it is convenient to speak of the lumbar and sacral ganglia, the remaining centres of the gangliated cord, before describing them. In the lumbar region the ganglia are situated somewhat nearer to each other. They lie along the inner edge of the psoas muscle. They vary in number. Most usually there are four, but the number may be three or five. They send branches to form the lumbo-aortic plexus over the lower part of the aorta, and thence supply branches to the superior mesenteric plexus, a plexus containing but few ganglia, and to the spermatic or ovarian plexus, a prolongation at once of the renal, solar, and lumbo-aortic plexuses.

The sacral part of the gangliated cord is composed of four and sometimes of five ganglia of ellipsoid form, connected with each other often by a twofold

The sacro ganglia

system of branches, one direct, the other having its origin from one of the anterior branches of a ganglion to descend to be connected either with the ganglion beneath it or with a branch from it almost as it leaves the ganglion. Their connection, too, with the sacral nerves is by thick roots, often more in number than are found in the parts of the gangliated cord higher up. Branches are sent across the part of the sacrum to join the corresponding opposite branches, and also to the plexus on the middle sacral artery. But the main branches run to form part of the hypogastric plexus, mingling with fibres from the lower division of the aorta, and from the inferior mesenteric plexus. From the hypogastric plexus are supplied fibres which, anastomosing with the lower hæmorrhoidal nerves, form the hæmorrhoidal plexus, also fibres to the vesical plexus, which contains a threefold network, subperitoneal, intermuscular, and submucous, of which the first encloses many ganglia. In the ureters, however, there are no ganglia. From this hypogastric plexus, moreover, are supplied fibres to the prostatic plexus, fibres to the vesiculæ seminales and vas deferens, and, in the female, fibres to the vaginal plexus connected with the vesical and hæmorrhoidal plexuses, fibres to the uterine plexus running along the great ligament, and containing few ganglia, and those near the cervix uteri.

Lower down on the coccyx itself lies the coccygeal ganglion—'ganglion impar'—equally connected with the lowest sacral ganglion on each side, and supplied also from the coccygeal nerves. It sends filaments into

Anatomy — The abdominal plexuses

the coccygeal gland to the coccyx itself and its ligaments. The nature of the coccygeal gland is doubtful. It seems to be made up of tortuous arteries with thickened coats, but some nerve cells are found scattered through it.

The abdominal plexuses seem all to find their centre of origin in the solar plexus. Connected as it is with almost every organ of the body, with a vast influence on the circulation, with direct power over all the secretions of the abdominal and pelvic viscera, with a reflex reaction on the heart that under certain conditions may lead to fatal syncope, it is scarcely to be wondered at that some physiologists have considered it the very centre of life itself.

It is placed behind the stomach. Subjacent to it lies the crus of the diaphragm and the aorta. It surrounds the cœliac axis and the origin of the superior mesenteric artery, and widens out to the region of the suprarenal capsules, its close association with which will be seen to be of importance in explaining the pathology of Addison's disease. It consists of a vast plexiform arrangement of nerves, partly sympathetic, partly from the right vagus, and of some of the largest sympathetic ganglia in the body. These ganglia, the semilunar, are somewhat on the lateral region of the solar plexus. Both of them, one on each side, seem composed of several ganglia. They are situated close to the suprarenal capsules. The one on the right side lies beneath the vena cava. Each of these ganglia receives the great

The abdominal plexuses

splanchnic nerve of the corresponding side. The small splanchnic nerve is sometimes united to the lower part of the semilunar ganglion; but this nerve may be lost in the solar plexus without any direct association with the semilunar ganglion.

Although practically the semilunar ganglia and solar plexus form one body, it is convenient to describe secondary plexuses as having their origin from one or the other, according to the apparent anatomical arrangement, premising, however, that every nerve uniting with the solar plexus or with these ganglia respectively, is necessarily brought into connection with the whole structure of ganglia and nervous network.

Associated then anatomically in special relation with the semilunar ganglia are the nerves forming—(1) the diaphragmatic plexus; (2) the suprarenal plexus; (3) the renal plexus; (4) the spermatic plexus.

The diaphragmatic plexus supplies the vasomotors to the phrenic artery, innervates the vessels of the diaphragm itself, and sends a fibre or two to the suprarenal capsule. It is composed of fibres from the upper portion of the semilunar ganglion, uniting with some from the phrenic nerve. On the right side, and on the right side only, a small ganglion exists beneath the diaphragm—'ganglion diaphragmaticum'—at the point of union of the sympathetic and the phrenic nerves. Branches from this plexus, besides supplying the suprarenal capsule, are distributed to the vena cava and to the hepatic plexus.

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The abdominal plexuses

The suprarenal plexus is made up of fibres from various sources, chiefly from the external portion of the semilunar ganglion, but also from the solar plexus, from the branch of the diaphragmatic plexus above mentioned, and from the small splanchnic nerve on the right side, also from the hepatic plexus. Where the small splanchnic nerve unites with the suprarenal plexus, a small ganglion is formed—'splanchnico-suprarenale.' This plexus supplies only the suprarenal gland, and the importance of this plexus and of the gland itself seems to be shown by the great size of the nerves and their number in the suprarenal gland. In the medullary portion of the gland between thirty and forty nerve trunks of some size have been distinguished. The renal plexus also derives its nerves from various sources—from the external and lower portions of the semilunar ganglion, from the solar plexus, slightly also from the aortic plexus, and from the smallest splanchnic nerve, the nerve derived from the twelfth thoracic ganglion. Numerous ganglia are interspersed in this plexus along the course of the renal artery. This plexus sends filaments on the right side to the vena cava, and on each side of the body it is the main origin of the spermatic plexus; but its chief function is following the renal arteries into the substance of the kidney. The spermatic plexus derives most of its nerves from the renal plexus, and therefore only indirectly from the semilunar ganglion and the solar plexus. Some of its fibres come also from the aortic plexus. Its connection with the renal plexus is shown patho-

logically by the renal pains so often experienced in disease of the testis or of the ovary. It distributes nerves along the spermatic vessels to the testis, and its position so high above the pelvis is probably owing to the position of the testis in embryonic life. It is connected with a branch from the vas deferens. In the female the plexus sends nerves to the ovary and to the fundus uteri.

From the more central position of the solar plexus important arrangements of nerves base their origin. The cœliac plexus, which is really part of the solar plexus itself, holds in its meshes ganglia of much smaller size than the semilunar, and receives branches from the small splanchnic nerves, perhaps sometimes from the great splanchnics, and on the left side from the vagus. It is arranged round the cœliac axis and follows the course of the arterial branches of this vessel. The plexuses, therefore, that emanate from the cœliac plexus are conveniently named after these arteries, the hepatic, the gastric, and the splenic. Of these, the hepatic is the largest branch ; it receives fibres from the left pneumogastric nerve and the right phrenic nerve ; it follows the course of the hepatic artery into the liver, supplying its various branches and ramifying in the vena portæ, the gall-bladder and the bile ducts. Pfluger thinks that each hepatic cell is a nerve termination. It sends filaments to the right suprarenal plexus. Following as it does the branches of the hepatic artery, it forms a pyloric, a gastro-duodenal, and a cystic plexus. The pyloric plexus unites with the gastric plexus and the pneumo-

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The abdominal plexuses

gastric nerves. The gastro-duodenal divides, like the artery, into the pancreatico-duodenal plexus and the right gastro-epiploic plexus. The former supplies the pancreas and the duodenum and unites with branches from the superior mesenteric plexus. The latter runs with the artery of the same name along the greater curvature of the stomach, uniting with branches from the splenic plexus.

The cystic plexus, as its name implies, provides nerves for the gall-bladder.

The gastric plexus, from the cœliac, is a smaller offset than the hepatic. It surrounds the gastric artery in its course along the lesser curvature of the stomach, uniting with fibres from the left vagus and supplying the stomach.

The splenic plexus derives branches from the left semilunar ganglion and the right vagus. The splenic artery is large and tortuous in its course; it sends branches to the pancreas, the pancreatica magna, and the pancreaticæ parvæ, the vasa brevia of the stomach, and the gastro-epiploica sinistra, besides supplying the spleen itself. All these subdivisions are followed by the nerves of the splenic plexus. Besides, therefore, supplying the spleen, it gives off branches to join the pancreatic plexus (an offset of the superior mesenteric plexus), and forms also the left gastro-epiploic plexus along the greater curvature of the stomach.

From the lower portion of the solar plexus arise two important plexuses, the superior mesenteric and the aortic. The superior mesenteric plexus receives

a special branch from the junction of the right vagus with the cœliac plexus. The nerves of this plexus have numerous ganglia connected with them, probably as centres for reflex arcs of moderate limitation. It surrounds and follows the ramifications of the superior mesenteric artery, the inferior pancreatico-duodenal, the vasa intestini tenuis, the ileo-colic, the colica dextra, and the colica media. On each of these arteries plexuses of the same name are distributed, supplying part of the pancreas, the small intestine, the ileo-cæcal valve, the cæcum, the ascending and transverse colon.

The aortic plexus ramifies along the abdominal aorta between the superior and inferior mesenteric arteries. It is derived chiefly from the semilunar ganglia and renal plexus of each side, receiving some branches from the third and fourth lumbar ganglia. It often contains a few small ganglia at its centre. It supplies the abdominal aorta itself, generally sends branches to the spermatic plexus, gives off the inferior mesenteric plexus from its left side, and terminates in the hypogastric plexus.

The inferior mesenteric plexus arises from the left side of the aortic. It surrounds and ramifies with the inferior mesenteric artery, passing in front of the left common iliac artery. As the artery divides into the left colic, the sigmoid, and the superior hæmorrhoidal, so the plexus subdivides into smaller plexuses, following the course of these arterial branches. The left colic branches may unite with the middle colic branches of the superior mesenteric

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The abdominal plexuses

plexus, and the left colic plexus unites at several points with the sigmoid plexus, supplying the descending colon and the sigmoid flexure. The superior hæmorrhoidal plexus follows the course of the superior hæmorrhoidal artery, descending with it into the rectum and supplying that region. Just as the superior hæmorrhoidal artery divides about four inches from the anus, opposite the middle of the sacrum, forming two branches, which descend one on each side of the rectum, eventually anastomosing with the middle hæmorrhoidal arteries from the internal iliac, and with the inferior hæmorrhoidal arteries from the internal pudic, so the nerves of the superior hæmorrhoidal plexus follow the course of the artery in its subdivisions, and are united with branches from the inferior hæmorrhoidal plexus, an offset of the pelvic or inferior hypogastric plexus.

The hypogastric plexus may be considered almost as a continuation of the aortic plexus, from which it receives eight or ten nerves on each side. It is also supplied pretty freely from the fourth lumbar ganglion and the first and second sacral ganglia. It is situated in the connective tissue of the space between the common iliac arteries. It contains no ganglia, apparently because it gives off no direct branches to organs, but indirectly supplies all the pelvic viscera by dividing into two plexuses, the pelvic or inferior hypogastric plexuses.

The two lateral portions of the hypogastric plexus form the pelvic plexus. This plexus is situated differently in the two sexes. In the male

it is placed at the side of the bladder and rectum; in the female, at the side of the bladder, vagina, and rectum. It is largely supplied from the second, third, and fourth sacral nerves, and slightly by fibres from the sacral ganglia. It contains small ganglia. The nerves which form the plexus, in their course from the hypogastric plexus, enter into repeated communications as they descend; and at the points of communication small ganglioform knots are found, which contain a little ganglionic matter.

It supplies all the pelvic viscera (except that part of the rectum innervated by the superior hæmorrhoidal plexus, and the testis or ovary supplied from the spermatic or ovarian plexus) by plexuses of greater or less importance. These vary greatly in the number of ganglia they contain.

They are—1. The inferior hæmorrhoidal plexus, from the posterior part of the pelvic plexus. It supplies the lower part of the rectum, following the course of the middle hæmorrhoidal artery.

2. The vesical plexus, from the anterior portion of the pelvic plexus. Its nerves accompany the vesical arteries. Those which accompany the superior vesical artery supply the fundus and much of the body of the bladder. In the male, branches are given off, forming small plexuses in the vas deferens and the vesiculæ seminales. The plexus to the vas deferens is distributed to that tube in its course to the testis, and in the spermatic cord communicates with the nerves of the spermatic plexus. In the male also, the nerves that

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The pelvic plexuses

form a plexus on the vesiculæ seminales are united with a branch or branches on the prostatic plexus. The plexus to the vesiculæ seminales follows the course of the middle and inferior vesical arteries, and supplies not only these organs, but the base of the bladder and part of the prostate gland.

3. The prostatic plexus is situated between the prostate gland and the levator ani. It supplies part of the prostate, and sends a branch to the vesiculæ seminales. It is then continued forwards to form the cavernous nerves of the penis. At the anterior margin of the levator ani they are joined by branches from the internal pudic nerve, pass under the pubic arch, and are distributed to the erectile structure of the penis. They consist of small cavernous nerves, which enter the corpus cavernosum near the root of the penis, and the large cavernous nerve, which passes along the dorsum of the penis, supplying its dorsal artery, and which eventually divides into two branches for the glans and prepuce, supplying also the artery of the corpus cavernosum. Both these arteries are terminal arteries of the internal pudic. Branches are distributed to the corpus spongiosum urethræ. In the female, the ovarian plexus is derived from the renal and aortic plexuses, is prolonged on the ovarian artery, and receives branches from the uterine nerves.

4. The vaginal plexus arises from the lower part of the pelvic plexus, and follows very much the course of the internal pudic artery. The superficial artery supplies the labia pudendi; the artery of the bulb

supplies the erectile tissue of the bulb of the vagina, whilst the terminal arteries supply the clitoris, the artery of the corpus cavernosum the cavernous body of the clitoris, and the arteria dorsalis clitoridis the dorsum of that organ. Following the course of these arteries, the vaginal plexus innervates all the parts of the vagina above named; but it is scarcely sufficiently arranged in meshes to be really a plexus.

5. The uterine nerves enter the pelvic plexus from the hypogastric. They are derived above the point of union of the sacral nerves with the pelvic plexus. The fundus uteri is innervated mainly from the ovarian plexus. The uterine nerves follow the course of the uterine arteries between the layers of the broad ligament, and form a plexus, that contains ganglia, round the arteries. These ganglia, like the nerves themselves, and all other structures of the uterus, enlarge considerably during pregnancy. After leaving the arteries, the uterine nerves plunge into the structure of the organ, supplying especially the neck and lower part of the body of the uterus. This organ may therefore be said to have three chief connections with the sympathetic—(1) From the ovarian plexus; (2) direct from the hypogastric plexus; (3) several important nerves from the pelvic plexus. It also derives nerve supply from the third and fourth sacral nerves.

The pelvic plexus also sends nerves to some of the branches of the internal iliac artery that do not run to viscera. These branches are the obturator, the ileo-lumbar, part of the sciatic, the lateral sacral, and the gluteus.

ANATOMY

The sympathetic of the viscera

The vesical and vaginal plexuses contain a large proportion of spinal nerve fibres.

Before finishing the account of the anatomical distribution of the sympathetic in the abdominal and pelvic cavities, the ultimate arrangement of the fibres in the various viscera may be touched upon.

In the stomach, gangliated plexuses are formed between the layers of the muscular coat and in the submucous tissue. In each of these plexuses small ganglia are found.

In the intestines, the nerves, after leaving the course of the vessels, form a plexus—'Auerbach's'—between the longitudinal and the circular muscular layers, sending off fine fibrils into the muscles; also an important plexus, that of Meissner, in the submucous coat, from which fibres supply the mucous membrane, seeming to send branches to the epithelium.

In the rectum, besides these plexuses, the plexus of Auerbach is joined by the plexus pudendalis with considerable ganglionic enlargement. Fibres travel between the muscular fasciculi of both sphincters, and of the external longitudinal muscular layers and the levator ani. It is to be remarked that these plexuses are largely composed of non-medullated fibres.

In the liver the nerves ramify with the hepatic artery and its branches.

It is probable that each fibril terminates in an hepatic cell, but all demonstrable nerves lie on the outer side of the lobules. (Hering.)

By pathological observation and experiment it seems certain that the vasomotors of the liver rise in the neighbourhood of the fourth ventricle.

The smallest fasciculi of nerves in the liver contain only non-medullated fibres.

In the spleen the nerves are chiefly composed of Remak's fibres.

In birds and in carnivorous animals ellipsoid organs are found, involving the capillary terminations of the vessels. These are scarcely seen in rodents and in man. Müller says that when they are well developed fine fibres of Remak occur in the interior of their granular mass.

It is not proven, but not improbable, that many parts of the cerebral cortex may form vasomotor centres. The sympathetic system is so easily excited and inhibited by reflex action, that it is uncertain whether some of the effects of lesion in the crura cerebri, the optic thalami, &c., are direct phenomena or reflex. The upper part of the pons, and a minute point in the floor of the fourth ventricle, close to the extremity of the calamus, are manifestly important vasomotor centres, or at least the main centres for controlling vasomotor action all over the body. In the floor of the fourth ventricle also lie the centres for governing the vasomotors of some internal viscera, notably the liver. That this region is directly connected with spinal cord centres is proved beyond doubt. It seems probable that the fibres of communication pass by way of the anterior cornua.

ANATOMY

The sympathetic in the cerebro-spinal centres

Experiment shows that section of cord or hemisection modifies the temperature and the vascular dilatation of the parts below it. Doubtless each segment of the body owns a vasomotor centre in the portion of the cord with which it is connected. Hemisection of the cord at the level of the last cervical vertebra produces a higher temperature of the head than does a transverse section of the medulla oblongata. But each spinal cord vasomotor centre is not only connected with the one below it, but with the one above it, and through this with the chief inhibitory centre in the medulla oblongata. There is therefore, as has been already stated, besides the chains of ganglia lying close to the bodies of the vertebræ united to each other, and sending branches to the viscera and vessels, another series of ganglia of still greater importance, connected with the so-called sympathetic ganglia by the spinal nerves and rami communicantes, but also united to each other by fibres that run probably in the anterior cornua, and so connected anatomically with the higher centres in the medulla oblongata and pons, each lower centre being more or less inhibited by the one above it, all being subject to the general inhibitory action of the highest.

This, however, is only one portion of an extensive system. Every sympathetic ganglion is a vasomotor centre, each probably possessing some independence of action, though more or less dominated by the higher ganglia. This is seen in the heart, deriving as it does some of its vasomotor influence from the

The ganglia of the vessels

sympathetic cardiac nerves, but possessing certain ganglia in the sympathetic plexuses on its surface and within its deeper tissue. The chain, too, of cardiac ganglia is known in some animals to possess an independent action, shown by the contraction of the heart persisting for some time after its removal from all attachments to the body. In the abdomen again the splanchnic nerves may be the chief pathway for nerve influences; but in the abdominal cavity the solar plexus and semilunar ganglia are immense vasomotor centres; and connected with them lie almost innumerable ganglia around and within every organ, sending branches wherever contractile tissue is found. Nor is this all. On every vessel of the body, perhaps particularly in those of the extremities, vasomotor influence is not carried down from the highest centres by lines of long conducting fibres, but from minute ganglion to minute ganglion, placed on the vessel, and particularly at the bifurcation of vessels, down to within a very short distance from the periphery, so that, according to the intensity of the stimulus, the reflex arc may find its centre in the smallest ganglion nearest the surface, or if more intense, in those higher up; whilst only extreme stimulus calls into action the highest centres of all. In partial blushing, in partial pallor, in myxœdema, in some of the phenomena of the various eruptive diseases, this independence of the smaller ganglia is recognised.

The vasomotors of the head and neck, and of the contents of the cranium, are seen to be supplied from

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The ganglia of the vessels

the cervical ganglion of the sympathetic, but those of the extremities own a more complicated origin. They arise from four principal sources. In the upper extremity—(1) Fibres arise from the spinal cord itself with the roots of the brachial plexus. (2) Fibres from the inferior cervical and first dorsal ganglion of the sympathetic, which join the brachial plexus about the level of the first rib. (3) Fibres that arise from the thoracic cord, springing from the roots of the third, fourth, fifth, sixth and seventh dorsal nerves, especially from the third and the seventh. (4) Fibres that arise from the inferior cervical and first dorsal ganglia, to be distributed directly to the vessels without association with the brachial plexus.

The vasomotors of the lower extremity have also several sources. (1) Fibres arise from the spinal cord with the roots of the sciatic and crural nerves. (2) Fibres from the abdominal portion of the fundamental cord of the great sympathetic, of which some join the sciatic and crural nerves, whilst others go directly to the vessels of the limbs.

In all vessels the vasomotors are arranged in two plexuses—one in the external tunic, one in the middle tunic of the artery. In the peripheral arterioles minute ganglia seem to exist almost everywhere on the external tunic.

The veins receive a less proportion of nervous filaments, having a less amount of muscular tissue. Krause considers that the nerve filaments terminate outside the muscular elements. Henocque and

Arnold, on the contrary, believe that they penetrate into the interior of the smooth fibres.

ANATOMY

The ganglia of the vessels

The sympathetic system contains some fibres like those of animal life, but smaller. But the majority of sympathetic nerves are fibres of Remak, pale, flattened, of small diameter, a rudimentary phase of nerve tube, presenting here and there oval, elongated, or fusiform nuclei, and being without any medullary coating. In the human embryo, and in that of all mammifers, the whole nervous system is constituted of fibres of this kind, and the olfactory nerve retains this constitution in adult life.

The cells of the sympathetic ganglia are surrounded by a thick capsule non-fibrillated, and with nuclei scattered on it. It has been thought that it was composed of connective tissue, but Beale and Remak believe that it is made up of nervous tissue. This conclusion is necessary, if the fibres of Remak are looked upon as nerve fibres, seeing that they are continuous with this capsule. Arnold and Beale have observed the existence of spiral fibres surrounding the nerve fibres at the moment of their entry into the cells of the ganglion.

'Each of the small plexuses of the arteries contain minute ganglia at the level of the crossing or the anastomosis of fibres. The terminal fibres separate from the plexus and end by punctiform swellings in the nucleus, or in the fibre, or extend to it in the interstices of the fibre-cells. The termination in the veins is the same. In the capillaries the minute nerve fibrils end probably in the nuclei of their walls.

ANATOMY

The ganglia of the vessels

'In the minute ganglia in the plexuses, in some the ganglion cells are interposed in a bundle of sympathetic nerve fibres; in others the cells are arranged along a small bundle of sympathetic nerve fibres, the substance of the ganglion cell being prolonged on the axis cylinder of a nerve fibre. In man the ganglion cell is multipolar, each process receiving a neurilemma from the capsule of the cell becoming a non-medullated nerve fibre.'

CHAPTER II.

THE PHYSIOLOGY OF THE SYMPATHETIC.

PHYSIOLOGY

THE sympathetic system is largely made up of, or mingled with, fibres from the cerebro-spinal nerves, even in the nerve-supply afforded to the viscera. It seems therefore impossible anatomically to dissociate the one from the other. They are as united, as mutually dependent on each other, as are the classes or the individuals in a well-ordered state. But as in the latter case each class has duties and conditions in which, though touching other classes at many points, and being more or less associated with and dependent on them, it yet retains a certain independence of action, so in the exquisitely ordered and balanced human frame the sympathetic system, though depending much for some of its power and usefulness on its relations with the cerebro-spinal axis, has in its influence on the circulation, and on the viscera connected with vegetative life, a certain independence of function ; it generates action of itself. It is continually not merely a nerve, a conductor, a humble handmaid of the cerebro-spinal axis, but in all essential points a nerve centre.

The truth lies, as it so often does, in the middle

PHYSIOLOGY

line. The associated dependence of one system of nerves on the other, whilst to each is assigned its own peculiar functions, is only in analogy with the arrangements of all the structures of the body, each of which has its own special purpose, and yet is unable to perform its full function except in relation with other tissues.

It may be said at once that it is very doubtful whether the sympathetic is the only nervous system of some of the lower animals. If this were so, the possibility of the independent action of this nervous system would need no argument. Nor is there much importance in the old distinctions between the two nervous systems, that the cerebro-spinal needs repose whilst the sympathetic does not, since it cannot be proven, and indeed is highly improbable, that the activity of the brain and spinal cord is ever completely in abeyance. On the other hand, there are certain points, apart from the close anatomical relationship between the two systems, which show how nearly the one is associated with the other. Thus:

1. It is found, by experiment, that most of the rami communicantes have their trophic centres in the spinal cord, only a small number having their trophic centres in the ganglia. This does not necessarily prove that the rami communicantes have their functional centre in the spinal cord.

2. Only a few of the rami communicantes enter the ganglion. Most of them only coast the side of the ganglion without entering, and join the main cord of the sympathetic.

3. Many of the vasomotors of the arm arise with the roots of the brachial plexus; and many of the vasomotors of the leg arise with the sciatic and crural nerves, without any origin from the sympathetic ganglia.

4. Lesions of the cervical cord may cause the same oculo-pupillary phenomena as lesions of the superior cervical ganglion.

5. Lesions of the cervical cord (seventh cervical and first dorsal) may cause the same vasomotor paralysis, evidenced by unilateral vascular dilatation of the face, ear and head, as is seen in lesion of the cervical sympathetic ganglion.

6. Hemisection of the lower third of the dorsal region of the cord produces vascular dilatation in the lower limb of the corresponding side, and so increase of temperature.

7. Hemisection of the middle of the dorsal region of the cord, or at the superior portion of it, produces greater dilatation of vessels in the lower limb, because the vasomotors of the lower limb have multiple origins, one being from the abdominal plexuses; and these are therefore not cut in a section of the lower dorsal. But this experiment also proves that the vasomotors of the lower limb that arise from the abdominal plexuses are indirectly also derived from the spinal cord.

8. All lesions of the spinal cord, and pressure on it, may enfeeble the vascular tone of the parts in relation by their vasomotor nerves with the region of the cord below the lesion.

PHYSIOLOGY

Independence of the sympathetic

But with a full appreciation of this mutual dependence of the sympathetic and the cerebro-spinal axis, there are certain phenomena which show that the sympathetic ganglia may manifest a partial independence.

1. The fœtus has been expelled from the uterus at, or almost at, full time, showing therefore a normal capacity of absorbing nutrition and a healthy circulation, without any trace of a cerebro-spinal nervous system, owning only the sympathetic system as the nerve element in its composition.

2. Parkes states that nutrition is properly carried on with complete destruction of the cerebro-spinal centres.

3. This fact renders probable the dictum of Golz, that the tone of the arteries is maintained by local centres, situated in their own immediate vicinity.

4. Reflex irritation of vasomotor nerves can be entirely limited to the particular organ or tissue supplied. Thus, in Vulpian's experiment, some days after the transverse section of the sciatic nerve, or of the brachial plexus, when the corresponding pulp of the paws of the animals had become quite pale and anæmic, he was able, by slight rubbing of these pulps, to cause a reflex congestion.

5. The not unusual fact of compression-myelitis (especially, strange to say, in the cervical region) being unattended with oculo-pupillary phenomena, or with symptoms of vasomotor paralysis. This has been noted by the writer in two cases of malignant tumour pressing on the cervical cord, and also in several instances of cervical pachymeningitis.

6. Many of the phenomena of blushing, of eruptions, of local congestions, point to the same idea.

7. Perhaps the phenomena of myxœdema and of the early stages of scleroderma are associated with functional independence of the sympathetic ganglia.

8. The fact of the continuance of the heart's action for a time in some mammals, after its separation from the body.

9. The peristaltic action of the intestine.

10. The reflex action of the stimulus of the blood upon vascular tone.

11. The phenomena of vasomotor neuroses of the extremities.

12. Irritation of the peripheral end of the cervical sympathetic nerve causes protrusion of the eyeball; section of it causes sinking of the eyeball and a slight flattening of the cornea. In the lids are sets of smooth muscular fibres innervated by the sympathetic, and by these the contraction of the lids is opened, and so the eyeball is uncovered.

13. From the cervical sympathetic some portion of the secretion of the parotid and of the submaxillary salivary gland is excited, and that on both sides.

14. Hermann, who looks upon the independence of the sympathetic as almost impossible, yet states that automatic and reflex co-ordinate movements, and secretions also, can be the outcome of the faculty of sympathetic ganglion cells, quite independent of the large nervous centres.

15. In the frog, after destruction of the brain and medulla oblongata, irritation will cause congestion of limbs.

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Independence of the sympathetic

16. In mammals, after section of the cord at the mid-dorsal region, sensory excitation of one posterior limb will cause reflex heat phenomena in the other.

17. The phenomena of pigmentation in the frog, when the brain and spinal cord are destroyed, show the same thing.

18. The richness of the sympathetic in central elements, like those of the brain and cord.

19. The wealth of the branches of distribution, gaining force the wider the distribution, and not the opposite.

20. It may perhaps seem somewhat fanciful to say that it seems necessary that the nervous system of the viscera should be more or less shut off from perturbation of our intellectual being. That this is only partially the case is due to the union and association of the two systems in so many ways.

Almost each histological unit has its own minute nervous system, which may suffice for it within feeble limits. A certain number of these elementary nervous actions are grouped about a common centre, as in a lobule of a gland. So farther on, from lobule to lobe, and so to external plexus (organ). And then several organs unite for one purpose, and are brought into relation by plexuses of the first order. It has been well said that the use of the central cord of the sympathetic is to make the animal and the vegetative worlds known to each other, so that revictualling should be proportionate to waste.

The question of mutual dependence of the two systems of nerves, or of the partial independence of the sympathetic ganglia, is necessarily reopened in considering the right of these ganglia to be looked upon as reflex centres. For coarse stimuli the spinal cord and medulla oblongata are the chief centres for vasomotor reflex action. But the heart is more or less independent of the great nervous centres. A reflex arc exists in its own substance. Reflex movements are excited from all sensory nerves, not only spinal but also sympathetic.

Claude Remard has shown that reflex action can occur in the submaxillary gland, when all nerves that communicate with the bulbo-spinal centre are cut.

If the superior cervical ganglia are separated from the higher nervous centres, oculo-pupillary phenomena can be reflexly excited. The phenomena of vascular contraction and dilatation can be excited in a limb that has been separated from the spinal cord by the destruction of the spinal nerves. This experiment has been considered to prove the independence of the vasomotor ganglia within the limb. This, however, is not anatomically certain. As long as a limb is attached to the body by means of its main artery there will always be a vasomotor connection with the trunk, some part of which nerve connection will have its origin from the spinal cord. The conclusion as to the independent action under such circumstances of the vasomotor ganglia of the limb is probably correct, but the data for this conclusion are not quite logically exact.

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The reflex action of the sympathetic

The phenomena of pigmentation in the frog can be excited reflexly by irritation of the cutaneous nerve, and may occur when the brain and cord are destroyed; so that the sympathetic ganglia must act here as independent reflex centres.

The ganglia of the uterus are manifestly centres for reflex action. This organ possesses within itself all the elements of a reflex arc. That the uterine centres can act independently, is seen in the occasional expulsion of the child after the death of the mother. The expulsion of fæces per anum after the death of the patient shows also that the sympathetic ganglia of the intestines are centres for independent reflex acts.

In a very interesting paper read at the International Medical Congress in London, Dr. Woakes speaks of the inferior cervical ganglion as a correlating nerve centre. It can only have this function as being a great reflex centre. Dr. Woakes has formulated, anatomically, data that are occurring to all practitioners every day of their lives; and his paper affords additional evidence both of the independence of at least the vasomotor portion of the sympathetic, and also of its multifarious connections with the cerebro-spinal system. He shows the association between injury of the nerves of the brachial plexus and loss of consciousness. The shock resulting from the concussion of the brachial nerves is propagated to the inferior cervical ganglion, and thence reflected as a wave of vessel-dilatation to the vertebral artery. This dilator wave is appreciated

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first of all in its peripheral branches, producing an immediate large accession of blood in them. This effect on the internal auditory branch of the dilated vertebral artery, the sudden tension of the intra-labyrinthian fluid, produces the phenomena of falling and unconsciousness. Through the inferior cardiac nerve from this ganglion great agitation of the heart may be induced. The vertigo attending indigestion under certain circumstances is an illustration of the same correlation, sometimes with, sometimes without a sensory aura. This vertigo may also be associated with mottling of the hands and forearms, due to congestion of the superficial arterioles.

The reflexes, of which the sympathetic ganglia, especially some of the abdominal, are centres, are too numerous to mention. The syncope induced by a blow over the solar plexus, the palpitation and even faintness consequent on indigestion, the vomiting and depression of spirits set up by the passage of a gall-stone, the transient hemicrania produced in some people by the presence of ice in the stomach, the flux from the intestinal vessels as a sequence of the irritation of some foreign body in the canal, some at least of the phenomena of renal calculus, the peculiar pulse of peritonitis, the collapse in perforating ulcer of the stomach and intestine, perhaps the increased circulation of blood in the liver and the augmented secretion of bile following injury to the solar plexus, are all instances of this. That in this region of reflex activity the great nerve centres play a most important part, will be seen in the considera-

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tion of the neuroses and of all emotional phenomena. It is only sought here to point out that, under certain conditions, the sympathetic ganglia can act as independent centres for reflex arcs. Especially is this taking place, as each blood-wave, in its passage along the whole arterial system, forms the stimulus to the eisodic conductors to one after another of those minute sympathetic ganglia that are found along the course of all the arterial canals, having for their function the reception of these stimuli from the fibrils excited by the blood-wave, and reflecting the order for contraction down the exodic fibres to the vessels again.

In those forms of hysteria that depend on definite uterine or ovarian lesion, the deep-seated sense of pelvic uneasiness, nearly similar in position and sometimes equalling in intensity the sacro-coccygeal pain attending piles, the paresis of intestine evinced by meteorismus, the increased flow of limpid urine, the vomiting, the hiccough, the frequent diarrhœa, the palpitation, the faintness, the sighing respiration, the globus, the difficulty in deglutition, the blushing, the dilated pupil, the tears, the tinnitus, the excitation of the emotional area, the occasional epilepsy, melancholia, mania, to which such patients are liable, are all examples of afferent irritation carried to the solar plexus, and thence from ganglion to ganglion of the sympathetic chain, to the three cervical ganglia; thence to the eye, the cerebral vessels, and the medulla oblongata.

A reflex irritation of stomach may frequently be

the result of pulmonary lesion. Many such instances may be met with in the domain of the visceral neuroses. Certain lesions of the kidney may perhaps be reflexly irritative, and cause paraplegia. According to Jaccoud, this paraplegia is a paralysis of the sympathetic; whilst Weir Mitchell thinks it is due to paralysis of peripheral origin. The latter observer has seen a wound of the nerves of the lower limb determine a paralysis of the upper limb.

In speaking of the sympathetic ganglia as reflex centres, it is impossible to resist a slight reference to so-called reflex paralysis. Brown-Séquard's experiment showed that the vaso-constrictors can be set in action in the spinal pia mater, and therefore probably in the spinal cord itself by reflex irritation. He obtained these results by ligature of the renal nerves, and that, too, on the corresponding side of the spinal pia mater. This constriction of spinal vessels causes anæmia of cord, and thereby paralysis. He gives instances of paraplegia caused by uterine lesion, by enteritis, colitis, and especially the ulcerative colitis of dysentery, by worms in the intestine, by pulmonary and pleuritic affections, by erysipelas of the leg, by lesions of the knee-joint, by neuralgia, dentition and diphtheria. Our own Stanley drew the attention of the profession to this form of paraplegia following diseases of the urinary bladder. Brown-Séquard's results are, however, exceptional. Ligature of the renal or suprarenal nerves will cause temporary constriction of many vessels, and might even, by its effect on the spinal

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vessels, induce a certain amount of anæmic paralysis. A somewhat similar condition is frequently met with in the obstinate constipation consequent on the irritation of a renal calculus, particularly of the left kidney. But it is certainly against all analogy with what is usually seen, to suppose that vascular contracture to the extent of causing paraplegia will be persistent. And it has been proved, in cases similar to those mentioned by Stanley, that the paralysis was due not to reflex irritation, but to a definite abnormal condition of the peripheral nerves, that affected the cord by lines of anatomical transmission.

That the irritation in one organ can be reflected through a sympathetic ganglion as a centre of a reflex arc is a fact that is the very essence of the pathology of the sympathetic system. As before stated, it is seen in irritation of stomach the result of pulmonary lesion, in the influence on the heart of irritation of the solar plexus, and almost any of its secondary ganglia, in the innumerable morbid phenomena that own uterine or ovarian lesions as their cause; but that such an irritation can reflect through a ganglion a persistent constrictive effect on a vessel seems to militate against the ordinary phenomena of physiology and of disease. And yet it is only fair to place upon record any facts that, exceptional as they may be, tell in any way against this commonly received dictum. The following instance is a case in point: A very sensible and active shopwoman, thirty-six years of age, gave the following account of herself. She believed that she had been born blind

of the right eye. At any rate, she remembered, when she was a very little girl, being taken to see an oculist in London, who said she would never see with that eye. She could not distinguish light with it. In January 1882 she had a canine tooth on the right side of the mouth extracted. She immediately become conscious of light, and in a few days entirely gained sight in this eye. The optic disc and retina were perfectly normal, yet for thirty-six years she was quite blind, apparently from some reflex influence connected with the alveolus of that canine tooth. With such an instance, it may be well not to speak of the impossibility of reflex irritation or reflex paralysis being persistent for long periods.

The innervation of the vessels opens up the question of contractions, dilatation, erectility, and reflex vasomotor effects. The function of the vasomotor nerves most frequently called into exercise is contraction. Many observers believe that the only nerve influence on the vessels is constrictive, and that arterial dilatation is merely the effect of paresis of the vaso-constrictors. The existence of vaso-dilators is, however, proved. Chapin sums up the history of these nerves in the following terms: 'They were described in the Chorda Tympani, in 1858, by Claude Bernard, who also found them in the kidneys. Eckhard showed the existence of the nervi erigentes in the sacral plexus in 1863. In 1874 Vulpian found dilators in the glossopharyngeal. Schiff has demonstrated them in the superficial, petrosal and carotid plexuses and

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other places. The latter authors, as well as Stricker and Ostrowmoff, are of the opinion that there are a certain number of dilator fibres scattered, in connection with the constrictors, throughout the body. Among other authors, who hold to the double innervation of the blood-vessels, are Lepine, Haidenhain, Grützner, Frank, Kendall, Lucksinger and Bernstein; and it is their opinion that there are cells on the walls of the vessels, or in their vicinity, which receive from the great centres vaso-constrictor fibres, and which also receive other fibres, less numerous than these, which act as inhibitors to them, and which consequently are practically vaso-dilators; that fibres go directly from these peripheral ganglia to the walls of the vessels. Thus we have a nervous arrangement throughout the circulatory system, not unlike that found in the heart. A fact which bears strongly on this point is that shown by Dastre and Morat, that, after the dilatation caused by section of the cervical sympathetic, we can obtain a hyperdilatation by strong galvanisation. The electric current, ascending to them, destroys by over-stimulation the previously unimpaired activity of the peripheral centres. With reference to the reflex arc in the vasomotor system, Rouget and Vulpian find that, in cutting the sciatic, the vessels of the corresponding muscles on the opposite side contract and the temperature falls, whilst the reverse is true of the side operated on. According to Vulpian, the contraction is due to reflex action, caused by irritation of the sensory fibres of the sciatic; and this results in

contraction of the vessels, not only of the opposite side, but also over a large part of the body. Schüller finds constriction of cerebral vessels on irritation of the proximal ends of the divided posterior roots of the sciatic and other spinal nerves. In general, if the sensory nerves in any part of the body are irritated, there results a contraction of vessels, more or less generally, throughout the body ; but there may be also, in certain cases, dilated reflex as well as constrictor reflex. Snellen and Rouget saw the vessels of the ear of a rabbit dilate on stimulating the central end of the divided cervico-auricular nerve supplying the part ; and Callenfels got the same effect by simply pricking the ear. So, on irritating the vagus, we get dilatation of the abdominal vessels by means of dilator fibres in the splanchnic (M. & E. Cyon). According to Vulpian, any sensory irritation, besides causing a general contraction of vessels, causes a dilatation of vessels in close proximity to the seat of irritation. Thus we get redness of the skin wherever we apply an electrode, a marked degree of heat or cold, or any other form of local irritation. We may even get a local dilatation and evanescent erythema from the action of intense light, as that produced by the carbon points of an electric light, even when it is too distant for heat to affect the part. So we constantly find throughout the alimentary and genito-urinary tracts, as well as in the superficial parts, variations in the circulation due to reflex action.'

Vivid contraction of arteries is sometimes followed by dilatation, due to the enfeeblement of the

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contracting nerve fibres. Slight irritation on the skin will produce a white line (vaso-contraction); more severe irritation a red line (vaso-dilatation), both reflex. The meningeal line, a red line easily excited on the trunk and limbs, and especially on the skin over the abdomen, is common also in typhoid. A pale line can be induced easily by very slight irritation at the beginning or in the convalescence of typhoid, not in the full course of the disease. In paraplegia by compression, white lines are induced more easily than red. In sciatica with spinal or membranous lesions, red lines are very easily induced. In a case of progressive muscular atrophy, slight irritation over the atrophied muscles gave a red line more easily than elsewhere; and so in a case of cerebral hemiplegia from hæmorrhage or softening. In hysterical hemianæsthesia a red line was induced less readily on the anæsthetic side.

In many, cold induces at first redness of the fingers from dilatation of the vessels; but this is probably not due to a simple result of reflex vaso-dilator action, but to contraction of arterioles with capillary and venous stasis. If the action of cold is prolonged, the fingers become white, the result of energetic contraction of vessels.

All arteries do not contract with the same power. Those of the brain, spinal cord, and glands are most contractile. Most of the veins are also contractile, but the muscular element being less abundant, the veins do not react to stimulus so well as the arteries. The veins of the brain and the sinuses of the dura

mater have no muscular tunic, and are not contractile.

The nervi erigentes of Eckhard, usually one on each side, spring from the sacral plexus, generally the first and second sacral nerves. Their direction is along the sides of the sacrum towards the lower part of the bladder and prostate, joining the hypogastric plexus. It is difficult to follow these nerves beyond this plexus, but they evidently communicate with fibres which terminate in the corpus cavernosum of the urethra. This region is supplied also from the pudic nerves, but their section causes no modification of the circulation in the corpus cavernosum. They play, however, an important part in the phenomena of erection, for it is by them that normally the centripetal excitations pass, that provoke erection.

Constriction of efferent veins does not account for erection, for their ligature does not produce it. Their constriction can only favour erection. There are some small ganglia and nerve-cells in the course of the erector nerves. These nerves probably act by dilating the arterioles.

Erection is either dependent on cerebral excitation, due to direct irritation of the vaso-dilator fibres contained in the erector nerves (as in emotion), or is the result of reflex action made by means of the spinal cord: this stimulus starting from a morbid irritation of the genito-urinary organs (inflammation of the urethral canal, pressure, &c.) or by absorption of cantharides, which irritates the urinary canal.

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Erectile phenomena are met with more or less clearly in the internal genital organs, in the uterus, the ovaries, &c., organs which possess a muscular tissue and a highly developed vascular apparatus.

Budge's genito-spinal centre is the ordinary centre of the arc for reflex erection. But erection also occurs in lesions of the cervical and dorsal cord, especially in cervical lesions from hanging, also in lesions of the cerebellum. Vulpian thinks the latter is connected with pressure on the medulla oblongata. But there is no doubt that lesions of the central part of the cerebellum lead to priapism, as this part of the cerebellum has some physiological connection with sexual excitement. Lesions of the lateral portion of this organ have no influence on erection.

The iris has a certain degree of contractility. The movements of the iris, however, are not due to modification of its vessels, but to contraction of its muscular fibres.

The chorda tympani is a typical instance of a nerve which, when excited, causes dilatation of vessels. This vaso-dilator action of the chorda tympani is independent of its action on the secretory function of the submaxillary gland. This independence of action of the chorda tympani is seen in animals poisoned by a strong dose of curare: in them secretion is abolished long before the action of the vasomotor nerves is lost.

The vaso-dilator nerves exercise a sort of suspending action on the vaso-constrictors—a true action of arrest, like that of the vagus on the heart. The

vaso-dilators are not antagonistic to the vaso-constrictors, unless excited. Section of a vaso-dilator nerve, as the chorda tympani, causes no change in the colour of the tongue, this organ receiving fibres from the sympathetic nerves that follow the course of the lingual artery, and also from fibres by way of the hypoglossal, lingual, and glossopharyngeal nerves. It seems probable that the sympathetic vasomotors are constrictors, and that the vaso-dilators come from the cerebro-spinal axis. But Vulpian, by his experiments on the splanchnics, thought that the dilator nerves might belong to the sympathetic system ; and Schiff thought that dilators are everywhere associated with constrictors in the sympathetic system, but that the constrictors are more numerous. It should be remembered also that the splanchnics contain a large proportion of spinal nerves.

The mutual action on each other of vaso-dilator nerves, acting through the small ganglia on their course, and of vaso-constrictors, plus the factor of cerebral inhibition, preserves vascular tone ; section of the great sympathetic dilates the vessels more than is normal ; on the other hand, irritation of this nerve contracts the vessels almost to complete effacement of their calibre. It seems certain, therefore, that the physiological state of the vessels is that of mean contraction—in other words, vascular tone ; and the preservation of this tone is one of the chief offices of this system of nerves.

Of the brain, the cord, the ganglionic plexus on

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the arterial walls, the sympathetic ganglia, a pathological standpoint enables us to choose the sympathetic ganglia as possessing a primary, if not a wholly absolute influence on vascular tone, associated with the vasomotor centres in the medulla oblongata and spinal cord.

The motor nerves that preside over the muscular contraction of vessels, and rule the local circulation, are the nerves that issue mainly from these ganglia of the great sympathetic, creep along the arterial walls, and can be followed into the middle muscular coat of the arteries. The vasomotor apparatus therefore is in a state of permanent activity, never in repose, never inert. The muscular tunic of the vessels is in a state of semi-contraction—in other words, of vascular tone. Variation in this tone will be the necessary sequence of various modifications of the nervous apparatus. This tone is modified by alterations in the vessels themselves, atheroma, sclerosis, fatty, calcareous, and amyloid degeneration, senile changes, syphilis, scrofula, alcoholism, &c.

There is at least some reason to believe that aneurism sometimes results from those changes that affect the vasomotor nerves, the consequent loss of tone permitting the formation of aneurismal dilatations.

This semi-contraction is in permanent strife against the excentric pressure exercised on the walls of the vessel by the blood.

That this condition of contraction may be induced by the action of two sets of nerves, each somewhat

inhibiting the other, is only in analogy with what is seen in the sphincters, in the muscles of the face, in the balance between the extensors and flexors of the hand and foot. It is in still closer analogy with the mutual inhibition of the vagus and the sympathetic on the heart itself. The arteries possess not elasticity only, but contractility also; the small arterioles contractility only. Vulpian thinks that at the moment of death the arteries undergo a vasomotor excitation, which ensures their emptying themselves, just as is often seen in the rectum, the bladder, and even the uterus; and for this the vasomotor ganglia are necessary.

What would cause this excitation? Would the carbonised state of the blood excite the vaso-constrictors, just as it is thought by some to induce general convulsion? It is a mere hypothesis. It is more reasonable to believe that the vaso-dilators, being fewer in number, and far weaker in function than the vaso-constrictors, die first, or at least are so enfeebled by the process of dissolution, that they lose their power of inhibiting undue vaso-constrictor action. Their inhibitory function being withdrawn, the vaso-constrictors act as they would in health, were it not for the presence and action of the vaso-dilators, and effect an undue and complete contraction of the vessels.

A lesion of the spinal cord and pressure on it may enfeeble the vascular tone of the parts in relation, by means of their vasomotor nerves, with the region of the cord behind the lesion; but it has been proved

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by experiment that vascular tone persists not only after destruction of the medulla oblongata, but after destruction of a portion of the spinal cord; and that, to abolish it, the whole bulbo-spinal centre must be destroyed. But where a child has been born fairly nourished, without any trace of a cerebro-spinal centre, it is evident that there exists a possibility of the persistence of vascular tone, when the only reflex centres are the sympathetic ganglia.

This vascular tone is due to a reflex mechanism—a mechanism brought into action by incessant centripetal excitations, and these excitations are probably the blood-waves.

For a reflex movement several factors are necessary—a contractile tissue, centripetal fibres, a centre of reflexion, and centrifugal motor fibres. In a vessel the factors of this arc exist; the middle tunic of the vessel, the centrifugal vasomotor nerves, the bulbo-spinal centre, and in addition to it the sympathetic ganglion that may act, and probably does act, as an independent centre for reflexion; and lastly, centripetal sympathetic fibres in the vascular walls, that are irritated or excited by the blood. Sensory nerves may often act as the centripetal fibres in these reflex actions.

All the phenomena of reflex congestion and of reflex dilatation of the vessels from any cause are only instances of enfeeblement or abolition, more or less complete, more or less persistent, of the vascular tone. The reflex mechanism of vascular tone is seen best in the heart and arteries. Let there be from

any cause a constriction of most of the small arteries of the body, there is, as a consequence, increase in the arterial tension. The heart strives to overcome this excess of tension, and must employ more energy for this purpose; its contractions become more vigorous, more rapid. This effect is not purely mechanical, but is under nervous influence.

Under increased intra-arterial pressure, the blood in the ventricle also undergoes at the moment of systole, and of the opening of the sigmoid valves, an excess of tension. This impresses some excitation at the endocardial extremities of the centripetal nerves of the heart, in this case the vagus. This impression is carried up to the bulb, from which and from the cervical cord is reflected a centrifugal irritation, by way of the sympathetic and its ganglia, to the intra-cardiac ganglia; and so result increased energy and increased rapidity of the movements of the heart.

The inverse phenomena, dilatation of vessels, induces inverse conditions, but the mechanism is nearly the same, except that a centrifugal nerve will be the spinal accessory.

Reversing the order of the phenomena, if the left ventricle, from any cause, be abnormally full of blood, the special impression on the peripheral extremities of the cardiac nerves is carried up by the depressor nerve—a branch of the vagus—to the bulb; and thence, by means of dilator nerves, a general reflex dilatation of vessels takes place, and especially by way of the splanchnic nerves on the

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vessels of the mesentery, and the heart is relieved of its pressure. So, once again, if an abnormally small amount of blood be in the heart, the reflex action originates from the cardiac nerves, and will react on the vaso-constrictors; the vessels contract, and the blood, receiving an increased vis a tergo, flows more abundantly to the heart. Thus the heart may, up to a certain point, play the part of regulator of the vessels, or at least it exercises a certain influence on their tone; whilst inversely, the vessels, too, rule, up to a certain point, the energy and frequency of the movements of the heart.

Nor is it necessary that the bulbo-spinal centre should always be the centre for these reflex arcs. The sympathetic cardiac ganglia, the ganglia of the cardiac plexuses, the ganglia in the walls themselves, may be the centres for action of the heart; whilst for the vessels the centres may be sought in the minute ganglia round the vessels themselves, and even in their walls.

The depressor nerve Ludwig and Cyon found on each side of the neck, arising from the internal surface of the heart, and mounting up to unite itself to the vagus, and so reaching the medulla oblongata. In rabbits this nerve is isolated, but not in all animals.

This nerve is formed of centripetal fibres, going from the heart to the medulla oblongata, and is sensory. Irritation of this nerve causes diminution of arterial pressure, and dilatation of all the arterioles of the body, and acts chiefly by causing dilatation of

the abdominal vessels, and so widening a considerable derivation of the blood. The mechanism of this effect is by the depressor nerve to the medulla oblongata, thence down the cord to the splanchnic nerves, and so to the abdominal vessels. It is by the extreme dilatation of the abdominal vessels that the extensive derivation of blood is effected, but it is doubtful whether this derivation of blood is enough of itself to cause death. It takes some part in the causation of death from a blow on the epigastrium. Such a blow may cause arrest of the heart's action. The excitation is carried from the semilunar ganglia to the spinal cord by the splanchnic nerves, from the cord to the medulla oblongata, from the medulla oblongata to the heart by the vagus, paralysing cardiac movements. But this paralysis of the heart is rendered more probable by deficient blood stimulus, the derivation of blood in the abdominal vessels starving the heart, so to speak.

The depressor nerve does not only arise from the trunk of the vagus, but one of its roots comes from the superior laryngeal branch of the vagus. It receives filaments from the inferior cervical ganglion.

Intra-arterial blood pressure is the product of several factors: 1. The constantly renewed introduction of waves of blood into the aorta by the ventricular systole. 2. The resistance experienced by the blood in traversing the small vessels. 3. The reaction of the elastic and muscular walls of the arteries on the blood. The second factor is the most

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important, and this is much under the influence of the vasomotor nerves.

Anything that causes paralysis or paresis of the vaso-constrictors of a vessel increases the blood pressure in it. Anything that unduly stimulates the vaso-dilators increases the blood pressure. In a word, healthy vascular tone is antagonistic to great blood pressure.

1. Thus, local arterial blood pressure is augmented in that enfeeblement, or even abolition of vascular tone after destruction of part of the spinal cord, especially of the anterior superior part of the cord or of the bulb.

2. Also in that diminution of vascular tone, seen in disease of vessels, senile change, atheromatous, syphilitic, and especially that arterial change that obtains in renal degeneration.

3. Under the influence of certain toxic agents, belladonna, alkalies, carbonic acid, &c.

4. Arterial blood pressure may be increased if the sympathetic ganglia are abnormally stimulated, even when vascular tone is healthy. The palpitation induced may be so great as partially to overcome the normal vaso-constriction.

5. Arterial blood pressure is increased, if, the sympathetic cardiac branches being normal, palpitation may result from paresis of the accessory branches of the vagus, in which case the palpitation would be continuous.

6. The action of the heart would go on for a time by means of the cardiac ganglia alone, if the in-

fluence both of the cardiac nerves and of the vagus were removed; but absolute palpitation would not ensue unless the paralysis of the cardiac nerves had caused such dilatation of the coronary arteries as would increase considerably the amount of blood brought for the nutrition of the heart, and so stimulate exceedingly the cardiac ganglia. It is probable, in such a case, the vasomotors of some of the vessels would also be paretic, and so there would ensue a double reason for increased blood pressure. Palpitation from terror would be by means of paralysis of the cardiac branches of the vagus; palpitation from indigestion, or from a gouty condition of blood, would be by way of stimulation of the sympathetic cardiac nerves, or of the ganglia from which they proceed. The ganglia in the centre of the heart are the reflex centres through which the excitation of the blood reaches the muscular apparatus. In suspense the action of the heart is short and sharp; in fear, almost paralysed; under excitement, usually intermittent.

Apart from the lesions of the cardiac walls themselves, or of the valves of the heart, or from paralysis of the accelerator nerves, blood pressure is lowered in the arteries mainly by the action of the depressor nerve already alluded to. On section of this nerve irritation of the peripheral end has no effect, but irritation of the central cord causes pain, lowers the pressure of the blood, and slows the heart. By the mechanism of the depression, viz.—a reflex action exercised on the splanchnic nerves, producing

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relaxation of the intestinal vessels—a large way is opened to the passage of the blood of the arteries into the veins, and pressure is lowered. It is not the activity of the constrictor fibres of the splanchnic vasomotor nerves, but the reflex suspension of their activity that is obtained by the irritation of the depressor nerve.

Dr. Broadbent believes that the main cause of arterial tension being resistance in the peripheral circulation, this is met with much more commonly in the capillaries than in the minute arterioles. This resistance may be met with in the minute arterioles on application of irritants to them, and in excitement, nervousness, &c. In hysteria this is protracted, with excessive amount of dilute urine. In the early stages of acute diseases of the brain, or throughout some chronic affections of the brain or spinal cord, it is sometimes found, and may even be unilateral. The heart may be stopped by the resistance induced, as in rigor, or in the cold stage of malignant ague, or in some cases of angina pectoris. This resistance is, however, more common in the capillaries. The affinity between the material of the capillary tubes and the liquid contained in them influences the passage of the liquid. This normal relation may be disturbed by changes in the tissue and in the blood. Inflammation is an instance of a change in tissue. In acute renal dropsy there is change in the blood. A change in the blood is the cause of arterial tension from capillary modifications in asphyxia or apnœa.

Most of the conditions of arterial tension are attended with accumulation in the blood of imperfectly oxidised nitrogenous waste. This is seen:

'1. In renal disease of all kinds, except acute suppurative pyelitis, nephritis, and tuberculous and amyloid degeneration.

'2. In gout, both acute and chronic.

'3. In lead poisoning, albuminates of lead are formed too stable to readily undergo dissociation and oxidation.

'4. In pregnancy always, either from general augmentation in the volume of the blood, or from the presence of effete matters derived from the fœtus.

'5. In anæmia, especially chlorotic anæmia. The deficient red corpuscles are not enough to carry sufficient oxygen for the oxidation of effete matters.

'6. In emphysema, in chronic bronchitis, and sometimes even in phthisis, partly from general fibrotic changes in the tissues as well as in the lungs, but mainly from imperfect aeration of the blood. Arterial tension is the rule with mitral stenosis.

'7. In inherited tendency.

'8. In constipation, which causes temporary increase of tension, and so may induce apoplexy or syncope.

'This resistance in the peripheral circulation is not quite the only cause of arterial tension. The capillary outflow being normal, the quantity of the blood poured into the aorta by the heart may be excessive. This is generally temporary, or the volume of blood, as a whole, may be too large, so that the arteries,

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capillaries, and veins may all be too full. This is seen in plethora, and for a time in the early stage of albuminuria.

'The consequences of arterial tension are manifold:

'1. The effects on the heart. Increased work is thrown on the heart. If the obstruction comes on gradually, hypertrophy is the result; if suddenly, dilatation. Dilatation, too, may occur where an hypertrophied heart gets fibroid or fatty; or from mental shock, or during acute illness, or from strain. In acute albuminuria there is, first, marked dilatation with diffused apex beat and a short first sound; secondly, dilatation partially compensated by hypertrophy. The impulse being now more heaving, the first sound over the interventricular septum reduplicated, and with accentuation of the aortic second sound. In acute albuminuria the absence of high tension is often of fatal augury. The heart does not rise to the occasion.

'2. There are functional derangements of the heart, palpitation, irregular action, a sense of oppression, from increased strain on the heart, due to arterial tension.

'3. The violence of the strain may set up valvulitis, with thickening, contraction, obstruction, leakage, even rupture of the aortic valve without any exertion.

'4. Aortic changes, atheroma, dilatation, and thinning; even aneurism.

'5. The arterioles become thickened and tortuous, and sometimes affected with senile cretaceous deposit.

'6. Secondary effects may result from changes in the heart and vessels; as, for instance, cerebral hæmorrhage.

'7. With a weak and fatty heart there will be imperfect blood supply to the tissues; thence general softening of the brain.

'8. Convulsions. Uræmic convulsions are not due to the presence of urea, but to derangements of the cerebral circulation from high arterial tension in kidney disease. Venesection is useful in uræmic convulsions by modification of the cerebral circulation. Convulsions may occur in arterial tension without kidney disease, and be cured by bleeding. In epilepsy, however, the condition of the pulse is usually the exact reverse of tension.

'9. Sometimes sleeplessness, dyspnœa, depression, loss of energy, of resolution, of memory, &c., with giddiness, fulness of head, pains and oppression in the chest, neuralgia, &c.'

The pulse of high tension is usually long, and full between the beats. If it is short, it will mean dilatation of the left ventricle and incipient failure of the heart. High tension pulse may be due to increased energy of the heart's action, the cardiac ganglia being stimulated by urea or some poison in the blood.

The arterial tension in anæmia is explained by Dr. Byron Bramwell as being due partly to the difficulty with which anæmic blood passes through the capillaries, but mainly to the contracted con-

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dition of the minute arteries, produced by stimulation of the vasomotor centre, which is shown to be stimulated not by the presence of the carbonic acid, but by deficiency of oxygen. There are no vasomotors in the lungs, and so in anæmia the blood passes through the lungs more easily than the normal blood.

If treatment is based at all on scientific principles, this question of arterial tension must never be lost sight of. Some remedies stimulate the heart, others depress it. Others again stimulate it in small doses, depress it in larger. Some drugs increase arterial tension, others lower it.

Of the drugs that stimulate the heart, ammonia, iron, digitalis, strychnia, alcohol in moderate doses, ether, belladonna, convallaria majalis, and, slightly, arsenic and jaborandi are the principal. Of drugs which depress the heart, prussic acid, antimony, bichloride of mercury, lead, nitrate of potash, emetics, aconite, tobacco, colchicum, chloral, chloroform, nitrite of amyl, quinine in large doses, lobelia, and veratrum viride are the chief; and croton-chloral, ergot, bromide of potassium, and gelseminum act slightly in the same direction.

Among drugs which in small doses stimulate the heart, and in large doses depress it, may be classed chloroform, aconite, opium, calabar bean, quinine, digitalis, camphor and veratria.

The drugs which increase arterial tension are bromide of potassium, the acids, ergot, belladonna,

nitrate of potash, squill, calabar bean, nux vomica, strychnine, turpentine, casca bark, and lead.

Those which lower arterial tension are antimony, alcohol, chloroform, nitrite of amyl, chloral, quinine in large doses, veratrum viride, gelseminum, and jaborandi.

Taking, then, some of these drugs in order, it is found that many of them influence both the heart and arteries.

The acids may contract the smaller blood-vessels by reflex action, or by directly astringing them locally. They arrest acid secretions, but stimulate those that are alkaline, as saliva, bile, and the secretions of the pancreas.

Prussic acid is a powerful sedative to the heart, from its influence on its nerves and muscles. It increases saliva.

Ammonia increases the force and frequency of the heart's action. It increases secretion from the bronchial and intestinal mucous membranes, and from the skin.

Aconite first stimulates the inhibitory cardiac ganglia, then depresses them. Its influence on the vasomotors is doubtful, but the sweating must proceed from some such influence.

Antimony acts as a sedative to the heart and the vascular system. It lowers arterial tension.

Arsenic stimulates the heart slightly.

Alcohol in moderate doses has a stimulating influence on the heart, probably by causing dilatation of the peripheral and cerebral vessels.

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Amyl nitrite accelerates the heart-beats, but weakens them. From its direct action on the muscular coats of the arteries, it dilates the arterial system and lowers arterial tension.

Belladonna generally arrests sweating. It paralyses the inhibitory nerve of the heart (vagus), and so gives the heart over to the sympathetic, increasing the force and rapidity of the heart's action. It raises arterial tension, contracting the small vessels by its direct influence on the muscles of the arterioles. Besides this, it dilates the pupil by paralysing the filaments of the third nerve of the iris, and so allowing the sympathetic to act on the radiating fibres.

Bromide of potassium slightly lowers the heart's action. It contracts the smaller arteries, and so increases arterial tension.

Colchicum acts as a cardiac sedative.

Chloral enfeebles the vasomotor system, especially probably the vasomotor centres in the brain. It paralyses the sympathetic ganglia of the heart and lowers the heart's action, even after the vagi are cut. It dilates the superficial vessels, and thus also lessens arterial tension.

Croton-chloral lowers the action of the heart less than chloral.

Chloroform at first slightly stimulates the heart, but soon paralyses the sympathetic ganglia of the heart, slows the pulse, and lowers arterial tension.

Calabar bean first excites the sympathetic, then depresses it. Under small doses the heart's action

becomes slower and stronger, and arterial tension is much increased. Later on, the heart's action becomes feeble and irregular.

Camphor in small doses stimulates the heart, but in large doses causes prostration.

Caffeine in physiological doses diminishes the frequency of the pulse and increases the energy of the cardiac contractions and vascular pressure. In poisonous doses it rapidly lowers blood pressure, paralysing vasomotor nerves. Convallaria majalis is a cardiac tonic and a diuretic.

Casca bark contracts the arterioles and increases the blood pressure. At first it causes slowing of the circulation from stimulation of the vagi; but eventually the action of the heart is quickened in consequence of paralysis of the vagi.

Digitalis is a tonic to the heart, making the beats slower and stronger, by its influence on the cardiac muscles, and by stimulating the inhibitory nerve (vagus). In prolonged doses the vagus becomes exhausted, and the heart is given over to the sympathetic. It increases arterial tension by stimulation of the sympathetic centres.

Ergot slightly depresses the action of the heart; it raises arterial tension by contracting the muscular tissue of the arterial walls.

Emetics have a sedative action on the heart.

Ether increases arterial pressure, and acts as a tonic to the heart.

Gelseminum slightly weakens the heart. It lessens the irritability of the excito-motor ganglia of the

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heart and diminishes arterial pressure by diminishing cardiac irritability and vasomotor tonus. Taken internally it contracts the pupil, but used topically it dilates it.

Iron acts as a tonic to the muscular structure of the heart

Iodoform diminishes arterial tension, and Professor Bozzolo inclines to the view that it exerts its influence specially upon the vasomotor centre.

Jaborandi produces great sweating from vasomotor paralysis, and consequent dilatation of the cutaneous arterioles. It rather increases the action of the heart.

Lead may induce sclerosis of the areolar tissue of the sympathetic ganglia, especially the cœliac and the cervical. The heart becomes slow, and the pulse harder and smaller from contraction and tension of the arterial system, probably from a primary effect on the sympathetic.

Lobelia depresses the action of the heart.

The bichloride of mercury lessens the action of the heart.

Muscarine causes direct respiratory paralysis and indirect cardiac, the latter resulting from stimulation of the cardiac inhibitory fibres.

Nitrate of potash in moderate doses raises arterial tension and slows the action of the heart.

Nux vomica stimulates the sympathetic system. It raises blood pressure by stimulating the vasomotor centres.

Strychnine causes a rise of the arterial pressure

and a contraction of capillaries. It stimulates vasomotor centres in the brain.

Opium first excites the sympathetic, then depresses it. The heart at first is slightly quickened, then slowed. Arterial tension is raised from the influence of opium on the cardiac inhibitory nerve. Opium causes some contraction of the smaller vessels.

Paraldehyde seems to have no action on the heart or on arterial tension, except in toxic doses, and then it causes (1) retardation, and (2) acceleration in frequency of the heart's action, with diminution in strength of the individual beats and a gradual fall of blood pressure. Quinine probably stimulates the great sympathetic. In small doses the frequency of the pulse is increased. In large doses arterial tension diminishes and the heart is depressed.

The hydrobromate of quinine diminishes arterial tension, causing paresis of the vaso-constrictors.

Squill causes increased arterial tension, and thus greater pressure on the walls of the Malpighian bodies, and so acts as a vasomotor diuretic.

Tobacco depresses the action of the heart.

Turpentine causes contraction of the smaller vessels.

Veratrum viride diminishes arterial tension: the pulse rate is lowered by direct action on the muscles of the heart and by stimulating the cardiac inhibitory nerve.

Veratria (the alkaloid of cevadilla) increases the action of the heart by stimulating its motor ganglia at first, and then slows and depresses it by exciting the action of the vagi.

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Cardiac nerves

The accelerator nerves of the heart come as the third filament from the inferior cervical ganglion. It is by this nerve only that the spinal cord can act directly on the heart. The branches from the cervical ganglia and the cervical sympathetic cord are probably those that rule the energy of the heart. Faradisation of the vagi causes no change in the vessels of the heart, but it arrests the movements of this organ. The inferior cervical ganglion seems to have no influence on the vessels of the heart. Baxt's experiments seem to show conclusively that the sympathetic cardiac branches have the power to increase the number of pulsations, whilst at the same time the length of the systole is decreased ; this being the reverse of the action of the vagus. According to Von Bezold, Lepine, and others, there are certain spots in gyri post and præfrontales whose excitation produces the same effect as excitation of the cardiac nerves themselves, and therefore Von Bezold claims that these parts of the encephalon are the cardiac accelerator centres. This is very uncertain. It is more probable that the portions of these gyri are simple vasomotor centres, and that the acceleration of the heart's action following irritation of these centres must be regarded as the result of an increase of the arterial pressure. Baxt, Stricker and Wagner believe the centres to be in the cervical portion of the cord, and that the fibres leave the cord by the rami communicantes as far down as the sixth dorsal vertebra, the upper fibres from the superior and middle cardiac nerves, whilst the lower

cervical unite with the thoracic branches at the annulus Vieussens, and form the inferior cardiac nerve. The peripheral ganglia, in the walls of the heart, are independent motor centres, or rather they can under certain circumstances act independently, as is shown by the fact that by their irritation the heart can be made to contract after it has been removed from the body. Besides this direct action on the heart, the sympathetic exerts indirectly the same influence by means of the reflex action excited by the depressor nerve of Ludwig and Cyon.

Opposed to all these is the vagus, par excellence the inhibitory nerve, or more probably so acting on the heart from its connection with the spinal accessory. It decreases the number of pulsations and increases the length of the systole; but it must be remembered that the connection of the vagus with the cervical sympathetic is tolerably close. By means of its ganglia it sends filaments to the superior cervical ganglion and receives branches from the same organ. This arrangement seems to be one of those exquisite instances of mutual inhibition, of which there are other examples in the body It may be presumed that, but for this arrangement, in certain lesions of the cervical sympathetic, the influence of the vagus would paralyse the motor action of the heart; or that, in certain morbid conditions of the vagus and spinal accessory, all inhibition of cardiac movements would be removed and fatal palpitation result, the current of blood being pushed too rapidly through the lungs to allow the due taking up of oxygen

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or the release of the products of systemic combustion. But the fact that these two antagonistic systems of nerves are united in various ways before they reach the organ over which their influence is exercised, connected. too, not only at the ganglia of the vagus, but by means of the bulbo-spinal centre from the nucleus of the vagus to the sixth dorsal vertebra, and even, as some observers think, to the first lumbar, prevents in large measure the evils that would ensue from the independent uninhibited action of one or the other of these nerves, and not only in health, but in many morbid states, effects that balance of cardiac action within limits that render life possible.

The heart's action may be maintained by the influence of the cardiac ganglia, which are part of the sympathetic system; but more probably the rhythmical movements of the heart are an expression of the peculiar vital endowments of its muscular tissue, as this rhythm is seen in the embryo heart with its parietes consisting of ordinary cells and without any nervous elements. The influence of certain drugs point to the same conclusion. Thus the chloride of barium acts not on the sympathetic system, but on the unstriped muscular system generally. This view of the rhythm of the heart depending on the properties of its muscular tissue, independent of nervous influence, is shared by Dr. François Frank of Paris. Dr. Roy considers that if the vagus is stimulated in the neck, it might produce a slowing of the rhythm alone without change in force, and vice versâ; and

from this it might be inferred that there were two sets of inhibitory fibres in the nerve in this situation. By stimulating the accelerator nerves also, he was able to produce acceleration without change in force, and increase of force without acceleration. There are, therefore, four sets of nerves controlling the heart differentiated physiologically, and also probably anatomically. The views of Dr. Gaskell of Cambridge are that the normal rhythm of the heart depends upon separate impulses, passing from the motor ganglia in the venous sinus to the auricle and ventricles, each impulse causing a contraction of the cardiac muscle, if that muscle is ready to contract; or more certainly on the rhythmical property possessed by the muscular tissue independently of any special nervous mechanism. The vagus does not cause inhibition by preventing the impulse from the motor ganglia reaching the muscular tissue. There are three different aspects in the action of the vagus —(1) acceleration, which usually occurs, but not always; (2) primary diminution of the force of the beats, which may extend to complete standstill, or may be absent altogether; (3) secondary increase in the force of the contractions, which may be primary. The vagus acts directly on the muscular tissue as a trophic nerve, not as a motor.

Recent observations seem to show that the action of the vagus on the heart is complicated. Physiologists have concluded—first, that there exists in the medulla oblongata a cardio-inhibitory centre, which is continually exerting a restraining influence

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on the heart; and second, that the action of this cardio-inhibitory centre may be intensified, i.e. the action of the heart may be still further retarded by—(*a*) direct stimulation, as by certain changes in the medulla itself; (*b*) impressions passing to it from the brain; (*c*) impressions passing to it from the peripheral parts (reflex stimulation). Some authorities also believe that the cardio-inhibitory centre may itself be inhibited—its restraining power taken off the heart, by impressions passing to the medulla from other parts of the central nervous system or from the periphery.

Dr. Gaskell shows—(1) that the vagus is able to modify all the great functional attributes of the cardiac muscle, viz.:

a. The rate at which automatic contractions are produced (the automatic rhythm).

b. The force with which the contractions, more especially the contractions of the auricle, are carried on. (The force of contraction.)

c. The facility with which the contractions are conducted by the muscular fibres. (The power of conduction.)

2. That the vagus sometimes produces depression, at other times exaltations of functions. He believes that the vagus may produce standstill or inhibition of the heart (auricles and ventricles) in the three following ways:

1. By producing cessation of its automatic contractions, i.e. by depressing the rhythmical power of the muscular fibres of the sinus in which the automatic rhythm originates.

2. By reducing the force of the auricular contractions so that they become invisible, i.e. by depressing the contraction power of the rapidly contracting reticulated muscular fibres of the auricle. The contractions, he states, may be so small as only partly to be visible, so that to the naked eye the heart appears to stand still.

3. By blocking the contraction-wave at the sino-auricular ring, i.e. by depressing the conducting power of the muscular fibres connecting the sinus and auricle.

All glands receive vessels, and are in relation with nerves. Under the influence of emotion the secretion of glands is increased, salivary, lachrymal, lacteal, gastric, intestinal, sudoriparous, &c. All glands are under the joint influence of cerebro-spinal nerves and of the sympathetic. Bernard thought that the former were the excitors of the activity of the gland, the sympathetic only the moderators; the one vaso-dilator, the other vaso-constrictor.

In the secretion of saliva the submaxillary, the sublingual, and the parotid glands are implicated. The two former are supplied by the chorda tympani, the latter by the small superficial petrosal, both nerves being branches of the facial. These cranial nerves are the vaso-dilator and secretory, whilst the sympathetic contains the vaso-constrictor fibres. But Giannuzi thinks that the sympathetic also contains secretory fibres, and that they arise at the third or fourth dorsal vertebræ, in a different space from

the vasomotors. Joenicke, however, denies that this is so in the parotid gland, and says that all the secretory nerves of this gland are in the petrosal. Eckhard and others place the intra-cranial centres for these nerves in the floor of the fourth ventricle. Other observers think that the cranial nerves contain secretory fibres for the secretion of the watery elements of the saliva, the sympathetic for the organic constituents. But there is no reason to say that irritation of the sympathetic causes a viscid saliva, and irritation of the cerebral nerves are aqueous, except in the sense that there is less serum in the saliva when the vessels are constricted. This does not prove secretory nerves in the sympathetic.

The pressure of the saliva in the excretory canals of the submaxillary gland, when this gland is secreting, is greater than the pressure of the arterial blood in the carotid. (It is not quite certain that the pressure is not greater in the vessels of the gland than in the carotid.) Various experiments show that the activity of the secretion is in no relation to the activity of the circulation necessarily, but is under the influence, direct or reflex, of the nerves distributed to the gland. Still it is proved that very often the vasomotors have much influence on the secretion of glands, as increase of blood in a gland leads to increase of secretion, but the secretory nerves must concur.

The submaxillary gland is generally set in action by the excitation of the fibres of the lingual nerve (by sapid substances). This excitation is carried to

the nucleus of the fifth nerve, is reflected in the medulla oblongata to the nucleus of the facial nerve, and thus sets in action the nervous fibres of secretion, and the vaso-dilator fibres of the chorda tympani. But the centripetal nerve may be the cervico-auricular, the sciatic, or any sensory nerve. A stimulation of these nerves causes contractions of most of the small vessels of the body; whence, increase of blood pressure; whence, dilatation of the vessels of the ears, and of the salivary glands. This irritation of sensory nerves produces less secretory effect than irritation of the lingual. The centrifugal nerve is not always the chorda tympani; it may be the sympathetic.

It seems probable that the salivary secretion is generally a reflex act. The movement of the tongue will excite it, or the presence in the mouth even of a tasteless substance. Much more frequently it is excited by the taste of sapid substances. But perhaps of all glandular secretions it is especially under the reflex influence of the nerves of special sense, not only of touch and taste, but particularly of smell also. The sight of food, especially of sapid substances, will excite the secretion of saliva, and often the same effect is produced by merely hearing the name of such substances mentioned. The very thought of food under certain circumstances increases the amount of saliva, whilst the secretion can be decreased by great mental agitation.

Tears.—The principal nerve of the secretion of the lachrymal gland is derived immediately from the

fifth nerve. 'Concerning the influence of the sympathetic on the secretion of tears, Herzenstein and Wolferz after watching the effects of irritation could formulate no definite conclusion; nevertheless the greater number of the latter's experiments, as well as some made by Vulpian, would seem to indicate that the sympathetic does exercise some control over the secretion, a theory with which Demtschenko's results agree.' That this is so seems also indicated by the influence of emotion on the lachrymal secretion.

A person under great sadness and distress can, by an effort of the will, keep his face like a rock, and prevent any movement of the orbicular oris, the depressor anguli oris, &c., but no effort of will can prevent the excito-secretory and vaso-dilator action on the lachrymal gland. The emotional phenomena of cerebral lesions, the greater the nearer these lesions are to the pons, teach the same thing. Excitation either of the lachrymal nerve, or of the sympathetic, increases lachrymation; but the secretion is thicker and less limpid if the sympathetic is excited.

Milk.—It would seem at first sight as if the lacteal secretion was in direct relation with the circulation in the gland. That this is so to a considerable extent seems certain. But, as in other glands with a secretion chemically complex, the secretory nerve fibres have a most important place. These fibres in the mammæ come from the fourth and fifth dorsal, and sympathetic fibres are largely associated with them. This gland is much under the influence of reflex excitation. The very first beginning of the

secretion of milk is a reflex act, due to the irritation of the fœtal movements in the uterus, carried in one of two ways, or perhaps in both: either by way of the sacral plexus to the cord, transmitted up the cord to the point where the nerves of the mammæ are given off (this is probably the case before the mother has any sensory impressions of the fœtal movements); or, when they are intense enough to be felt, the impression is carried up from the cord to the brain, and reflected thence to the dorsal region of the cord, and so to the mammæ; or perhaps from the uterine ganglia by means of their sympathetic connection to the semilunar ganglia, and thence to the dorsal sympathetic ganglia, and so either to the mammæ direct, or to the brain and spinal cord.

The touch of a finger, still more of the child's mouth, affords the afferent stimulus to the nerve centre of the secretion. It is a purely reflex act; and although the mother is generally sensitive to this stimulus, it is not necessary that she should perceive the sensation, as lactation will go on during profound sleep.

In giving directions to ensure good quantity and good quality of milk during the period of lactation, other things being equal as to the amount of food, of work, and of exercise, the two important points in practice are the avoidance of indigestion, and the avoidance of exciting or depressing emotions. Both these points prove the influence of the sympathetic on the secretion of milk. The diet of a nursing mother is carefully regulated, not because this or

that substance eaten materially alters the quality of the blood and so of the milk, as one of the secretions from it, but because certain aliments that are not rapidly digested excite an irritating influence on the mucous membrane of the stomach or bowels, and so on the nerve plexus beneath (an impression often not felt by the mother herself), that is at once transmitted to the lacteal nerves, at this period almost as sensitive as a galvanic calorimeter, and either inhibits the secretory fibres, or irritates the vaso-constrictors, and so may influence either the quality or the amount of the secretion. Still more are these nerves under the influence of emotion. The best wet-nurses are the most unimpressionable. Quiet domestic animals, with a love for the baby unalloyed by grave anxiety, free from all the trials and troubles of life, content to eat and sleep and take exercise without much thought, except for the day itself, are pattern nurses. Anger, passion, terror, wearing anxiety, not only hinder the secretion of milk indirectly by their effects on the general health, but directly by inhibition of the secretory nerves. Even where the secretion is not stopped, the quality of it is altered, and in extreme cases the immediate death of the child has resulted as if from poison. It is a question whether mammary abscess may not be caused by the emotion of fear. If the secretory fibres can be inhibited by fear, whilst the vasomotors are not, or if the secretion is altered in quality, and some spasmodic contraction of a lacteal tube occurs, there would be the materials for mammary abscess, though not the most usual ones.

Sweat.—The secretion of sweat is presided over by a distinct set of nerves. It is most probable that the spinal nerves contain the secreting fibres of the sweat glands, and that the sympathetic contains moderator or inhibitory fibres, such inhibitory fibres being possibly only vaso-constrictor nerves. Nerve fibres have been traced directly into the secreting cells of the sweat glands. There is considerable difference of opinion among physiologists as to whether the nerves that influence the secretion of sweat are spinal or sympathetic. 'Luchsinger says that these nerves for the lower extremity are contained in the abdominal sympathetic, and leave the cord by the first four lumbar and two or three last dorsal nerve-roots; whilst for the upper extremity they are in the spinal roots of the superior thoracic ganglion. Adamskiewicz believes that in the cord these fibres are contained in the anterior cornua, and are transmitted from centres on the surfaces of the brain.'

In speaking of hyperidrosis, it will be seen that section of the cervical sympathetic causes an increase of sweat on the side of the lesion. But, though sweating follows paralysis of the sympathetic, vaso-motor paralysis sufficient to cause extreme dilatation of vessels is not necessarily associated with sweating. It is certain, therefore, that one of the functions of the sympathetic is that of inhibiting over-action in the sweat glands, of maintaining tone in these organs; and that hyperidrosis depends, not on the vasomotor nerves, but on secreting fibres emanating from the

cord through the rami communicantes to the sympathetic ganglia. Seeligmüller asserts that the position of the sweat-centre is doubtful. It resides possibly in the spinal cord, possibly in the upper surface of the brain. That the irritation which excites hyperidrosis may be reflex, is seen in those cases of extreme perspiration of the palms of the hands induced by indigestion; and instances of perspiring feet, not only hyperidrosis, but osmidrosis, are not wanting, in which the exciting cause is to be sought in abdominal and pelvic irritation.

Although vasomotor paralysis is not necessarily accompanied by the secretion of sweat, the reverse statement does not hold good. The secretion of sweat is accompanied by vasomotor paresis; and if there be a paralysis of the vasomotor centres that preside over the vascular tone of the sweat glands, hæmatidrosis (bloody sweat) may result. It is certain that such bloody sweat is met with, depending on paralysis of the sympathetic fibres inhibiting the action of the sweat glands, plus an intense vasomotor paralysis of the vessels of these organs, and such a consequent congestion as may lead to rupture of vessels. Poincaré thinks that it is due to spasmodic irregular contraction of vessels, which imprisons the blood in kinks of the vessels, and exercises a strong pressure on it, and so ruptures the vascular walls. Cases of simulation and of the pressure of red fungus must necessarily first be excluded.

Gastric juice.—The stomach possesses nerves that supply the superficial and deep muscular tunics, the

gastric glands and the vessels. The splanchnic is the chief means of vasomotor supply, containing both vaso-dilator and vaso-constrictor fibres. It is the principal, but perhaps not the only, channel by which certain influences from the base of the brain are conveyed to the gastric vessels; the vagus having no vasomotor influence on the stomach. Lesion of the base of the brain, especially of the crura cerebri and the optic thalami, are sometimes associated with red softening of the stomach, or with ecchymosis of its mucous membrane. But the gastric glands are under the influence of a sympathetic plexus, having origin in the solar plexus and the semilunar ganglia, acting sometimes independently, sometimes in connection with the spinal cord. Here, too, the vasomotors are an important factor, regulating, as they must, the amount of blood necessary for the secretion from the gastric glands; but the nerves which rule the secretion itself are quite separate, and are probably filaments of the vagi. But the nerve supply of the gastric glands is not fully understood. The chief stimulus to gastric juice secretion is the presence of food in the stomach. Stimulation of any of the nerves going to the stomach has no effect on the secretion. The secretion will go on, under the stimulus of the presence of food, when all the nerves going to the stomach are cut. It is certain, therefore, that the ganglia in the walls of the stomach are sufficient centres for the act of secretion, although possibly in health they only play a small part in this function. The most reasonable view is that the vagi

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are the secretory, the sympathetic the vaso-constrictor nerves. Liable as sympathetic nerves and ganglia are to reflex irritations, it is not to be wondered at that, besides the influence of variations in blood-supply, the gastric secretion should be so easily modified and altered by irritations starting from distant organs, the brain, lungs, spinal cord, kidney, uterus, &c., to say nothing of the effect on it from emotion.

The intestinal secretions own similar anatomical elements, viz., vasomotor through the splanchnic chiefly, secretory by means of the mesenteric plexus, from the higher plexuses in the abdomen, into which filaments of the vagi largely enter. Lesions of certain brain regions act on the intestine, especially injury to the cerebellum, the corpora quadrigemina, pons, crura cerebri and cerebelli.

Bile.—In speaking of diabetes mellitus the special influence of the nervous system on the work of the liver will be again referred to. For the secretion of bile the vasomotor influence is transmitted through the splanchnic, the secretory from the hepatic plexus derived from the solar. Vulpian says the vagi are secretory nerves to the liver, but this is doubtful. Injuries to the solar plexus have been found to induce an increase in the secretion of bile; and this secretion, both in quality and quantity, is peculiarly under the influence of irritation from without, lesion of so many other organs, besides the liver itself, resulting in changes in the bile; whilst the effect of many remedies, generally a reflex effect, is also re-

markable. But not only has it been hitherto impossible to determine whether bile is formed from the blood of the portal vein or the hepatic artery, but the exact rationale of nerve influence is also doubtful. It is quite probable that the influence of the hepatic plexus largely depends on the filaments of the vagi contained in it.

Urine.—The renal nerves come—(1) from the great splanchnic through the solar plexus; (2) a few fibres direct from the small splanchnic; (3) a few fibres from the solar plexus itself. Probably the vagus acts through the solar plexus. Probably, too, there is the same antagonism between the splanchnic and the vagus, as in the salivary glands between the chorda tympani and the sympathetic. Section of all the nerves running to the kidney generally causes renal congestion. Section of the great splanchnic causes congestion, polyuria, and albuminuria. Galvanisation of the peripheral end of the cut nerve causes pain and cessation of the flow. Thus a form of albuminuria may exist from splanchnic lesion, and without real alteration of the renal tissues. The splanchnic contains at least part of the vaso-motor nerves of the kidney.

Irritation by calculus in the kidney may set up reflex constriction of vessels and arrest of urine. Vaso-dilators of the kidney also exist in some measure in the splanchnic.

It is a question whether the kidney can be considered a true gland, and the urine as a glandular secretion. It was an old observation of Claude

Bernard that the venous blood in the kidney is always red, because this organ produces nothing and only filters.

Section of the sympathetic in the neck, or of the cervical ganglia, or of the roots of the last cervical and first thoracic nerve in the spinal canal, causes polyuria. The higher nervous centres, therefore, act on the kidney through the sympathetic. Polyuria is also caused by pricking the floor of the fourth ventricle at a minute point in close approximation to the region, irritation of which produces glycosuria. Eckhard found the same polyuria by irritating the vermiform lobe of the cerebellum. It seems, therefore, that the regulation of the blood supply comes through the splanchnic, the secretory fibres, if this title may be used for the kidney, from the solar plexus directly. Vulpian thinks that the sympathetic contains both vasomotor and secretory fibres. The immense variations in the constituents of the urine, differing as they do in health at different periods of the day, in beautiful relation with the presence of gastric fluid in the stomach, as well as the extreme differences in these constituents in disease, are under the influence of the solar plexus.

Semen.—Movements of the vesiculæ seminales follow, according to Budge and Loebe, irritation of the trunk of the sympathetic from the fifth lumbar vertebra downwards. These fibres also arise in the centrum genito-spinale. The testes are the chief glands for the secretion of semen, the only glands for spermatozoa. That their secretion is under the influence

of emotion is a matter of daily experience. That it is favoured by congestion of the centrum genito-spinale is seen in the numerous instances of nocturnal emissions, that seem to depend on dorsal decubitus. But that their secretion under ordinary circumstances is much under reflex influence is proved by the process of coitus. The secretory nerves of the testis are derived from the spermatic plexus, which is a branch of the renal, and therefore only indirectly connected with the semilunar ganglia. Through the renal plexus it is also brought into close connection with the smallest splanchnic nerve, and thus with the spinal cord. Its vasomotor nerves are also derived from the spermatic plexus, and ramify on the branches of the spermatic artery. They penetrate the external wall and membrana propria of the seminal tubes, but the precise mode of their termination is still a matter of dispute.

In all these glandular structures the wonderful system of mutual inhibition seems to obtain. They all possess vasomotor fibres and secretory. They are all inhibited more or less from above, from brain, or cerebellum, or medulla oblongata, or spinal cord. Vasomotor congestion will not induce hypersecretion by itself, nor can secretion go on without a due amount of action of the vasomotors.

Probably the persistence of vascular tone is the most important factor for absorption. Thus, in the frog, absorption takes place when the nervous system is intact, and not when the cerebro-spinal

Physiology — Absorption

nervous centres are destroyed. This seems mainly due to the loss of vascular tone, and the arrest of the circulation, when the nervous centres are destroyed; because, if the circulation is normal, absorption can take place wholly irrespective of the cerebro-spinal nerves. The apparent paradox of the non-absorption of strychnin by the stomach, if the vagi are cut, is only another proof of the same fact. It is found by experiment that the absorption of strychnin by the stomach is always slow, even under normal circumstances. If the vagi are cut the movements of the stomach are paralysed, and so the poison remains in the stomach, and is not rapidly pushed into the duodenum, where the absorption is more active.

The nutrition of an organ must depend primarily on the amount of blood supplied to it, and on a normal condition of absorption. Both of these phenomena are largely, if not exclusively, under the regulating influences of the sympathetic.

The nervous centres exercise a direct influence on the nutrition of nerves. This is seen in section of the sciatic. Dilatation of the vessels of the muscles is an immediate but not a permanent effect. A gradual fatty degeneration of the nerve supervenes.

The nervous centres, too, exercise influence on the nutrition of the muscles. The changes in a muscle after section of its nerves are quite different from those in muscles that have become inert from other causes. They do not depend on vasomotor paralysis.

Section of the trigeminus nerve between the

Gasserian ganglion and the protuberance affects the nutrition of the eye; but the ocular alterations are less intense and less rapid than when the section of the nerve is made beyond the Gasserian ganglion. In debilitated animals, conjunctivitis and keratitis are caused by section of the cervical sympathetic, or by destruction of the superior cervical ganglion. Vasomotor paralysis is caused in the eye by intra-cranial section of the trigeminus. It acts as a predisposing cause to the ocular alteration, the exciting causes of which are foreign bodies.

Probably in section of the nerve, trophic branches coming from the brain are cut.

Section of renal nerves leaves the kidney structure intact; the lesions observed are due to disturbance of the circulation.

There are very few vasomotor fibres to the lungs in the vagi. Coincident section of both vagi causes death; but section of one causes lesions of nutrition in the lungs, that resemble catarrhal pneumonia. Is this from the connection of the vagus with the anterior pulmonary plexus, through the superficial and deep cardiac plexus, especially the former? It is likely. The pulmonary plexuses seem to send filaments mainly, if not wholly, to the vessels of the bronchi. Or are the results seen from section of one vagus due to partial paresis of the coronary plexuses, the consequence of this section interfering with the circulation of the blood through the lungs?

Vulpian considers that the atrophy of the testicles, the result of section of the spermatic nerves, is not

due to vasomotor paresis, with consequent dilatation of vessels, but rather to consecutive contraction of vessels following the paralysis.

Section of the cervical sympathetic is sometimes, but not always, followed by atrophy of that side of the brain.

Perforating ulcer of the foot is connected with disease of the nerves, and dilatation of vessels. Some instances of this morbid phenomenon have been seen in connection with tabes dorsalis. But perforating ulcer is not constantly due to the same causes. The labours of Duplay and Morat have proved that degeneration of the peripheral nerves is unquestionably in some patients the *fons et origo* of this affection; and in this respect the numerous cases in which it appears to follow section or compression of the sciatic nerves are fraught with a deep significance.

Traumatic alterations of the spinal cord have the same influence on the nutrition of muscles as section of the nerves. Very generally the nerves are diseased also. Infantile paralysis, due to an inflammatory lesion of the anterior cornua, and often of the antero-lateral columns; progressive muscular atrophy, a non-inflammatory lesion of the anterior cornua, and bulbar paralysis, all these prove the dependence of the nutrition of muscle on the nerve-cells in the bulbo-spinal axis, and not on disease of vasomotor fibres

Sir Charles Bell, however, thought that the sympathetic was concerned in progressive muscular atrophy. In this disease, besides lesion of the ante-

rior cornua, the cervical ganglia of the sympathetic may be converted into fat; but many observers have found them healthy. Jaccoud, however, thinks that the cervical ganglia are first affected, and Schneevogt and Jaccoud have recorded a case of progressive muscular atrophy with degeneration of the sympathetic.

Dr. Shingleton Smith has recorded a case of acute atrophy of the liver, in which there was an atrophic condition of nerve cells in the sympathetic ganglia; but it is doubtful whether these lesions stood to one another in the relation of cause and effect.

The spinal origin of some of the vasomotors of the leg is higher up than the origin of the sciatic nerve. If, therefore, atrophy of muscle occurs with lesions below the origin of the vasomotors, it must probably depend on lesion of centres extraneous to the vasomotor centres of the leg. Many physiologists think the sympathetic has no effect on nutrition; but, as Claude Bernard says, the sympathetic indirectly influences assimilation by variation of the blood tension. And the important fact remains that nutrition has gone on for months in an embryo, in which at birth there was no trace of a cerebro-spinal system. The facts of pathology go far to prove the correctness of the views of Mayer and Vulpian, that the nutrition of a part is maintained by a certain trophic influence, which is exerted by the nerves, motor, sensory, or secretory, by which the part is supplied; and that no other nerves of a solely trophic

nature are needed. All nerves own a trophic function in addition to their special one.

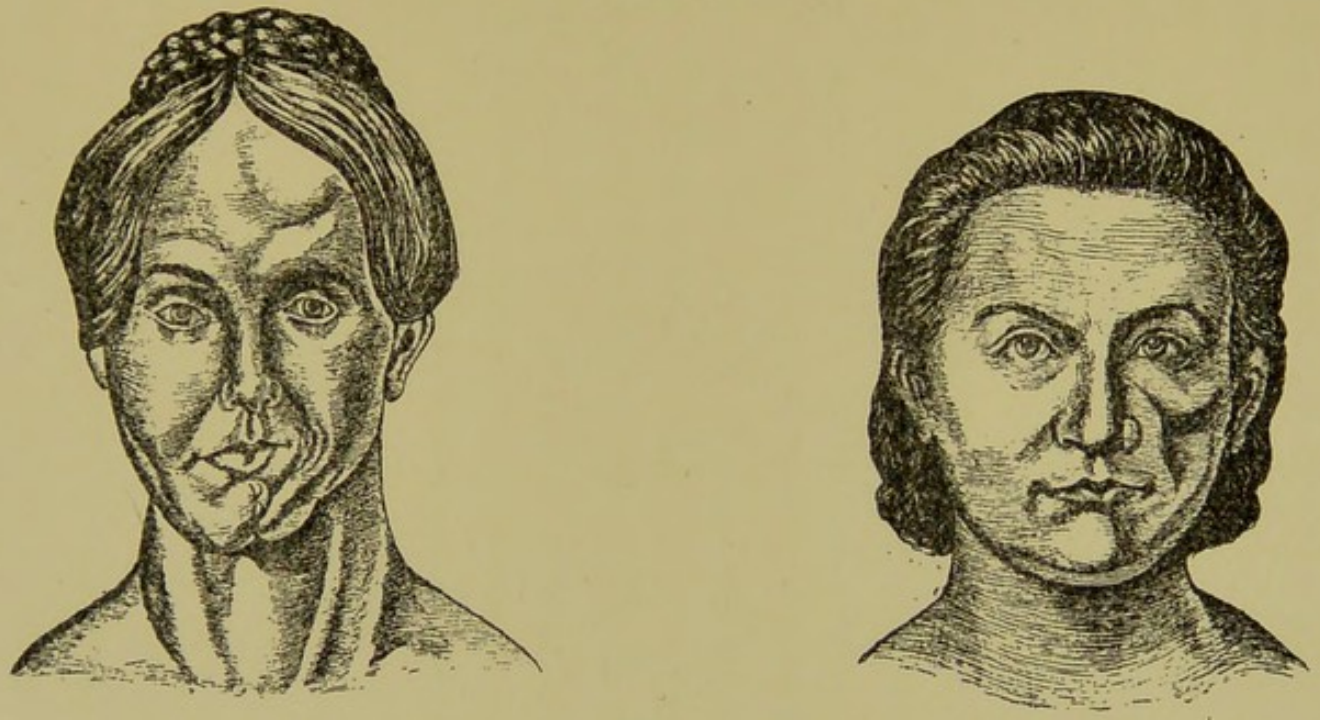
The difficulty lies partly in the fact that there are probably no nerves, motor or sensory, with which fibres of the sympathetic are not commingled.

Not only are the phenomena of partial atrophies, of infantile paralysis, of progressive muscular atrophy, constantly met with without any special lesion of the sympathetic, except in so far as variations in the blood supply are concerned, but the peculiar symptoms of progressive hemiatrophia facialis seem to have little or nothing to do with distinct sympathetic lesions.

Pierson, it is true, speaks of the sympathetic being affected, as evidenced by the prominence of the eyeball, and dilatation of the palpebral fissure, diminution of temperature in the auditory meatus of the affected side, and atrophy of the affected ear. Most authors mention some change of colour in the skin, pale or brown patches on the face and neck, sometimes preceded by an eruption of an œdematous or herpetic character. But neither paralysis nor irritation of vasomotor nerves, experimentally induced, give rise to simple progressive atrophy of all or most of the tissues. Romberg considers the lesion a tropho-neurosis. Baerwinkel places its seat in the ganglia of the trigeminus. Stirling thinks it is due to disturbed function of the vasomotor nerves included in the trigeminus, and destined for the vessels of the head.

Eulenburg and Guttman speak of slight atrophy

PLATE V.



Progressive Hemiatrophy of Face. (After Seeligmüller.)

Progressive facial hemiatrophy

of one side of the face as observed in some cases of injury of the sympathetic in the neck; and they quote Brunner's case, in which more or less of the phenomena of progressive facial hemiatrophy seemed to depend on persistent irritation of the cervical sympathetic. Dreschfeld agrees with Romberg. In his case the vessels were not affected; but this is not always found. Sometimes there are distinct changes in the vascular tone, not necessarily connected with the atrophy; the power to blush may be lost, and may be restored without any other improvement. Dr. Dreschfeld considers that in his case the atrophy was not due to any affection of the facial sympathetic, or of the motor or sensory portion of the fifth nerve, but that the disease is a tropho-neurosis following the course of the fifth nerve.

A case has been recorded by MM. Mierzejensky and Erlizky, in which an epileptic developed atrophy of all the muscles innervated by the third branch of the left trigeminus (the motor fibres of the lower maxillary nerve). The skin was atrophied, secretion normal, electrical contractility of the atrophied muscles good. There was nothing abnormal in the vasomotor domain of that side of the face. The authors considered that the affection depended on a central and very circumscribed lesion, which they localised in the floor of the fourth ventricle, near the locus cæruleus, in the motor nucleus of the fifth.

Another theory, that of Laude, denies the disease to be a neurosis at all, but a primary atrophy of the

PHYSIOLOGY — Progressive facial hemiatrophy

fatty tissues, the elastic tissue remaining unaffected, its retraction causing the falling in of the other soft parts and the contraction of the capillaries, the latter leading to further disturbance of nutrition.

Virchow says the exact seat of the disturbance lies within the domain of the peripheral nerves—that is, a primary inflammatory lesion of the throat and face escapes on to the nerves, and so to the ganglia.

That the symptoms are due to a neurosis is demonstrated by their association with headache, with paralysis, with irritation of the cervical sympathetic, or with epilepsy; neuro-paralytic ophthalmia, neuralgia, and anæsthesia of the fifth nerve often accompanying them. The disease may exist coincidently with migraine. The muscles take no share in the atrophy. The subcutaneous fat, the proper tissue elements of the cutis, and even the epidermoid structure, take part in the disease. The secretion of the sebaceous follicles is arrested; that of the sweat-gland persists, but is frequently diminished. The bones of the face, and even the nasal cartilages, share in the atrophy, and sometimes the tongue, the vault of the palate, the soft palate, and the uvula. The eye, if affected at all, shows lesions associated with intra-cranial disturbance of the fifth nerve, which may end in destruction of the organ.

In a case of Dr. Henschen, of Upsala, the symptoms seemed to own their origin in disease of one ankle, and to have either been reflex, or due to peripheral irritation that spread to nerve centres. In the very few recorded cases of definite recognisable lesion of

the cervical sympathetic in this disease there has almost always been an absence of oculo-pupillary phenomena. It seems certain, therefore, that all the branches of the nerve are not simultaneously implicated. Is not the connection between the cervical sympathetic and the phenomena of this disease less absolutely direct? The lesion may be one of the fifth nerve, whether of its sensory and trophic branches or of its trophic and vasomotor, or sometimes of its vasomotor trophic and sensory together, if at least it possesses any trophic fibres separate from the rest. In some instances it may be a direct disease—that is, from blood-changes, from a depressed condition of the central centre, from definite lesions of the nerve itself. But instability of the nerve or its ganglia being present, the phenomenon may be set up from without; and here certain conditions affecting the cervical sympathetic may act in a reflex manner. No morbid state of the cervical sympathetic, moreover, could exist without some vasomotor phenomena, some interference with the vascular tone of the facial vessels, and this would render the tropho-neurosis of the fifth more certain in its course. The position of the sympathetic is important, often almost all important, but in most cases secondary, affording the centripetal irritation in a reflex arc. The objections to any theory are manifold; not only that the absence of post-mortem records leave all theories unproven, but because the atrophic influence of this supposed morbid state of the fifth nerve is not in accordance with experiment.

PHYSIOLOGY — Animal heat

It was a dictum of Claude Bernard's that the vasomotor system is the regulator of heat, of nutrition, and of force. The vasomotor nervous system, having dependent on it the rapidity of the movements of the blood, rules the expenditure of heat. The regulation is really effected by the temperature itself. Heat relaxes the vessels, and accelerates the movements of the heart. Cold contracts the vessels and slows the cardiac rhythm. Animal heat is to a great extent a measure of the chemical changes that are going on within the body. That human beings can live in such opposite external conditions as the arctic and the torrid zones is due to the fact that the heat generated by these chemical changes in the body is being constantly lost by the excretions, by evaporation of perspiration and of respiratory moisture, and by radiation from the blood as it passes through the capillaries of the skin and lungs. The thermometer, therefore, shows not the amount of heat generated, but the amount less the loss by these means.

Heat is caused, (1) by combustion in the tissues, forming excrementitial products, which are eliminated, and recrementitial products, which are reabsorbed into the circulation; (2) by molecular work in secretion; (3) by vasomotor contraction; (4) probably, as Schiff says, by nervous action.

The influence of the vasomotors upon animal heat may be exercised either in the production of heat, or in modifying its loss. The blood acts on this combustion by bringing oxygen, and by exciting

the vitality of the organised substance. Dilatation of vessels favours these points, and this dilatation is somewhat under the influence of the vasomotors; though, when this dilatation depends on excitation of vaso-dilators, it comes into the domain of the cerebro-spinal nerves. The amount of blood in an organ largely depends on the power of the heart, the accelerator nerves of which are sympathetic.

It has been seen that the vasomotors play an important, but by no means the only, part in regulating the secretion of sweat. Still the evaporation of sweat from the skin, and the radiation from the skin, will be the greater the more blood is brought to the skin, and thus the cooling of the blood is increased. Inversely, constriction of the cutaneous vessels checks cooling of the mass of blood. The calibre of the pulmonary vessels may determine the amount of oxygen absorbed. Pulmonary evaporation is greater, the more frequent the respiration. The greater the pulmonary exhalation, the greater the loss of heat, and *vice versâ*. Rapid respiration will induce rapid action of the heart, and therefore loss of heat by the skin.

The maintenance of equal heat in the body under variations of climate and temperature is brought about by the vasomotors, that are excited to constriction of vessels by the reflex action of cold (and so central heat is kept up), or are paretic, and so allow dilatation of vessels (and thus evaporation takes place). Not only so, but the physiological work of the heart is modified, and respiration

Physiology — Animal heat

organic combustion, the work of nutrition, are all more active in a cold medium, and less so in a hot one.

The influence, then, of the sympathetic on animal heat is exercised (1) by the vasomotors of the whole body, regulating the amount of blood for combustion in the tissues; (2) by the vasomotors of the skin partially regulating transpiration from the skin; (3) by what vasomotor fibres there may be in the vagi (if any) that may regulate in any so feeble a way the transpiration from the lungs; (4) by the accelerator nerves of the heart, ruling, not only in part the amount of blood in the tissues, but especially regulating the amount passed through the lungs, and so, indirectly, the quantity of oxygen assimilated by the blood. The passage of the blood through the vessels themselves must produce some heat. But the mere access of more blood to a part will cause no more increase of heat than just what is generated by the passage of blood through the vessels. There must be action for the creation of heat. Bernard called the cerebro-spinal nerves the calorific nerves, because they increase temperature by dilating vessels and so bringing more blood, and by augmenting chemical activity. The first act, that of heat-creation, is the consequence of the chemical phenomena of nutrition. The distribution of the heat created, the diminution of it by constriction of vessels, and by restraining chemical activity, these are the functions of the sympathetic.

There seems to be a moderating centre in the

pons, the nerves from which may be injured or paralysed, and thus, as has been well said, the tissues 'are abandoned to the voracity of combustion.' This is shown by increase of temperature on section of the pons or of the medulla oblongata; but this section causes dilatation of vessels by paralysis of the vascular tone. It has been said, too, with great probability of truth, that such a section only increases temperature if convulsions are excited. Vulpian says that a galvanisation of nerves does not increase the heat of a muscle to which it is distributed, except by contracting it, and so getting more chemical action out of it. Curare stops the heat-production. The chorda tympani only causes heat by increasing the chemical production of more saliva. Vulpian thinks the sympathetic affects temperature simply by bringing more blood to a part, or by crowding it in the interior by contraction of peripheral vessels, or by dilatation of peripheral vessels, by which radiation of heat and evaporation of sweat can be caused. The vasomotors, therefore, are a thermic regulator. Poincaré suggests an interesting theory, that in certain cases the nervous system restores to the economy some heat proceeding from anterior combustion, that it detains in a latent state. As the muscles consume heat in mechanical work, so the nervous system transforms heat into its own work. This amount of heat may not be shown by the thermometer; but from various causes this transformed heat may take on its primary form.

PHYSIOLOGY
Fever

There are three categories as to the mechanism of fever.

1. There is no excess of the production of heat in fever, and the elevation of temperature consists in a work of distribution under the influence of the nervous system.

2. Fever is connected exclusively with an exaggeration of the chemical phenomena of nutrition, and is independent of the nervous system.

3. There is coincidently exaggeration of organic changes, and the indispensable intervention of the nervous system.

In the first category Marey believes the vasomotor system to be mainly involved, and that combustion is not materially altered, though there is some increase in heat-production. In rigor there is spasm of the vessels of the periphery. The blood is thrown internally, and, all cooling conditions being thus removed, heat accumulates. In the second stage there is dilatation of the vessels of the periphery, but the patient is in bed and surrounded by hot things, so the heat is not lost, and even accumulates. He admits, however, that when exhalation of carbonic acid is increased, there must be a certain increase of combustion.

Traube thinks that there is no increase of caloric produced, but loss is diminished. The vascular spasm suffices for him, and he believes that in the second stage of fever the blood only uses up at the periphery the accumulated heat. His theory does not account for the hot stage of ague, for hyper-

pyrexia, for the heat of acute rheumatism with intense perspiration, or the pyrexia of phthisis coincident with perspiration and diarrhœa. The increase of heat, too, that is due to the loss of evaporation has been calculated to be far less than febrile heat.

Hueter asserts that in fever micrococci agglomerate with the white blood corpuscles, and obstruct many cutaneous and pulmonary capillaries, and that so less blood is submitted to cooling from the skin and lungs ; thence accumulation of heat. He agrees also in the importance of the initial vascular spasm, and does not quite deny the increase of combustion.

Most of the authors under the second category hold that the production of heat is increased, that this increase is general, that the heat thrown into the blood from the exaggerated combustion of an inflamed part does not explain the general increase, as the part inflamed is often not so hot as other parts. Senator thinks the elevation of temperature is quite independent of vasomotor modifications, and that innervation only plays an accessory part, such as diminishing the loss of heat by contraction of the vessels of the skin. He saw in rabbits, when the temperature was equally exaggerated after purulent injection, the vessels sometimes dilated, sometimes contracted, sometimes normal. (The diminution of carbonic acid may be apparent in each breath, but, as breathing is hurried, the whole amount of carbonic acid exhaled may not be diminished). Senator believes that the exaggerated combustion is at the expense of the albuminoids, and that the cessation of

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the combustion of the hydrocarbons explains the tendency to fatty degeneration of the tissues in fever.

Hirtz gives the vasomotors the power of diminishing the loss of heat. He finds the weight of the body lessened and all the organic combustions increased. In seeking for the primary cause of the activity of organic combustion, he finds it in infectious fevers in the miasm absorbed by the blood; in zymotic fevers, in the septic poison; in fever connected with local inflammations, in the alteration the blood experiences in passing through the inflamed organ. At the present day the primary cause will be considered in a large number of diseases attended with pyrexia to be the bacilli or microbes peculiar to each disease. The initial rigor, according to Hirtz, is provoked by the sensory nerves of the skin transmitting to the cerebro-spinal axis the sensation of relative cold compared with the heat of the nervous centres. The effect of the spasm renders the difference of temperature greater, and so reflex spasm is augmented. The heart and large vessels, irritated by the reflux of blood, tumultuously throws the blood where there is least obstruction, notably into the cord, causing stiffness of joints, chattering of teeth, even convulsions. In the second stage the blood flows to the surface, and, notwithstanding cooling, continues to be hotter than usual, because excess of combustion goes on. The vascular dilatation slows the circulation; whence suppression of sweat, congestion, gangrene, darkness of lips,

petechiæ, and even hæmorrhages. The heat itself injures the nervous centres; whence headache, insomnia, dreams, subsultus of tendons, adynamia, and ataxy.

Jaccoud says the elevation of temperature precedes the rigors by two or three hours, and in some cases rigor does not occur. If in the second stage there was paralysis of the sympathetic, the heart ought to slow instead of the opposite.

Under the third category, Liebemeister admits exaggeration of combustion, but attributes it to the influence of the nervous system. He says there exists in the brain a special inhibitory centre for chemical combustion, the paralysis of which shows itself in fever. Pyrogenic, miasmatic, or septic substances may paralyse this centre. He thinks the increase of blood-heat by the reflux of blood to the internal organs is a mistake, as fever patients yield from one and a half to two and a half times more heat to a cold bath than healthy people. A mixed formula obtains with many, viz., that pyrogenic substances exercise at first an irritating action on the vasomotor centres, shown by spasm of the peripheral vessels; whence rigors and diminished loss of heat. Thus under the influence of the weakening of those centres, or by continuation of a toxic action, an inverse effect is produced, viz., vasomotor paralysis, whence a greater irrigation of tissues, inducing greater activity of organic combinations, and so greater production of heat.

Claude Bernard would not hold to paralysis of

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the vaso-constrictors, but rather to excitation of vaso-dilators, originating from the cord.

Poincarè thinks that fever consists in a morbid state of the whole nervous system. The sensory nerves take a prominent part, as in Bernard's experiment of running a nail into a horse's foot, with the result of general inflammation if the nerves of the part are unaffected, but with no result if the nerves are cut, and all communication with the cerebro-spinal axis destroyed. Poincarè has seen many local inflammations in paralysed limbs without any general effect.

Hirtz remarks that all febrile substances are poisons to the nervous system.

Many questions suggest themselves in the consideration of fever. Does the temperature depend directly on increased chemical action? or does the morbific cause affect the nervous centres first, being directly or reflexly transmitted to the tissues? The first method doubtless occurs. The second is proven by the equilibrium of temperature under various conditions of climate, &c. Is this by means of vasomotors? Does the nervous system, as, for instance, the isthmus, as Tscherchin thinks, moderate the production of heat? Certainly hyperpyrexia occurs after certain injuries to the cervical cord. The nervous system acts on heat formation, (1) by the influence of vasomotors on vessel; (2) by various other sorts of fibres acting on extra-vascular anatomical elements. A very high temperature in cerebral lesion may almost lead to a diagnosis of pons injury. The writer has more than once met

with a temperature of 110° Fahr. in hæmorrhage of the pons. In another case of extensive fracture of the skull, and in which the temperature rose to 112° Fahr. on the day of death, there was much clotted blood outside the dura mater, especially over the right parietal region. A clot had broken up the cerebral tissue within the left sphenoidal lobe, involving the left optic thalamus, the lower end of the left internal capsule, and almost all the left crus cerebri, pressing on the pons; but the pons and medulla oblongata seemed healthy.

Traumatic fever does not originate from a nervous influence, starting from the lesion and carried to the nervous centres, but from morbid liquid, or liquid containing bacilli, &c., absorbed into the circulation, and so acting upon the nervous centres. Pain plays no part in the alteration of temperature in traumatic fever. The nervous system acts (1) by vasomotors, influencing the calibre of vessels; (2) more directly by the fibres of nerves of animal and of organic life on the anatomical elements, or more generally on the organised substance of the different tissues. And the morbid causes may act directly on this organised substance without implication of the nervous system.

The whole subject has been submitted to careful experiment by Dr. Wood of Philadelphia. Like all other phenomena in the body, various systems take part in the production of fever. It is against all analogy, as it is contrary to direct experiment, to take any but the broadest views of the causation of fever, including exciting cause, cerebro-spinal centres,

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cerebro-spinal nerve, sympathetic nerve, vesseis, and tissues generally. All these are more or less concerned in heat production and in heat dissipation.

It will be convenient to formulate some of Dr. Wood's observations and experiments in numbered propositions, under the belief that this controversial question of fever will be adequately explained by these means.

1. External heat, applied to the body of the normal animal, so as to elevate the temperature, produces derangement of the functions of innervation, circulation, nutrition, and secretion, similar to those seen in natural fever, the intensity of the disturbances being directly proportionate to the rise in temperature.

2. Excess in temperature is the essential symptom of fever.

3. There is a decided increase in the dissipation of heat by the animal after division of the cord.

4. Section of the spinal cord above the origin of the splanchnic nerves is usually followed by an immediate very decided increase in the amount of heat dissipated from the body, and also by a decided lessening of the amount of heat produced.

5. The known influences, which follow the section of the spinal cord, capable of affecting the production of heat, are vasomotor paralysis, lessening of the cardiac force, muscular quiet, diminished respiration, lowering of temperature.

6. The primary cause of the lessened heat production is vasomotor paralysis, which probably acts

directly and also indirectly by causing an excess in loss of heat, and such a lowering of internal temperature as to check chemical reactions in the body. A general dilatation of all the vessels produces a sluggishness in the movement of the blood in all parts of the body.

7. The fall of temperature of the body, usually following spinal section, has a very important effect in lessening the amount of heat produced, i.e., the chemical movements of the body.

8. When the effect of the cooling of the body is done away with, section of the cord is sometimes followed by a decreased, sometimes by an increased, heat production; and the latter occurs especially in robust animals.

9. The dominant vasomotor centre is in a region, according to Owsjansikow, the upper boundary of which is one or two millimetres below the corpora quadrigemina, whilst the lower boundary is four to five millimetres above the point of the calamus scriptorius. Dr. Wood found himself by experiment that the chief governing vasomotor centre is placed in the lower portion of the floor of the fourth ventricle, near the point of the calamus.

10. Some injuries of the medulla below the calamus produce results similar to, but greater than, those caused by section of the cord, viz., fall of the animal temperature, and of the arterial pressure; and confirm the belief that the fall of temperature is due to vasomotor paralysis.

11. Section of the medulla oblongata, at the line

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Fever

of its junction with the pons, in the dog, usually leads to an elevation of animal temperature, if the medullary vasomotor centres are not in any way compromised.

12. Wounds of the medulla oblongata so situated as to paralyse the vasomotor centres cause decreased heat production, with (certainly sometimes and probably always) primarily increased heat dissipation.

13. Section of the medulla oblongata at its junction with the pons is followed by increased heat dissipation and increased heat production; the increased dissipation usually not keeping pace with the increased production, so that the bodily temperature rises.

14. The rise of bodily temperature and of heat production, following separation of the pons from the medulla, is paralytic, and due to the removal of some active force.

15. Tscherchin considered that there is in the brain, somewhere above the pons, a nerve centre, whose function it is directly to inhibit or repress the chemical movements of the body—i.e., the production of animal heat. This has been called the inhibitory heat centre.

16. At present we can only conclude that the rise of bodily temperature, following separation of the pons from the cord, is due either to paralysis of the inhibitory heat centre, or of a muscular vasomotor centre.

17. Galvanisation of a sensitive nerve produces a fall of the arterial pressure by acting upon some

nervous centre situated either in or above the pons.

18. Eulenberg and Landois, after destroying in dogs a region of the cortex cerebri near the sulcus cruciatus, found that the temperature of the opposite extremities rose. This region embraces a gyrus corresponding to the gyrus postfrontalis.

19. Destruction of the first cerebral convolution in the dog is followed at once by a very decided increase of heat production, whilst after irritation of the same nervous tract there is a decided decrease of heat production.

20. The probabilities are that the calorific centres are situated in the pons, and that the power of the first convolution depends upon habitual co-action.

21. After section of the vagi, in the curarised animal, mild irritation of the Hitzig region in both sides of the brain does not abate the blood pressure.

22. Probably the centre which directly controls the production of animal heat is not situated in the cortex. Its existence and position are unknown, but it is probably situated in the pons or higher up.

23. In the animal economy there are two distinct general sources of heat—1, the destruction, which probably occurs in the blood, of the excess of crude food material; 2, nutritive changes in tissue, including all changes in the blood itself at the expense of its permanent constituents.

24. It has not been proved, but it is most probable,

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that the heat centre affects solely the latter source of animal heat.

25. In the pyæmic fever of dogs, the heat production is mostly in excess of the heat production of fasting dogs, but less than that which can be produced by high feeding. Usually the production of animal heat rises in the febrile state with the temperature and with the stage of the fever; but sometimes the heat production becomes very excessive, although the temperature of the body remains near the normal limit.

26. Fever is a complex nutritive disturbance in which there is an excessive production of such portion of the bodily heat as is derived from chemical movement in the accumulated material of the organism, the surplus being sometimes less, sometimes more than the loss of heat production resulting from abstinence from food. The degree of bodily temperature in fever depends in greater or less measure upon a disturbance in the natural play between the functions of heat production and heat dissipation, and is not an accurate measure of the intensity of the increased chemical movements of the tissues.

27. Fever due to the introduction of a poison into the blood appears at first sight to be probably produced by an action of the poison upon the general protoplasm. But in malarial fever, the chill, the fever, and the sweating own a neurotic origin.

28. Irritative fever, if it exists, is produced by an action upon the nervous system. Fever occurring in cases of blood-poisoning is often, and probably

always, the result of a direct or indirect action of the poison upon the central nervous system, and hence is a neurosis.

29. In health there is in man a fixed mean and a normal variation of temperature having a regular rhythm: this variation is beyond the control of all disturbing causes, which do not force the organism beyond the condition of health.

The maintenance of the normal temperature and its rhythm is dependent upon the nervous system, which within certain limits controls both the production and the dissipation of animal heat. So far as our present knowledge goes, the chief factors in controlling heat dissipation are the vasomotor nerves, including in man such nerves as control sweat secretion; these nerves being able, by contracting the capillaries of the surface of the body, and by drying the secretions of the skin, to reduce the loss of heat to a minimum, and by a reverse action to increase it to a maximum.

The only nerve-centre proved to exist, capable of influencing the heat production without affecting the general circulation, is situated in the pons or above it; and whilst it may be a muscular vasomotor centre, it is more probably an inhibitory heat centre. Of whichever nature it may be, it must act through subordinate centres situated in the spinal cord.

30. The causation of fever. Fever is a state in which a depressing poison or a depressing peripheral irritation acts upon the nervous system, which regulates the production and dissipation of animal heat. Owing to

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its depressed benumbed state, the inhibitory centre does not exert its normal influence upon the system, and consequently tissue change goes on at a rate which results in the production of more heat than normal, and an abnormal destruction and elimination of the materials of the tissue. At the same time the vasomotors and other heat-dissipation centres are so benumbed that they are not called into action by their normal stimulus (so elevation of the general body temperature), and do not provide for the throwing off the animal heat until it becomes so excessive as to call into action, by its excessive stimulation, even their depressed forces. In some cases there is added a complete paralysis of the vasomotor centres presiding over heat production and dissipation.

Vasomotor congestion is the result of direct or reflex vasomotor paralysis, except in those cases in which the presence and excitation of vaso-dilators can be proved. Vasomotor congestion is direct in section of the cervical sympathetic (blushing being the effect of such section, or of emotions imitating such a paralytic action, or of transitory paralysis caused by such substances as nitrite of amyl); in certain lesions also of cord where the circulation of the heart is interfered with; in certain central lesions (hæmorrhage, softening, &c.), with increase of temperature in the paralysed limbs. Gubler thinks that the 'tache méningitique' of a paralysed limb is due to reflex excitation of vaso-dilator fibres; but

there can be little doubt that it is really due to suspension of tonic activity of a vasomotor centre, and so to paralysis of vaso-constrictors.

Vasomotor congestion is reflex when it is set up by a sensory irritation, as when the vaso-dilator fibres of the chorda tympani are set in action by irritation of the lingual branch of the fifth nerve. So also in faradising the peripheral end of the glossopharyngeal nerve, after section. The vaso-dilator fibres in this arc do not belong to the facial nerve, but probably to cells in the course of the glossopharyngeal itself. But vaso-dilator fibres are not indispensable to the production of vaso-dilator action from peripheral excitation. Reflex congestion is generally due to suspension of tonic activity of the vasomotor centres, medullary or ganglionic, under the influence of irritation at the periphery of the centripetal nerves, transmitted to these centres by certain fibres of these nerves. M. Notta observed conjunctival redness in 34 out of 61 cases of neuralgia of the fifth nerve. This congestion may extend over half the face, and even inside the mouth. It may be explained by the hypothesis of vaso-dilators, but, as this cannot be universally proved, it is enough to say that the transmission of the excitation along the sensory centripetal nerves to the vasomotor centres of the region to which the nerves are distributed may suspend the tonic activity of these centres, so as to cause a paralysis of the vessels of the corresponding regions. A similar reflex congestion may occasionally be seen in connection with neuralgia of other nerves.

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The congestion of the second stage of ague owns a somewhat different causation. It is due to a dilatation of the vessels of the integument that depends on a certain degree of vasomotor paralysis, succeeding the stage of excitation of the cutaneous nerves in the first stage. This is not owing merely to fatigue of the nerves, as the dilatation is in no proportion to the duration or intensity of the cold stage, but to a special modification of the vasomotor apparatus, produced directly or indirectly by the morbific agent. Fatigue of nerves may in many cases add to this. Vulpian attempts to explain the specific congestion of the exanthemata in a similar way, as a dilatation of the subepidermic vessels; and he says that in measles the eruption seems to show that the dilating cause acts on sets of neuro-vascular territories, distinct one from another; and that congestion of the internal viscera, the lungs for instance, in typhoid fever, is due to a functional modification of the vasomotors of the lungs, similar to that of the vasomotors of the skin; whilst, although the fulness of the spleen seems to depend on a proliferation of the cellular elements in that organ, yet its rapid variation in size in ague and in typhoid fever must be induced by successive weakness and activity of the vasomotor apparatus of this organ. There is a difficulty in accepting Vulpian's theory as to pulmonary congestion in typhoid, from the fact that vasomotors of the pulmonary vessels are either absent, or so sparsely distributed (and that only in connection with the vagus) that such an influence

could scarcely be attributed to them. Besides, these pulmonary congestions are passive, and are usually the result of gravitation of blood from position, of the condition of the blood itself, and of deficient cardiac power.

The opposite relation between the lungs and the skin is, however, sometimes seen. The congestion of the cheeks in pneumonia, from reflex dilatation of the vessels of the cheek, is not uncommon, unilateral pneumonia being often associated with unilateral congestion of the cheek; and the occasional phenomenon may be seen, in inflammatory disease of the lungs, of the arm of the affected side being hotter than the other, probably from reflex irritation carried to the bulbo-spinal axis and reflected along the brachial plexus and the vasomotors included in it. Of the same order is the congestion of internal organs, notably of the intestinal mucous membrane, following burns on the surface of the body. Ulceration of the duodenum is not unfrequently met with as a sequence of such burns; and the mechanism is centripetal irritation from the skin to the spinal cord, reflected down to the semilunar ganglia and the subordinate vasomotor nerves to the intestine.

An interesting form of congestion is seen from the effects of lightning on the human body. The markings, so like fern-fronds or branches of trees, are caused by the direct action of the electric fluid in paralysing the nervous system, by causing congestion and redness in the capillary vessels.

The influence of emotion on the sympathetic

PHYSIOLOGY — Congestion

system will be referred to hereafter at greater length. It suffices to say here that some physiologists place the centre of emotion in the annular protuberance. It is at least more probable that emotive centres may be found in all parts of the cortex, forming one of the layers of cells seen on horizontal section of the cortex. Wherever placed, the emotive centre, when excited, reacts in disturbance of the medulla oblongata and cord, and this induces contraction of certain muscles of animal life, or a sudden condition of muscular feebleness, or disturbance in the cardiac movements, or alteration of intestinal secretion, or modification of vascular tone in various parts of the body, particularly of the face and head. These modifications of vascular tone consist sometimes in exaggerations, sometimes in a cessation more or less complete of the tone of the vessels, and consequently an anæmia (pallor) is produced, or a congestion (redness) of the tissues, in which these modifications are manifested. The congestion is due to a transitory, but more or less complete, relaxation of the vasomotor centres, the permanent activity of which maintains the tone of the vessels of the face. These centres are the cervical and upper thoracic ganglia of the great sympathetic, and the intra-medullary and intra-bulbar centres of origin of the vasomotors of the face.

This emotional congestion is not limited to the face and neck. In certain cases not only is the skin of the chest, down to the level of the mammæ, the seat of a similar congestion, but even down to the

lowest point of the sternum. The writer has seen in a female patient, of an exceedingly emotional type, the face, the neck, the arms, the back, the chest, the upper part of the abdomen, assume a deep purplish-red hue, from causes so slight, that it seemed difficult to believe any strong emotion could have been excited by them; and the patient asserts that the same extreme congestion affects every part of the skin of the trunk and legs. Vulpian mentions the case of a man of fifty, nervous and subject to neuralgia, in whom this emotional congestion occupied the position of the anterior upper portions of the thorax, the shoulders, the anterior region of the abdomen, and the upper half of the thighs. In the case of the lady above mentioned, the skin of the neck and trunk was not suddenly and universally affected. The deep colour appears in islets here and there, round or irregularly oval. These islets seem to enlarge their borders, and eventually become confluent. A similar gradual congestion has been noticed by other observers. Dr. John Ogle sends the writer the following note: 'A friend of mine says that in Paris he had a young man sitting to him as a model, naked or nearly so. Suddenly some political news of great importance was announced, and the man became quickly excited and enraged, and my friend noticed not only that he became extremely red in the face and neck, but the entire surface of the body became bright red and lobster-like.'

Determination of blood is a frequent precursor of

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inflammation, but it is not part of it. Reflex congestion produced by stimulation of sensory nerves is not the same as inflammation.

The physiological history of inflammation is briefly this :

1. Some source of irritation—cold, a blow, a burn, a septic focus.

2. The centripetal nerves, whether sensory or not, which are within reach of this irritation, are excited more or less violently.

3. These nerves transmit to the vasomotor centres of the region the excitation which they have undergone.

4. The tonic activity of these centres is disturbed, and suspended, more or less completely.

5. Hence follows cessation or diminution of the tone of the vessels that are subordinate to these centres.

6. Consequently, more or less considerable dilatation of these vessels occur.

7. But this vasomotor disturbance can only be considered as favouring the development of inflammation. It is only secondary in importance, and does not suffice of itself to make up the phenomena that are called 'inflammation.' It places the vessels in a condition for easily and necessarily receiving more blood ; it offers facilities for the emigration of leucocytes ; but the initial phenomena of inflammation consist in the disturbance of the intimate nutrition produced in the organised living tissue. The vital condition of the tissues having been gradually altered

by the previous state of its nutrition, and the peripheral resistance in the capillaries having been induced, the part is placed in a condition of vulnerability, and is ready at any moment to respond to morbid impressions. They may be reflex, as the impress of cold, or direct, as from the presence of morbid germs in the blood; and the vasomotor action on the vessels, which without the previous alteration of the cell-nutrition would stop short at non-inflammatory congestion, is of enormous importance in determining the various stages and symptoms of the progress of inflammation, although independent, and unconnected directly with the initial phenomenon.

Some of the capillaries are blocked early by thrombi; others, that are permeable, are dilated; and the course of the blood, instead of being continuous, as in the normal state, becomes jerky, as in the arteries; and thence is caused, partly at least, the sensation of pulsation experienced in an inflamed region under certain circumstances.

As to the mechanism by which the vasomotor centres of the bulbo-spinal axis, or at least of the vasomotor ganglia, induce, under the influence of the irritation transmitted to them, a dilatation of vessels in the inflamed region, it probably consists in a suspension of the tonic activity of these centres. Hence the advantage of cold as a therapeutic agent in constricting the vessels.

This theory of inflammation is practically a mere statement, in other words, of the dictum of Vulpian, that in pneumonia, besides the mechanism of the cold

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impressions influencing in a reflex way the nutrition of the lungs through the nervous centres, there is need to admit, first, a general predisposition to inflammation, and secondly, a local predisposition, which renders the respiratory organs more sensitive to the reflex action of cold than other parts of the body. At the present day this local predisposition may perhaps be considered to depend on the presence of the pneumonic microbe.

But although vasomotor paresis has little or nothing to do directly with the initial phenomena of inflammation, there are numerous instances of indirect action. Why is pulmonary congestion, or a similar condition in any other portion of the system, looked upon with grave suspicion? Is it not that a region so affected is particularly liable to take on active mischief?—i.e. a part that for a time has been affected only by means of partial paralysis of the vasomotors can very readily be found to be the seat of inflammation.

It was this prominent fact that probably misled observers as to the rôle of the vasomotors in inflammation. Congestion so often passes into inflammation that the former was supposed to be the cause of the latter. But the explanation of the connection is that given above. A congested part became gradually of necessity a part in which nutrition is badly performed. The affected spot, becoming less and less perfectly nourished, is ipso facto more or less vulnerable to influences external to itself; in other words, it is predisposed to inflammation. The external influence

arrives; modifies directly or reflexly still farther the nutrition of a part already vulnerable, already possessing unstable cells; and the predisposition, the external influence, the modification of nutrition by the exciting cause, and the vasomotor paresis, make up the necessary factors in the causation of inflammation.

Dr. Handfield Jones goes much farther than this in attributing inflammation to paralysis of the vasal nerves. The grounds for his belief are:

1. That in a cat purulent ophthalmia may be produced by division of the sympathetic nerve in the neck.

2. The inflammation of the intestines and cerebral membranes, in pernicious malarial fever, can only be ascribed to extreme local determination of blood, the result of sympathetic paralysis.

3. The occurrence of inflammatory disorganisation of the lung in cases of primary cancerous tumour at its root, destroying the pulmonary nerves.

4. Severe eczema or pemphigus may be cured by arsenic, a nerve tonic.

5. The occasional cure of ophthalmia by quinine.

6. The fact that severe neuralgia is sometimes attended with marked erysipelatoid inflammation of the parts affected. This seems fairly to be explained by assuming that, in common neuralgia, the sensory nerves alone are deranged, whilst in the case here considered the vasomotors are involved also.

In spite, however, of these views of Dr. Handfield Jones, it will be generally admitted that vasomotor

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paralysis does not act as causing inflammation; it is only accessory to it.

The slow, predisposing influence of congestion is markedly increased if it results in œdema. The vasomotor nerves are implicated in the causation of œdema in more ways than one. Paralysis of vasomotors dilates the arteries and fills them with blood; the capillaries become abnormally distended, profusely dilated; and in the case of any hindrance to the venous circulation œdema results. Or œdema may be reflex, following a dilatation of vessels due to reflex sensory irritation. The capillaries may remain patent, or be thrombotic; and thus traumatic irritation of centripetal nervous fibres may be seen, suspension of tonic activity of certain vasomotor centres, paralytic dilatation of the muscular tissue of the vessels, diminished vis a tergo in the veins, and so œdema. Or again, reflex paralysis of vessels, with enfeebled vis a tergo, gives rise to a relative intravenous stasis, an increased pressure on the capillaries, and issue of liquid from the vessels. From whatever cause, the waterlogging of a tissue by œdema must necessarily interfere with its nutrition.

That the cause of œdema, however, is vital rather than mechanical, is shown by the experiments of Ranvier. 'He found that, by ligating the femoral vein, the circulation was not enough interfered with to give rise to any transudation; but after section of the nerves supplying the vessels, the flow became so much more retarded, that œdema soon appeared.'

It scarcely needs saying that in some instances œdema is due mainly to the morbid condition of the blood itself; but even then the vasomotors play some part, as some kinds of morbid blood will not act by way of centripetal irritation on the ganglia of the vessels, and thus the first factor in producing vascular tone is lost. The explanation of œdema, for instance, in chronic nephritis is far from being simple. It depends on the condition of the blood itself, on the state of the vascular walls, on the influence of the cardiac power, and, to some extent, on the amount of renal secretion; but the influence of the blood, or rather its diminished influence on the ganglia of the vessels, has much to say to the production of the œdema. Very markedly, too, does a similar explanation obtain in the cerebral œdema of chronic alcoholism. Even in the common forms of œdema of the lower limbs, which may spread over the whole body, associated with mitral disease and dilatation of the heart, the diminished vis a tergo, though the chief, is not the only factor. The state of the blood itself, its influence over the vascular ganglia being enfeebled, plays no mean part.

Lesions of certain portions of the cerebro-spinal system may be followed by hæmorrhage in various parts of the body. Thus lesion of some nervous centres, especially of the optic thalami and crura cerebri, may cause ecchymosis in the mucous membrane of the stomach. Lesions of the isthmus may cause hæmorrhage in the intestine. Lesion of the

Physiology — Hæmorrhage

dorsal region of the cord may be followed by hæmorrhage in the suprarenal capsules; lesion of the pons by hæmorrhage in the lungs, and not there only, but in the liver, the kidneys, the endocardium, pericardium, myocardium, or also of the pleura. And these hæmorrhages occur in the organs on the opposite side of the body to the cerebral lesion.

Hæmorrhage of the mucous membrane of the stomach has been seen to follow long-continued irritation of the sciatic nerve, lesion of the auditory labyrinth, or transverse injury of the semicircular canals, or lesion of the corpora quadrigemina. The influence of asphyxia and the poison of strychnia may often cause similar hæmorrhages, or an ecchymotic condition of the lung just beneath the pleura.

It is doubtful whether, in these cases, the arteries are by any means always affected. The rationale is often a vasoconstriction of veins and rupture of capillaries behind the constrictive obstruction. But in some instances the mode of causation is vasomotor paralysis, causing first an intense congestion, and secondly, a rupture of vessels, if the exciting cause of this congestion persist.

It seems also extremely doubtful whether the epistaxis or the uterine hæmorrhage that are met with in the eruptive fevers—in typhoid, for instance—are connected with modification of the vasomotor functions in any but the very slightest degree. The condition of the blood, and the definite lesions of the vascular walls, will account for the phenomenon, almost without any reference to the vasomotors.

But, as has been already stated, hæmatidrosis may occur with deep disturbance of certain parts of the vasomotor apparatus, especially of the vessels of the sweat-glands, in the subjects of hysteria and hystero-epilepsy. The symptom is due to a momentary paralysis of the vasomotor centres which rule the tone of the vessels of the sweat-glands, often caused by irritation of the sensory fibres of the region of the skin affected with the hæmatidrosis. It sometimes accompanies excessive neuralgic pains, or it may follow them pretty quickly, the neuralgia attacking regions at a little distance.

Irregular or suppressed menstruation may give rise to vicarious hæmorrhage in various parts of the body, hæmatemesis, intestinal hæmorrhage, hæmoptysis, hæmaturia, epistaxis, and less frequently tears of blood, hæmorrhage from the mammary glands, from the alveoli of the teeth, or subcutaneous. All these follow the catamenial suppression. The mode of action lies in the fact that the suppression of the catamenial flow causes a state of suffering in the vasomotors of the nervous centres which preside over the function; this is transmitted to other vasomotor centres, or to all, causing more or less general congestion, with the symptoms consequent on them, viz. suffusion of face, tinnitus, headache, giddiness, &c. The hæmorrhage from a fibrous tumour of the uterus is also reflex. Irritation of the centripetal nerve-fibres of the uterus, due to the development of a fibrous body in its walls, is carried up to the centres that rule the tone of the uterine vessels. The tonic activity of these centres is thus enfeebled or

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suspended; the vascular tone is diminished or abolished in the uterine walls, and congestion and hæmorrhage may result.

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CHAPTER III.

THE PATHOLOGY OF THE SYMPATHETIC. GENERAL PATHOLOGY.

PATHOLOGY

In speaking of the general pathology of the sympathetic system it is necessary to say—1st, that a multitude of symptoms, evidently dependent on a morbid condition of the sympathetic, are met with without any such diseased state of the nerves or ganglia being recognised by minute examination. 2nd. That many morbid symptoms can be explained by a coarse lesion of nerves or ganglia, such as the pressure of a tumour, &c. 3. That very many nerves and ganglia, the latter especially, are found pigmental, sclerosed, with great increase of interstitial connective tissue, with proliferation of cell elements, or in various stages and conditions of degeneration, and yet the patient during life has manifested no phenomena that could be attributed to such lesion: the explanation probably being in the amount of ganglion left healthy. 4. That many of the lesions of the sympathetic found after death are due to the ganglia being secondarily involved in the original disease, and having nothing to do with its causation, although it is likely enough that some of the symptoms witnessed during life may

have been due to this secondary implication of the ganglia. PATHOLOGY

In a word, the sympathetic system may be said to possess a very special pathology, but by no means in all cases a recognised pathological anatomy. The important part it plays in so many morbid states is owing to the fact of its being so constantly affected by reflex irritations.

Anything useful will come under the domain of special pathology; but it may be well to throw together some of the recorded observations of former times. Thus, both in ataxy and in tetanus, redness of the semilunar ganglia has been observed. Inflammation of the semilunar ganglia has been associated with headache, hypochondriasis, vomiting, and death from marasmus; inflammation of the left portion of the solar plexus with pertussis, spasmodic vomiting and convulsions; inflammation of the ninth and tenth thoracic ganglia after retrocession of an exanthematous disease with opisthotonus; vascularity of the sympathetic nerves in the chest, and of the semilunar ganglia with tetanus; great increase in the size of the lower cervical ganglia with cretinism; great increase in the size of the ganglia with idiocy; enlargement of all the abdominal sympathetic and the splanchnic nerves with diabetes; increase in size of the semilunar ganglia with a case of tuberculous suprarenal capsule; increase of size of the same ganglia with cancer of the stomach. One of the semilunar ganglia was of the size of a filbert, and cartilaginous in a case of madness. The abdominal

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ganglia have been found large, lobulated, yellowish, and of firm consistence in chronic peritonitis. Cholera has been sometimes associated with inflammation of the solar plexus and of the semilunar ganglia.

The occasional mingling of cause and effect in some of these observations is due to many of them having been made in an early stage of the history of pathology. It will be needful to pass some of them in review in speaking more individually of special features of the sympathetic system.

According to Pio Foà, lesions are most commonly seen in the cervical and the abdominal ganglia. These lesions are simple and fibrous atrophy, hyperæmia, sclerosis, fatty and pigmentary infiltration, amyloid degeneration, accumulation of colourless blood-corpuscles, and the presence of micrococci in the blood-vessels of the ganglia. These changes are well marked in syphilis, leukæmia, a high degree of cachexia, pellagra, tuberculosis, cardiac disorders, and infectious diseases. In tuberculosis the vessels of the ganglia were often dilated and over-filled with blood. When tubercular disease of the abdominal organs predominated, the ganglia were anæmic and atrophied ; and where the course of the disease was very acute, the blood-vessels were crowded with white corpuscles ; and after inflammation of the lungs, and in cases of heart-disease, the ganglia were overloaded with blood and strongly pigmented. In leukæmic conditions (lymphatic leukæmia) the white corpuscles were present in abnormal quantity. Syphilis was accompanied with a remarkably copious

development of connective tissue and by pigmentation of the cells. In profoundly cachectic states there was amyloid degeneration of the vessels of the ganglia; in pellagra, the vessels were much dilated, and the cells were full of pigment and fat; and in infectious diseases there was an abundance of white corpuscles in the stroma.

Fournier thinks that the sympathetic system is affected in secondary syphilis, as shown by variations in temperature, by sweating, and even by epileptic seizures; but his views seem founded more upon symptoms than on pathological anatomy.

The most usual lesions are pigmentation, colloid degeneration, with proliferation of endothelial cells, and secondary fatty metamorphosis, interstitial hyperplasia leading to atrophy and sclerosis of nerve elements. Such lesions are more than enough to modify vasomotor functions, or to lead, according to their seat, to very various morbid phenomena.

Morselli found fatty degeneration and atrophy of ganglion cells with thrombotic obliteration of the vessels of the cervical ganglia; whilst in a case of unilateral sweating Ebstein has seen very dilated and varicose blood-vessels in the ganglia of the affected side. Colomiatti has seen lipoma of the sympathetic, and a tuberculous nodule in the last left dorsal ganglion but one of the great sympathetic, and a similar nodule on the communicating branch between this ganglion and the one above it. The same observer has seen cancer of the semilunar ganglia, compressing and atrophying the cells, entering into

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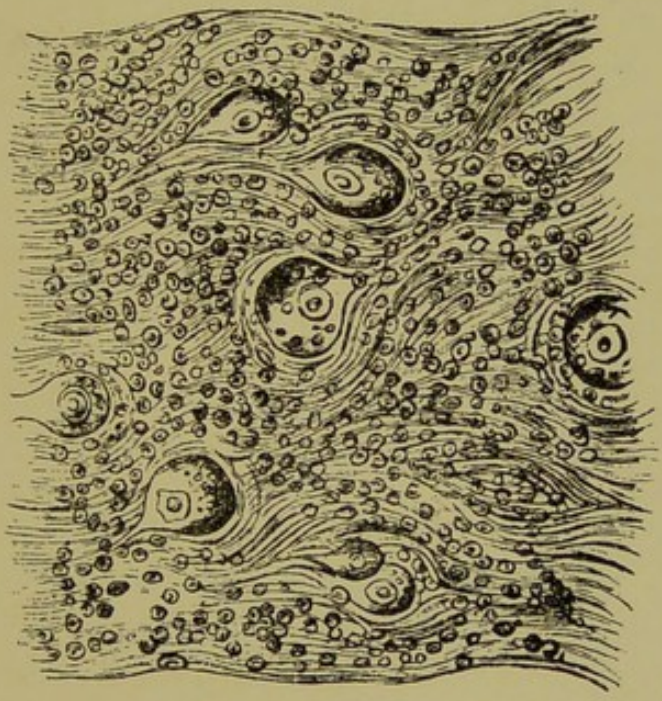
the nerve-trunks and the substance of the neurilemma.

The semilunar ganglia may be enlarged and diseased, following a blow upon the abdomen, and may give rise to lesion of the liver and kidney.

In sunstroke, hæmorrhage has been found in the upper cervical ganglion. Hilton saw attenuation of the right side of the heart associated with shrivelled ganglia of the same side of this organ. Giovanni considers fatty degeneration rare, but thinks that the sympathetic resists most of the diseases affecting the whole system; and that lymphatic infiltration of its ganglia is the expression of the peculiar influence which it suffers. This condition, he believes, he has found in pleuro-pneumonia, cardiac disease, tuberculosis, aneurism of the aorta, diffused tumours, acute and chronic nephritis, chronic enteritis, cirrhosis of the liver, suppurating ovarian cyst, typhoid fever, cancer, puerperal peritonitis, puerperal fever, pyæmia, pellagra, syphilis, scurvy, leukæmia splenica, tabes mesenterica, tabes dorsalis, erysipelas, epidemic cerebro-spinal meningitis, hydrophobia, exophthalmos, angina pectoris, and diphtheria. The very numerous morbid changes associated with this condition of the sympathetic is nearly proof positive that this nerve has little or nothing to do with their causation.

An atrophic shrinking of the nerve-cells proper, and a calcareous mass occupying the position of the inferior cervical ganglion, were conditions found by Dr. Shingleton Smith in a case of exophthalmos. A late observer has found very definite lesion of the

PLATE VI.



Extreme Lymphatic Infiltration. (After Giovanni.)

sympathetic in phthisis, dilatation of vessels in the ganglia, proliferation of the connective tissue, hyperplasia of the epithelial covering of the capsules of nerve-cells, atrophy and pigmentation of cells. In chronic cases there was found an enormous development of dilated blood-vessels; the external membrane of the ganglia (with its internal processes), the external capsule of the ganglionic nerve-cells, the neurilemma of the nerve-fibres, and the tunica adventitia of the blood-vessels, were all greatly thickened. This was especially the case in the inferior cervical ganglion. The lesions of the sympathetic on the healthy side were but slight. The sympathetic lesions may be considered secondary. But the ganglionic affection, though caused by the previous disease, may itself induce some of the morbid phenomena, as the hectic of the cheek and the facial perspiration, even if it does not determine certain trophic changes in the bronchial mucous membrane. The writer has frequently found dilated vessels and increased connective tissue in the inferior cervical ganglion in phthisis, and extreme cell proliferation in the inferior cervical ganglion of the same side in scirrhus of the breast.

The investigations of Dr. Saundby seem to prove that the relationship between the renal lesions in Bright's disease, and the changes observed in the ganglia and surrounding connective tissue, must be regarded as secondary rather than primary.

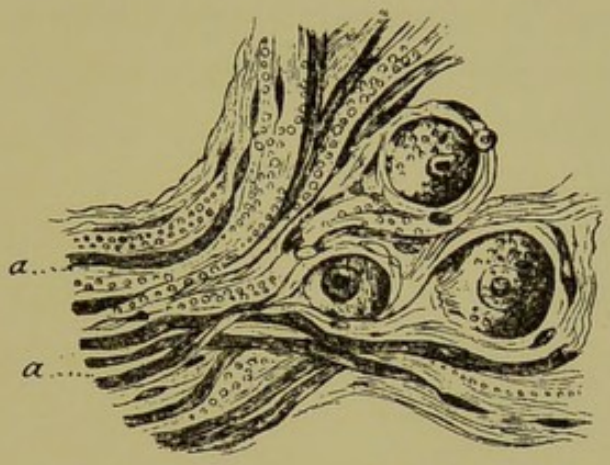
It is easy to understand that such lesions, secondary in themselves to the diseases with which they

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are connected, yet impress upon the course of the disease certain special phenomena. Flushing, sweating, tinnitus, headache, faintness, palpitation, constipation, diarrhœa, vascular congestion of the intestines, diuresis, &c., are all examples of this.

These facts are all more than mere matters of pathological interest. Given a recognisable lesion of a sympathetic ganglion or nerve, certain phenomena are found following this as a sure consequence. It is nature's own experiment to teach the physiology of the sympathetic. But, on the other hand, given these same phenomena without a coarse lesion of the sympathetic nerve or ganglion, is it not justifiable to say that they depend upon a morbid condition of these structure, even though such a condition cannot be recognised by the usual means of investigation? This is what happens frequently. The common distinction between organic and functional disease of the sympathetic is only an unscientific method of expressing this thought. A ganglion, or a series of ganglia, apparently healthy, may be changed in some occult way by the sun's rays, by the circulation of blood altered from its normal condition, by what is called 'irritation' carried to it from disease in a distant organ, or by emotion. It cannot be doubted that these influences change in some way the equipoise of the ganglion; for as their result are seen phenomena precisely corresponding to the effects of coarse experiments upon the sympathetic in animals, and of easily recognised lesion upon these organs in man. The starting-point of the

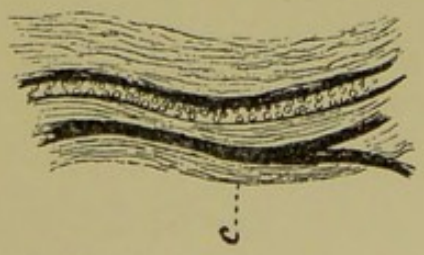
PLATE VII.



a. Fatty Degeneration of Fibres from a Sympathetic Ganglion.



b. An individual Fibre so degenerated.



c. Extreme Fatty Degeneration. (After Giovanni.)

irritation is seen, the channels by which the irritation is conveyed, the consequences of the irritative action beyond the ganglion; but the absolute condition of the ganglion itself, in so far as it differs from its state in health, is incapable of being, in all cases, demonstrated. The irritation may arise from some portion of the same nervous system or from any part of the cerebro-spinal. It may be reflected only on its own fibres, or upon cerebral or spinal nerves. The exodic response from the ganglia may be carried back solely to the point of origin, or in very various directions to many organs, and through many and various channels. The reflected effects may be sensory, motor, or vasomotor, or all together; and yet the ganglionic centre of this reflex arc may seem, even microscopically, to be unchanged.

That change of some kind ensues cannot be doubted. All that can be said is that it is a change so minute as so far to baffle the usual means of research, or so transient as to pass away before the possibility of post-mortem examination. It is almost an axiom, that irritations which induce sympathetic phenomena are generally reflex rather than direct.

Before the diseases consequent on lesion of the sympathetic are entered upon in order, it will be well to give a short résumé of the effects on the sympathetic by lesions of various portions of the cerebro-spinal system. Some of these points have already been referred to:

1. Lesion of the isthmus may be followed by

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Effects on the sympathetic of cerebro-spinal lesions

emphysema and by spots of pulmonary apoplexy. The influence on the vasomotors in the lungs, slight as it is, is by way of the upper thoracic ganglia, and so through the pulmonary plexuses.

2. Lesion of the medulla oblongata, of the pons, the crura cerebri, the vermis cerebelli, middle crura cerebelli, and optic thalami may be followed by glycosuria.

3. The same phenomenon is found with lesion of the anterior columns of the spinal cord in their whole extent, and also of the posterior columns, probably by irritation carried thence to the medulla oblongata. Schiff says, too, after section of the sciatic nerve; and many observations confirmatory of this view have been made.

4. Lesion of the base of the brain, especially of the crura cerebri and optic thalami, may cause softening of the stomach.

5. Lesion of certain brain-centres acts on the intestine and on the stomach, especially injury to the cerebellum, pons, corpora quadrigemina, crura cerebri and cerebelli.

6. Lesions of the cerebellum (probably from pressure on the medulla oblongata), and lesion of the dorsal and cervical cord, especially cervical lesion from hanging, cause erection.

7. Lesions of the medulla oblongata, about two millimetres behind the corpora quadrigemina, and of the anterior superior part of the cord, cause enfeeblement or even destruction of vascular tone. All lesions of cord may enfeeble the vascular tone of the

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parts in relation, by their vasomotor nerves, with the region of the nerves below the lesion. Schiff found that the hemisection of the cord, at least at the last cervical vertebra, produces a greater heating of the head than does a transverse section of the medulla oblongata.

8. Section of the cervical cord causes increase in the temperature of the limbs below. Further hemisection of the dorsal region increases the height of temperature and of vascular dilatation. By lesion of the cervical cord paralysis of the facial vasomotor in man is more frequently produced than irritation.

9. Lesions of the isthmus, the optic thalami, and the corpora striata may modify the temperature in the limbs of the paralysed side.

10. Excitation of the cord, as in myelitis, may set up vasodilator actions.

11. Section of cord, especially in the dorsal regions, causes elevation of temperature in the limbs. Hemisection causes elevation of temperature in corresponding lower limbs, and often cooling of the opposite limb. The first phenomenon is explained by paralysis of vessels and increased circulation. The cooling of the opposite limb is produced either by reflex contraction of vessels, or because the paralysed limb contains an undue amount of blood. This cooling of the opposite limb is far from being constant.

12. Hemisection of the lower third of the dorsal region of the spinal cord produces vascular dilatation

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in the lower limb of the corresponding side, and so increase of temperature.

13. Hemisection at the middle of the dorsal region, or at the anterior superior portion of it, produces greater dilatation of vessels in the lower limb than lesion at the lower dorsal region, because the vasomotors of the lower limb have a multiple origin from abdominal plexuses, and in union with the sciatic nerve, and are not therefore all cut in lesion of the lower dorsal cord.

14. Section between the seventh and eighth dorsal vertebræ, besides causing heat in the lower limb, produces vascular dilatation; and this in the upper limb also, as fibres come off from this spot (from third to seventh dorsal) to go to the upper limb.

15. Section a little higher, towards the first and second dorsal vertebræ, causes vascular dilatation in all parts of the corresponding side of the body, and even of the head.

16. Section in the cervical region produces the same effect more clearly.

17. Section of the anterior portion of the pons has no effect on the vessels of the limbs, head, or trunk.

18. Lesion of cerebellum has no vasomotor influence except, perhaps, glycosuria.

19. Lesion of the cerebral hemisphere, or of the corpora striata, or of the optic thalami, may have a paralytic influence on the vasomotors.

20. It is certain that the dilatation of the vessels of the mesentery, under the influence of the depressor nerves of Ludwig, takes place through the bulbo-

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spinal nervous system; but sometimes reflex vaso-dilator actions are set up by irritation of the spinal cord itself, as in some cases of myelitis.

21. Lesion of the cervical region of the spinal cord is followed, as has been said, by dilatation of vessels; but this phenomenon is not invariably accompanied by increased heat. Hutchinson's case proves this. The body of the fifth cervical was crushed, and the cheeks of the patient became very florid, but also cold. A similar result is sometimes met with in lesion of the cervical sympathetic. The explanation of this is given in the succeeding section.

Consult—

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CHAPTER IV.

SPECIAL PATHOLOGY. THE EYE, MYOSIS, MYDRIASIS, GLAUCOMA, AND THE EFFECTS OF PRESSURE ON THE CERVICAL SYMPATHETIC.

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It has been clearly shown by experiment that section of the cervical sympathetic produces contraction of pupil, the sinking of the cornea, and narrowing of the palpebral fissure.

In Chapin's account of the history of the anatomy involved, it is stated that Valentiu was the first to describe a dilator muscle in the iris, but constrictor fibres had long been recognised. Arnold distinctly speaks of dilator muscular tissue in the posterior part of the iris, to which are distributed pale nerve fibres. Many others describe the same in man, and even their opponents are forced to admit the existence of a dilator muscle in the lower animals. Among those that deny this muscle in man are Grünhagen, Salkowski, Rouget, Arlt, and Bernard. They believe that the dilatation of the iris is due to the constriction of its blood-vessels. This is disproved by the fact that the pupils dilate before the vessels contract, and dilate after death. Müller describes a muscle of unstriped fibre receiving its nervous supply from the spheno-palatine ganglion, the action of which is to

draw the globe of the eye forward; also similar fibres in the lids, which tend to open the palpebral fissure. Sappey describes unstriated fibres in the orbital aponeurosis which would aid in producing exophthalmos. With the exception of one or two ciliary nerves of separate origin, all the branches destined for the iris and ciliary muscle proceed from the ciliary ganglion.

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What influence is exercised on the ganglia by each of the three nerves by which it is supplied?

1. The oculo-motor has undoubted action on the sphincter of the pupil. The pupil becomes dilated and immovable in paralysis of this nerve. This nerve is the condition, sine quâ non, both for reflex and accommodative movement of the pupil.

2. The filaments of the sympathetic, acting on the pupil, arises from the spinal cord, and pass into the anterior roots of the two lower cervical and six upper dorsal nerves. There is slight contraction of pupil on section of this nerve, and gradual dilatation on irritation of it in the neck. After division of the sympathetic in the neck, the upper part passes into fatty degeneration. The action of the sympathetic root consists in a persistent exaltation of tone of the radiating fibres. It is not proved that it acts on the accommodation. Irritation of the sympathetic in the neck causes contraction of the vessels of the iris. Dilatation of the pupil from irritation of the sympathetic nerves is not due to contraction of vessels (the diminution of blood in the iris lessening contraction

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of the sphincter muscle), but it depends on contraction of the radiating fibres of the iris.

3. The influence of the fifth nerve is doubtful, but it is probably sensory; as a motor influence, it may act on the ciliary ganglion, either to increase the action of the fibres of the oculo-motor, or to diminish that of the sympathetic.

If these views are correct—and they are those of Donders—the position of the sympathetic in the causation of myosis is not necessarily a very important one. Certainly in general paralysis of the insane, where the lesions are largely intracranial, the myosis is due to irritation of the oculo-motor nerve rather than to paralysis of the sympathetic; and if, in addition to this state of the third nerve, there be irritation of the fifth also, the effect of the sympathetic would be rendered nil. The influence of the sympathetic on the pupil can only be seen when neither of the other nerves supplying the ciliary ganglion is irritated or paralysed.

The intermissions of general paralysis depend on the greater or less congestion of the brain or its membranes. Vulpian, indeed, goes farther, and suggests that many of the so-called apoplectic attacks in this disease are not due to over-distension of the vessels, but to anæmia of parts of the brain, such anæmia being the result of reflex vasoconstrictor phenomena. The foci of white softening, sometimes found in general paralysis, may have this origin; but far more frequently this softening is the sequence of thrombotic blocking of minute arteries.

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'But besides the direct action of nerves on the size of the pupil, there are several reflex acts that are worthy of notice. In the first place, the pupil contracts on the admission of light to the retina, to a certain extent by a reflex taking place in the anterior tubercles of the corpora quadrigemina, between the optic and motor-oculi nerves. But it is also probably caused in other ways; for it occurs after the brain is removed, and also after death. Janeway also records a case in which there was blindness, with complete atrophy of the optic nerves and corpora quadrigemina, and yet there was perfect reaction of the pupil to light. Brown-Séquard and Harley think this takes place by a reflex in the ganglionic cells of the iris, the sensory nerves of the iris being directly affected by light. The pupil is dilated reflexively, through the sympathetic, by irritation of sensory or even tactile nerves. The absence of any sensory irritation during sleep is considered by Raehlmann and Witkowski the reason for the marked contraction of the pupil during this state. To this same cause is attributed the myosis in various forms of narcosis. It was first noticed in anæsthesia by Westphal. Labour pains, muscular movements, mental excitement, dilate the pupil through the medium of the sympathetic.'

In a recent case in the Bristol Royal Infirmary, marked myosis was caused by the pressure of an aneurism on the cervical sympathetic. A series of similar cases have been given by Dr. John Ogle. In one of these cases, described by Dr. Gairdner, there

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was not only contracted pupil, but a remarkable irregularity of temperature, cold sweats followed by flushing accurately limited to that half of the face on which the pupil was affected. In another case, a surgeon, in removing a tumour from the right side of the neck, apparently divided the nerve. Next day the face on that side was deeply congested, and marked with well-defined patches of a violet red. The right pupil was contracted, the conjunctiva normal. In another case, an American soldier was shot through the neck with a bullet. Two months after the pupil of the right eye was very small, that of the left eye unusually large. There was slight but distinct ptosis of the right eye, and its outer angle seemed to have dropped a little lower than the inner angle. The ball of the right eye seemed smaller than that of the left, and its conjunctiva was somewhat redder. The pupil was not only contracted, but slightly deformed, oval rather than round. In a dark place, or in half lights, the difference of the pupils was best seen; but in a very bright light the two pupils became nearly of equal size. The left eye watered a good deal, but had the better vision, the right eye having become myopic. He complained of frontal headache, of loss of memory, and of red flashes in his right eye when exposed to the sunlight, and also, after long exposure, in the left eye. With all this there were no abnormal retinal appearances. The patient's face presented a curious appearance after walking in the heat. It became distinctly flushed on the right side and pale on the

left. The flush extended to the middle line, but was less definite as to its limits on the chin and lips than above these points. Other cases have been mentioned by Eulenberg and Guttman.

That the size of the pupil may be altered by reflex irritation is well seen in hystero-epilepsy. M. Féré has found that compression of the ovary modifies the size of the pupil, whilst at the same time influencing the attacks. Where there is hemianæsthesia very precise relations exist between the conjunctival and corneal sensibility, and the area of the field of vision and the perception of colours. During the attacks he has noticed at first contraction of the iris, then dilatation, and afterwards alternations of contraction and dilatation in the various stages of the attack. In true epilepsy the pupil is usually dilated throughout, though occasionally it is contracted in the tonic stage.

It may be inferred that atropine acts on the nerve fibres, or on the ganglionic cells. (1) The sphincter muscle becomes paralytic ; reflex and accommodative movements are abolished, and, moreover, paralysis of accommodation (of the ciliary muscle) ensues, which however remains much longer incomplete than that of the sphincter of the pupil. Hence it follows that the elements of the oculo-motor nerve are paralysed, the more deeply-seated (of the ciliary muscle) being the last to be affected. (2) The dilator muscle becomes strongly contracted. The proof consists in the fact that, as Ruete was the first to show, in complete paralysis of the oculo-motor nerve, the size

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of the pupil is still considerably increased by atropia; additional dilatation also occurs under atropia after removal of the nerve in question in animals. To explain this we assume a stimulating action on the sympathetic nerve, which we can scarcely imagine to be persistent, unless it takes place by intervention of ganglionic cells. After a powerful effect of atropia further dilatation of the pupil will arise on stimulation of the sympathetic nerve; a proof that this nerve at least is not paralysed. If division of the sympathetic nerve have previously taken place, the pupil on the same side is not so fully dilated by atropia as that upon the other. Biffi and Cramer saw also in this phenomenon a proof of the stimulating action upon the sympathetic nerve. Donders cannot admit it to be such; the difference between normal and increased action of the sympathetic nerve, as opposed to paralysis of the oculo-motor nerve, may be sufficient to explain the difference observed.

Dr. William Ogle recorded a case, some years ago, which afforded a very important addition to the pathology of the sympathetic.

The case was that of a soldier with abscess of the right side of the neck, involving the sympathetic. At first his wife remarked that the right eye appeared smaller than the left one, and that the right ear was redder than its fellow. Two years afterwards his condition was as follows: The palpebral fissure of the right eye was much narrower than that of the left. This was partly due to a lowering of the upper lid, partly to an elevation of the lower one, and perhaps

rather more to the latter than to the former cause. The eyelids followed perfectly the motion of the eyeball when the patient was directed to look upwards or downwards. The right eyeball was somewhat retracted. The muscular motions of the eye were perfect. The right pupil was very much contracted. The contracted pupil reacted to the light but slowly, and within very narrow limits. The conjunctiva in both eyes was somewhat congested. The sight was equally good on the two sides, and on ophthalmoscopic examination the media and rètinæ were the same one side and the other. The ear on the right side was much redder than the ear on the left, and sensibly warmer to the hand. The skin of the right cheek and the skin just above the right eye were also sometimes, but not always, pinker than the corresponding parts on the left. The right side of the face was invariably hotter than the left. Notwithstanding the greater vascularity and the higher temperature of the right side, the man asserted that he only sweated on the left side of the face; that when he walked in the wind the left eye alone watered; that though abundant mucus was discharged from the left nostril, none ever came from his right one; and lastly, that the right side of his mouth often felt drier to him than the other, and that so dry altogether was his mouth, that he was obliged, for comfort, to take acid drops, so as to promote the flow of saliva. Both legs and both sides of the body were sweating equally. After violent exercise the relative conditions of the two sides of

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the face, as regards temperature, were inverted. When the man was at rest, the right side was the warmer of the two; when he had exerted himself, it was the cooler. In a Turkish bath not a drop of moisture appeared on the right side of his face, head, or neck, though the corresponding parts on the left, and all the rest of his body, were bathed in sweat.

Such were the symptoms. There can be no doubt that they were the result of lesion of the right cervical sympathetic, and of a lesion that was equivalent to division of that nerve. The abscess in the neck had probably eaten through, or, at any rate, thoroughly disorganised the nerve, and this probably not only at one small spot, but for some little distance, so as to prevent reunion. That the consequent hyperæmia should have persisted so long is somewhat remarkable; for, as a rule, this symptom is of comparatively short duration in animals; but on one occasion Schiff saw the hyperæmia persist in a dog two entire years without any great diminution. The retraction of the eyeball, the narrowing of the palpebral fissure, and the contraction of the pupil are all symptoms which result from section of the cervical sympathetic. It is an important point that the vascularity was not greater in one retina than in the other. Nor was there any morbid sensitiveness to light. This disposes of the opinion held by some physiologists, that the section of the nerve operates simply by exaggerating for a time the sensibility of the retina, as it does that of the integument, and that the partial closure of the eyelids and pupil is a

secondary consequence of that condition. It also goes far to bear out the statement of Schiff, that by no means are all the vasomotor nerves of the head contained in the cervical sympathetic.

That the right side of the face was redder and hotter than the left was accounted for by the palsy of the circular muscles of the blood-vessels and the consequent hyperæmia. What is astonishing is that violent exertion should have had just the opposite effect; but it is precisely what has been found in dogs by experiment, or by producing artificial fever, or by giving chloroform. The probable explanation is, that when the nerve-lesion first occurred, the hyperæmia of the right side was much more strongly marked than it is at present; that in those early days there was increased activity of secretion on the side of the nerve-lesion; but that gradually, owing to the contraction of the palsied muscular coat, the hyperæmia has been reduced to its present insignificant amount, and that in some parts of the face this contraction has so entirely balanced the vascular palsy, that in them there is no hyperæmia left, though at the same time their inability to sweat shows that they are in some abnormal condition. Heat, exercise, fibrile excitement generally, are unable to produce any great increase of vascularity, and so any secretion on the affected side, partly because of the unyielding nature of the arterial walls, partly because the actively dilating nerve fibres have been severed. The effect of these stimulants, which act with full force on the vessels of the opposite side, is therefore

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to invert the condition of the two sides as regards vascularity, and consequently as regards temperature.

Another case has been recorded that bears somewhat on this.

An officer was wounded on the right side of the neck; he suffered from slight ptosis of the right eye, with contraction of pupil and imperfect vision. Two years later there was no unilateral flush, but there was a marked defect in the secretion of sweat on the side of the lesion extending to the neck, arm, and chest, whilst on the left side, which was not wounded, the secretion was perhaps excessive in amount.

It is very doubtful whether lesion of the sympathetic has very much to say to the causation of glaucoma. The disease consists in an increase in intra-ocular pressure, generally depending on inflammation. 'Wagner thinks, from observation of two cases of glaucoma, accompanied by neuralgia of the fifth nerve, that the sympathetic may take part in the formation of glaucoma in three ways:

'1. It may be chiefly concerned in the inflammatory process.

'2. It may be irritated by pressure.

'3. It may be excited reflexively by the fifth.' The causes of the increased pressure are inflammatory changes (choroiditis and disorders of nutrition in the vitreous body).

Most physiologists deny the influence of the sympathetic in this disease, except in so far as it has to do with all inflammatory changes; but Schmidt-

Rimpler considers that the existence of an influence exerted by the sympathetic on increase of intra-ocular pressure, and thus on the occurrence of the glaucomatous process, is not to be denied.

So also in neuro-retinitis, and in ophthalmia neuro-paralytica, the influence of the sympathetic is almost nil, except that partial paralysis of the vaso-motors seems always one of the factors in the causation of inflammation.

In so-called sympathetic ophthalmia, it is far more probable that the irritation travels along the fifth nerve than by the circuitous route of the sympathetic and the cilio-centre in the cord. But perhaps the irritation travels by the sympathetic fibres contained in the fifth.

Division of the sympathetic in the neck is attended by a gradual diminution of the intra-ocular pressure. Most of the vaso-contractile branches of the eye join the sympathetic at the level of the superior cervical ganglion. The nerve fibres, through which the iris contracts, enter the cervical sympathetic at the middle part of its course.

As to the vasomotors of the brain and its membranes, some of the nerves of the pia mater, entering with its vessels partly into the cerebral cortex, come from the vertebral sympathetic plexus.

But the cranial vessels contract on irritation of the cervical sympathetic, especially of the upper ganglion.

As to the vasomotor branches of the ear, the

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vessels of the cavity of the tympanum become dilated after division of the cervical sympathetic on the same side. Variations, too, in pressure within the labyrinth are caused by much the same conditions as those of the intra-orbital pressure.

In lesion of the cervical sympathetic oculo-pupillary symptoms are more frequently observed than vasomotor. This is explained by Eulenberg and Guttman by the view that the oculo-pupillary fibres are more superficial in the ganglia than the vasomotors. But the ganglia are too small and too delicate in texture for this explanation. The real reason, probably, is that the vasomotor phenomena are more transitory than the oculo-pupillary. In some cases the lesion is of the spinal roots; and then the oculo-pupillary and vasomotor phenomena will only co-exist if the roots of the two first dorsal nerves, and of the third dorsal, are coincidently affected.

Traumatic paralysis of the brachial plexus may be accompanied by vasomotor and oculo-pupillary phenomena coincidently. Dr. Ross mentions a case in which there was rupture of the brachial plexus in a young man, aged nineteen. The patient's left arm had been caught, nine months before, by the strap of a revolving wheel of large diameter; he was lifted from the ground by the entangled arm, and fell on the opposite side of the wheel. When he recovered consciousness, it was found that his left arm was completely paralysed, all the muscles of the hand, forearm and arm, as well as the sternal portion of

the pectoralis major, were completely paralysed, and became atrophied. The clavicular portion of the pectoralis major, the pectoralis minor, the internal and external rotation of the humerus, and the latissimus dorsi were spared, while the serratus magnus on the left side was enfeebled, but not paralysed. The patient manifested on the left side relative contraction of the pupil, diminished palpebral aperture, flattening of the cornea, diminution of the intra-ocular pressure, and, five months after the injury, a slight relative increase of temperature in the left external auditory meatus.

Injuries to the cervical sympathetic, the cervical cord, and the brachial plexus lead also to redness of the conjunctiva and a flow of tears, due to functional derangement of vasomotor nerve fibres passing from the sympathetic to the first branch of the trigeminus, which supply the conjunctival vessels. Paralysis of these causes partial relaxation of vessels, an increase in the flow of tears, and of conjunctival mucous secretion.

Myopia, the necessary consequence of persistent paralytic myosis, is caused by the presumed direct influence of the sympathetic on the muscles of accommodation.

Similar phenomena are seen in non-traumatic diseases.

Myosis is met with in sclerosis of the medulla oblongata and in some diseases of the spinal cord, as tabes cervicalis and progressive muscular atrophy.

De Rosset's views as to the action of belladonna

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are important in considering myosis and mydriasis. 'The ciliary muscle contracts, is tetanised, not paralysed, when atropine is applied to it. The ciliary muscle is the only structure which actuates the lens. It has no special relation to the lenticular changes. Accommodation for a distance is an active product, whilst that for near points is passive, or due to resiliency only. Eserine paralyses the muscular system of the eye, just like pilo-carpine, which also paralyses.'

Harley and Meryon show that belladonna stimulates the sympathetic. It increases peristalsis, remedies night-sweats, diminishes glandular action and the bronchial secretion, and gives tone to the sphincter vesicæ in the enuresis of children.

Section of the sympathetic in the neck produces often an adjustment for near points, with myosis; but the results are not invariable, probably because all the sympathetic fibres do not pass through the first cervical ganglion, or through the portion of the trunk above that. An excitation of the oculo-motor may determine an apparent paresis of the sympathetic, whilst paralysis of the oculo-motor may give a dominant effect to the functions of the sympathetic, and the converse of both may be seen. What is said to be paralysis of the ciliary muscle, due to pressure on the third nerve with impairment of its neurility, may thus be interpreted to be a spasm due to the dominant effect of the sympathetic, or of some other nerve centre.

In a case mentioned by Dr. Chavasse, a girl, aged

six, was operated upon for a tumour lying in the position of the right submaxillary gland. It was found to dip down deeply into the neck. Posteriorly it was necessary to dissect away the trunk of the sympathetic nerve, as the tumour rested on the transverse processes of the vertebræ. At the conclusion of the operation the pupil of the right eye was seen to be contracted to the size of a pin's head. There was also drooping of the upper lid and well-marked diminution in the size of the palpebral fissure. The sight of the eye was unimpaired. Two months later the pupil of the right eye contracted irregularly, but responded slowly to alterations in the intensity of light. Accommodation good, as in the other eye. No appreciable difference could be detected in the size of the vessels of the retina. Two months later still the narrowing of the palpebral fissure was barely perceptible; ptosis was slight. The pupil was larger than it had been, but was not equal in size to its fellow. There was no emaciation of the cheek, and no flushings or redness of the conjunctiva on the affected side had been noticed. The symptoms did not improve beyond this. In the operation no large nerves were cut, but it is possible some very minute communicating branches may unknowingly have been severed. The trunk of the cervical sympathetic was grasped, and held for some time in a pair of forceps, and hence a pinching of the nerve elements and consequent interference with the function of the nerve.

Remak refers the decrease in size of the palpebral

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fissure, and the ptosis, when the cervical sympathetic is paralysed, to an alteration in the muscular actions of the levator palpebræ, orbicularis palpebrarum, and the retractor plicæ semilunaris, and thinks that these muscles are under the control of a tonic power communicated through this nerve-trunk.

In some of the observations of tumours of the neck the pupil of the corresponding side was dilated, and it has been thought that this was due to excitation of the cervical sympathetic.

Dr. John Ogle's observations, and M. Poiteau's, together form a total of nine cases in which this phenomenon of dilated pupil has been thought to proceed from a slight compression of the cervical cord, causing a stimulation of the muscular dilator fibres of the pupil. Vulpian throws some doubt on this explanation of the direct influence of the compression. He seems, indeed, to look upon it as depending on a reflex irritation of the sympathetic fibres of the iris. But surely Ogle's explanation is more truly analogous to the results of experiments on animals.

As has been stated above, lesion of the cervical sympathetic does not always induce both oculo-pupillary and vasomotor phenomena; but on one occasion Bidder excised a piece of the left cervical sympathetic from a half-grown rabbit. This was followed by the usual symptoms which result from the section of the cervical sympathetic. In about a month the rabbit had grown just as the other rabbits, and appeared quite sound. The left pupil was only half as large as the right, and the left eyeball projected

much less from the orbital cavity than the right. The left ear was distinctly broader and longer than the right, and was more hyperæmic and warmer. A fortnight later the difference in size was more striking. The author attributes the increase in the size of the ear to the increased continued supply of blood to the ear of the young animal, when all the nutrition processes are going on actively. Drs. Gee and Abercrombie have recorded a case of a child four years and a half old whose illness began with right hemiplegia and partial right ptosis with myosis, and passed on in wasting to paraplegia, an elastic swelling appearing on the right side of the spine in the upper dorsal and lower cervical region. Besides the myosis there was further evidence of sympathetic involvement, in the fact that the temperature of the right ear was three degrees higher than that of the left. The diagnosis arrived at was vertebral caries with abscess involving the inferior cervical sympathetic ganglia, and accompanied by a subacute descending polio-myelitis. The condition proved to be a sarcomatous tumour, seated deeply in the neck and upper thorax, and growing into the spinal canal through the foramina. The ganglion was not to be found, and the spinal cord showed some evidence of change in the cervical region and below.

The whole question of optical delusion is more or less under the influence of vasomotor action. In health, the impression of an external object is carried to the retina, and thence to the corpora quadrigemina. Thence it is transmitted to the angular gyrus as a

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sensory centre, and reflected on to the anterior lobes for perception. But in certain variations of the vascular tone of the vessels of the angular gyrus this centre seems to have the power of evolving optical delusions, wholly irrespective of external impressions. Many of the phenomena of febrile delirium, of delirium tremens, and of mania are produced by the vascular congestion or by anæmia of the angular gyrus. Probably, too, the false sensations of optical impressions depend on a similar congestive condition of the gyrus, including many of the varieties of hallucination and illusion.

In lesion of the cervical sympathetic, not only are the oculo-pupillary phenomena more frequently met with than the vasomotor, but the phenomenon in the domain of the vasomotor nerves is inconstant and transient. Vulpian thinks that there are vasomotor symptoms at the beginning of every affection of the sympathetic, which may disappear later; and this view is most in accordance with the result of observations of sympathetic lesions in other parts of the body. The alternation between irritation and paralysis is rare. Although a few cases are on record of injuries to the cervical sympathetic, chiefly from gunshot wounds, yet uncomplicated injury of this portion of the sympathetic system is very rare. Cases, too, of irritation of the cervical sympathetic are much less often met with than those of paralysis; but they do occur in connection with abscesses in the neck, with the pressure of tumours in the early stage, with injuries in the neck, injuries of the spinal cord and

lastly with extension of inflammation to the cervical ganglia from inflammatory lesions of the pleura. Such irritation is sometimes met with in the superior cervical ganglion. It is not very unusual, in tubercle of the lungs and pleura, to find the inferior ganglion with decided hyperplasia of the connective tissue, and sometimes even tuberculous. Paralysis of the cervical sympathetic, except from accidents, will most probably depend on the presence of tumours that have advanced so far as to have passed the stage of irritation, and to have led, either temporarily or altogether, to the destruction of the functions of the cervical sympathetic. The word 'temporary' can only be used in this connection when the tumour is one that can be cured, or can be eradicated without injury to the nerve. In some of the cases, for instance, in which the pressure had been caused by aneurism, the myosis has disappeared after ligature of the carotid.

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Rarely do oculo-pupillary phenomena occur from the pressure of glandular swellings. They are more common as the result of carcinomatous growths. Next perhaps in importance are mechanical injuries, especially gunshot wounds, affecting either the cervical sympathetic itself, or the brachial plexus, or a portion of the spinal column. The sympathetic is often involved in injuries to the spine or to the brachial plexus. Myosis is met with sometimes as the result of lateral curvature of the spine and of various diseases of the cervical cord, but this is by no means constant.

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In several cases of tumour pressing on the cervical cord, the writer has found neither oculo-pupillary nor vasomotor symptoms, except that in one case glycosuria was present. Two cases have been recorded of pressure of enlarged vessels upon the nerves giving rise to sympathetic symptoms. In one of these cases the pressure had induced unilateral sweating.

The sympathetic may be affected also in connection with pulmonary disease of the corresponding side. Is this connection direct or reflex? Sometimes reflex, sometimes direct. The ganglia may be compressed by tubercle in cases where the lungs are tuberculous, and especially where the lung of the corresponding side is thus affected. The unilateral blush of pneumonia, on the other hand, is more probably a reflex phenomenon.

The sympathetic is thus brought into association with a considerable number of diseases, not always as the structure primarily affected. This is especially the case with disease of the spinal cord, with cervical myelitis, tabes dorsalis, and progressive muscular atrophy. In somewhat the same degree is the sympathetic associated with progressive hemiatrophy of the face.

As regards diseases that are more closely connected with the cervical sympathetic, the influence of this portion of the nerve may be felt to a certain extent in glaucoma, neuro-retinitis, and ophthalmia neuro-paralytica; whilst it is of primary importance in some forms of optical delusion and in exophthalmos. In sleep, and in its antagonist 'insomnia,

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in sunstroke, in some forms of hemiplegia, in many varieties of headache, and particularly in hemicrania, in general paralysis of the insane, in paralytic dementia, in all the inflammatory diseases of the brain and its membranes, and in epilepsy, the rôle of the cervical sympathetic is an exceedingly important one. In the various forms of acute mania, where the symptoms of the greatest excitement alternate with periods of perfect health, the phenomena could not be reasonably explained, except by variation of the blood supply and the calibre of the vessels, nor can unilateral ephidrosis depend on anything, except on lesion of the cervical sympathetic, or of those nerves or nerve-centres with which the cervical sympathetic is closely associated. Dr. W. Ogle's case shows that this need not necessarily be on the side corresponding to the sweating. The sweating may be a normal process, the abnormality being the absence of this function on the opposite side, dependent on the long-continued lesion of the cervical sympathetic on that side.

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CHAPTER V.

EXOPHTHALMIC GOÎTRE.

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THE protrusion of the eyeballs, the enlargement of the thyroid gland, and the cardiac palpitation may exist in exophthalmic goître in such varied degree, that it is difficult to assign an overwhelming importance to any one of them. It will be seen, however, that, much as the protrusion of the eyeballs may be delayed, little as the thyroid gland may be enlarged, it is exceedingly rare that the palpitation on slight exertion should not show itself at an early stage of the disease.

Certain coarse lesions have been found in the cervical sympathetic with this group of symptoms. Eulenberg and Guttman have collected nine such cases. The inferior cervical ganglion is most frequently affected. In Dr. Warner's case there were lesions of the sympathetic on one side of the head. The right side of the face was flushed up to the middle line, the right pupil dilated, the right iris much darker in colour of late years than the left, the thyroid normal.

In a case quoted by Woods, the lower cervical ganglia, especially the right, were thicker and redder

than normal. There was increase of connective tissue, growth of nuclei and of spindle-shaped cells; the ganglion cells were few.

In Dr. Shingleton Smith's case there was marked shrinking of the cells of the inferior cervical ganglia. In one case, mentioned at the International Medical Congress in London by Dr. Guéneau de Mussy, there was decided pigmentation of the face, due to defect of innervation, caused by the enlargement of thoracic glands about the bronchi and trachea in the immediate vicinity of the vagus, giving rise to irritation of the vagus. This association of exophthalmos with pigmentation has also been observed by the writer in one case.

Seeligmüller mentions a smith, in whom, eight days after a severe blow above the clavicle, dilatation of the pupil and of the palpebral chink, exophthalmos, pallor, diminution of temperature, and flattening of the left cheek occurred, depending on clonic narrowing of the arteries. Other observers have noticed induration and hypertrophy of connective tissue, chiefly of the lower cervical ganglia, with pigmentation of cells. Perhaps the most consistent view has been expressed by Professor Mobius, when he says that exophthalmic goître is not an independent disease, but is a group of symptoms, partly depending on local lesions of the medulla oblongata, or of the cervical cord, or of the sympathetic, but partly occurring more or less in the course of severe neuroses, such as hysteria and the various psychoses.

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But whilst some of the phenomena point to lesion, direct or reflex, of the sympathetic, it is not possible to credit this system of nerves with the causation of all the symptoms.

The exophthalmos itself could only be associated with the sympathetic by means of a persistent tetanic contraction in the unstriped ocular muscles, and for this there is little physiological analogy. This protrusion of the eyeball seems to depend on deposit of fat behind the eyeball and on venous hyperæmia, even if some spasm of the unstriped orbital muscles co-operate in its production.

One symptom connected with the eye, first noticed by Von Graefe, is due to disturbed innervation of Müller's unstriped orbital muscles; an immobility of the upper lid, which no longer follows the movements of the eyeball, as in health. This interference with the consensus of the movements of the lid with that of the globe, especially when the latter is directed downwards, may precede the exophthalmos, and is not seen in protrusion of the eyeball from mechanical causes.

The two chief sympathetic symptoms then are the goître and the accelerated action of the heart, and both these phenomena are connected with paresis of nerve rather than with irritation. The goître seems wholly caused by enlargement and dilatation of vessels in the thyroid. The arteries are tortuous and pulsating and the veins engorged. The temperature of the part is somewhat higher than that of other parts of the body. It is true that division of

the cervical sympathetic is not followed by swelling of the gland, but in exophthalmos there are not only conditions answering to division of the sympathetic in the neck, but extreme acceleration of the heart's action as well.

The palpitation is more difficult of explanation. If it be due to irritation of the sympathetic, the irritation must be persistent, which is contrary to physiological experience. If due to paralysis, the usual effect would be syncope; because, the influence of the sympathetic cardiac nerves being cut off, the inhibitory action of the vagus would suffice to antagonise the automatic energy of the cardiac ganglia. Eulenberg and Guttman seek to explain it by allowing a paralysis of the cervical sympathetic, the first effect of which will be a vasomotor dilatation of the cardiac vessels (the coronary arteries); and this dilatation, by permitting a greater flow of blood to the muscular tissue of the heart, stimulates the cardiac ganglia to abnormal activity.

It seems strange, however, that a similar result does not always ensue when the action of these cardiac nerves is cut off. What need, indeed, is there, under this theory, that syncope should ever take place? Dr. Handfield Jones, however, believes that the palpitation in exophthalmic goître is due to paralysis of the vagus.

The implication of the sympathetic is shown by the perspiration and diarrhœa so common in exophthalmic goître, as well as in the occurrence of pigmentary disturbances, instances of which have

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been already given. Trousseau has seen vitiligo in connection with it, and Leube scleroderma.

As in many diseases that own a sympathetic origin, lesions of the ganglia are not always to be demonstrated. Nor is the pathology of the affection free from controversy. The phenomenon has been sought to be explained by poverty of blood, by an affection of the heart, by a special disease of the nervous system, by the mechanical effects of the goître. With reference to some of these so-called explanations, it is enough to point out that the symptoms of exophthalmic goître are seldom if ever met with among the large class of chlorotic and anæmic patients; that of the numerous conditions of the heart associated with palpitation, acceleration of the cardiac action has never been known by itself to lead either to the thyroid enlargement or to the exophthalmos, to say nothing of the immobile condition of the eyelid; whilst mechanical pressure of the goître is proved to be unable to produce the same phenomenon, partly by the large number of cases of thyroid enlargement in some specially goîtrous districts, in whom no cardiac or exophthalmic symptoms are found; partly by the fact, so frequently recognised, that all swelling of the thyroid may be absent throughout the whole course of a case of exophthalmos, whilst very often the condition of the heart and eyeball precede for a considerable time the swelling of the thyroid gland.

One case is on record in which the lower cervical ganglia, especially on the right side, were thicker and

redder than normal. The connective tissue was increased, as well as the nuclei and spindle-shaped cells. The ganglion cells were few in number.

Besides the leading phenomena referred to above, there have been noticed various coincident paralyses of face or of some of the muscles of the eyes, ulceration of the cornea, and dilatations of the retinal veins. The pupil is generally normal, but is sometimes constricted. Dilatation of the pupil is not so common; but in a recent case the writer observed great dilatation of the left pupil, with exophthalmos, enlarged thyroid, palpitation, and crepitation at the right apex.

Great irritability is sometimes present, and some intellectual disturbance, which, in a few instances, has developed into mania. There is generally some elevation of temperature. Sensory disturbances of the fifth nerve are sometimes coincident with exophthalmos, with occasional redness of the face, unilateral or on both sides. It will be seen that the infrequency of this symptom is very remarkable, taken in connection with the dilatation of the thyroid vessels. Basedow says the disease is a result of a scrofulous dyscrasia; Aren calls it a neurosis of the sympathetic; Trousseau, a congestive neurosis of the whole ganglionic system; Charcot believes in a psychical origin; Jaccoud considers it a paralysis of the vasomotor centres and cervical ganglia, and says that the beating of the heart is due to the less resistance in the dilated vessels, that this dilatation is increased by the heart-beats, that thence there is

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tumefaction of the thyroid gland, that the cerebro-spinal axis is in its turn excited by the peculiar blood-flow, that the brain shows psychical disturbance, and the cilio-spinal region produces exophthalmos by spasm of the orbital muscles and of the muscles of Müller, and dilatation of pupil by spasm of the radiating fibres of the iris. Daviller believes it to be an anæmia of the cilio-spinal regions of the cord, causing an exaggeration of the reflex power; whilst Eulenberg and Guttman think it a paralytic condition of the cervical sympathetic.

Of the exophthalmos, the goître, and the cardiac disturbances, no one of the three causes the rest. Certainly the phenomena cannot be caused by the compression of vessels or of the cervical sympathetic by the goître. Experiment on the cervical sympathetic may produce all the phenomena, but not coincidently. They are generally associated with a neurotic diathesis and history. Albuminuria and diabetes may accompany them, the albuminuria being due to paralysis of the sympathetic of the kidney, coming from the renal plexus (Begbie). The symptoms may be associated with psychical phenomena. Thus three cases of completely developed Graves's disease have come into Bethlehem Hospital within the last few years. All three of these cases have been young women, and there was evidence of hereditary insanity (in two of them in the direct line). One case began, as far as was known, with exalted delirium and excitement; the other two were originally depressed, and had melancholic delusions;

but in all there was a tendency to impulsive violence, to refusal of food, and to dirtiness of habits; the sexual functions seemed little or not at all affected. With these may be connected three other cases in which some of the symptoms of Graves's disease have been noted. In one, an instance of recurrent mania in a lady of twenty-one, most marked exophthalmos is present with each recurrence of excitement; this patient was most markedly benefited by hyoscyamine. In a second case, the symptoms are slowly developing (1880); and in a third, a male of thirty-nine, the exophthalmos accompanied general paralysis, and his pulse varied from 100 to 150.

M. Noel Guéneau de Mussy has described four cases which have come under his notice. Two of these occurred in males and two in females. In all four the ordinary symptoms were observed, while two were conspicuous for enlargement of the tracheobronchial glands, two for distinct choreiform movements, and two for muscular weakness and trembling of the limbs. In two there were cardiac complications, and in one pigmentation of the face. M. G. de Mussy, who is an advocate of the theory of defect of innervation, lays great stress on the enlargement of the thoracic glands about the bronchi and trachea, and in the immediate vicinity of the pneumogastric nerve. These may form, he thinks, the initial factor of the phenomena of the disease. In support of the defect of innervation theory he further adduces the pigmentation of the face, of which he sees

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a plausible explanation in the irritation of the pneumogastric nerve.

It would seem strange pathology to attribute one set of symptoms to paralysis of the sympathetic and another to irritation, although this has been done by some. Thus it may be said that the exophthalmos may be a result of a spasm of the muscular fibres of Müller, and that the palpitation is the result of over-activity of the sympathetic cardiac nerves. But the swelling of the thyroid gland, the consequence of accumulation of blood, the vascular state of the head, the arterial pulsation, are all due to dilatation of vessels; and this dilatation is usually met with as a result of sympathetic paralysis. To meet this difficulty it has been suggested by Poincaré and others that the face is not always congested, that it is more often pale, but that it varies from moment to moment, and that it certainly gives no evidence of vasomotor paralysis; whilst the disturbances of the pupil vary, the pupil being sometimes dilated, sometimes contracted; and that the enlarged thyroid is not the result of a purely passive congestion, but of an active one, and is probably rather the consequence of vaso-dilator activity than of vaso-constrictor inertia. The vessels are in a peculiar condition. There is often an irregular spasmodic constriction, forming little vascular ampullæ, and almost amounting to an erection. The arterial beats are not constant and regular, but appear and disappear, unlike the state of arteries dilated from sympathetic paralysis, and are due rather to energy of the cardiac impulse than

to relaxation of the vascular walls. The general excitation of other portions of the sympathetic system seems to bear out this view. An exophthalmic patient is apt to suffer from abdominal pain, possibly due to intestinal contraction. Diarrhœa may be present, not a mere flux, a relief from overdistended paretic intestinal vessels, but from hypersecretion. Catarrhal bronchitis is met with, excessive emotion, and even mania, as before stated.

It is only right, in the interests of science, to mention cases in which post-mortem appearances have been found. To theorise truthfully is only possible on ascertained data; and the important decision between a pathological anatomy of sympathetic irritation and sympathetic paralysis can only be arrived at by the records of fatal cases.

In a case mentioned at the Société de Médecine of Paris, the sympathetic nerve was carefully examined. The lower cervical ganglia, especially on the right side, were much enlarged and reddened. Under the microscope the ganglionic structure was found almost obliterated, what remained of it being inclosed in the meshes of hypertrophied connective tissue, which composed the greater part of the ganglion. Predominance of the connective element and diminution of the nervous element constituted the alteration. In a case of Dr. Reith's, the left eye protruded very much, and seemed to be larger than the right. The right eye was normal for a long time, but became nearly as prominent as the left. The eyeballs were motionless, but sight was good. The

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patient had complete power over the eyelids, but could not close them over the eyes. Pulse 100, full and regular, but quickened before death, which took place two days after he came under observation. At the autopsy the glands of the neck were somewhat enlarged, hard, but not adherent, not pressing upon the vessels or nerves; the thyroid body about half as large again as usual, the enlargement being entirely produced by the lateral lobes extending upwards by the side of the thyroid cartilage, but not pressing either backwards or outwards, and therefore not pressing upon the vessels or nerves, and not forming any marked external tumour. The pericardium contained about 3 oz. of pale albuminous serum, but without any trace of disease. Liver large, weighing 64 oz. Spleen large, weighing 20 oz., much loaded with blood; splenic pulp very friable, breaking down with the least force. The sympathetic nerves of both sides, but especially of the left, large; the middle and lower cervical ganglia of the left side much enlarged, very firm and hard. The middle cervical ganglion thick in length, varying in width from $\frac{1}{8}$ to $\frac{1}{4}$ inch, the lower cervical $\frac{7}{8}$ inch long, nearly uniformly $\frac{1}{4}$ inch wide; the connecting cords correspondingly enlarged. Of the branches, those proceeding from the middle cervical to the inferior thyroid artery, and those from the lower cervical to the vertebral artery, were much more enlarged than the remainder, which varied little from the normal size. Under the microscope the ganglia seemed loaded with granular matter, observing to a great

extent the appearance of nerve tubes and cells, and resembling more than anything else the aspect of a lymphatic gland in the early stage of tuberculous deposit. The dorsal, lumbar, and semilunar ganglia of nearly the natural size. On the right side the middle and lower cervical ganglia, especially the latter, were enlarged, firm, and hard, presenting appearances similar to those of the left side, but not to such an extent. The cellular tissue surrounding the ganglia of both sides was thickened and hardened.

This case is somewhat peculiar by the absence of any record of palpitation. As has been said, the goître may be absent, the exophthalmos may be very late in its development, or may never be present; but the cardiac phenomena are in most instances early, persistent and inevitable.

Careful examination of the cervical sympathetic has been made also in a case of exophthalmic goître by Dr. W. Hughes. The chief point observed in the cervical ganglia was their dense infiltration with round cells. A certain number of cases seem to own a reflex origin, and here coarse recognisable lesions of the cervical ganglia are not to be expected. The fact, too, of this reflex origin tells more in favour of the irritation theory than of the paralytic.

Such a case is reported by Mr. Hutchinson. Six months before admission proptosis occurred; four months afterwards the thyroid became enlarged. On admission, the right lobe of the thyroid was larger than the left, but neither was greatly enlarged. Distinct pulsation was felt over both lobes. Occa-

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sionally she suffered from palpitation, which she thought was due to nervousness. Palpitation had come on quite recently since she noticed the proptosis. She had a voracious appetite, never feeling satisfied. Had suffered from diarrhœa continuously for the last four months. Had lost a little flesh. Since the onset of the disease has become very cross-tempered, and ready to snap at anything, without being able to help it. She often became flushed without any cause whatever, and at night complained of heat. Sometimes she saw double, but this was not constant. Had muscæ volitantes. The catamenia were regular, but had been very scanty the last four or five times; and the first favourable symptom was the return of the catamenia to their natural condition. The only physical sign of cardiac disturbance was that the first sound was much intensified. Under belladonna she became much better.

The symptoms coexist sometimes with cerebral phenomena, or with some cerebral lesion

Dr. Fenwick has recorded cases of a similar nature, in one of which, besides the proptosis, the bronchocele, and the palpitation, there was cessation of the catamenia, tinnitus, occipital headache, flushing, thirst, and alternate diarrhœa and constipation; in another, frontal headache, irritability, subjective feeling of heat, with arrest of the catamenia.

Injury to the restiform body may lead to exophthalmos in animals; and with reference to this Brown-Séquard has observed exophthalmos in animals

born of parents in whom protrusion of the eyeballs had followed this injury. This interesting fact has been witnessed a good many times; and Brown-Séquard has seen the transmission of the morbid state of the eye continue through four generations. In these animals, modified by heredity, the two eyes generally protruded, although in the parents usually only one showed exophthalmos, the lesion having been made in most cases only on one of the corpora restiformia.

Dr. Banham has recorded a case with severe paroxysms of neuralgia, affecting the whole of the left side of the face, but at times concentrating itself with special violence at the supra- and infra-orbital regions, and over the malar bone. At such times there is great tenderness over the affected parts. The patient complains also of a constant feeling of formication, especially in the skin about the chin. There was ptosis of the left eyelid, dilatation of the left pupil, some amount of external strabismus, the movements of the eye being extremely limited. Marked protrusion of the left eyeball: some deafness of the left ear, apparently of central origin. There were also other paralytic symptoms—slight protrusion of the tongue to the left, suspicion of some amount of paralysis of the right side of the face, defective sight of the right eye. The diagnosis was of a tumour at the base of the brain, corresponding with the middle fossa of the skull; its influence by meningitis or otherwise extending beyond this limit so as to affect the auditory and hypoglossal nerves.

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The case is interesting in this relation, as an example of paralytic exophthalmos.

Dr. Story has recorded a case in which symptoms of exophthalmic goître occurred temporarily from sudden shock. A young girl opened a letter telling of her brother's death. The pulse became 140, with exophthalmos and thyroid enlargement. In forty-eight hours the exophthalmos receded, and her pulse fell to normal. The disease appeared to be of neurotic origin.

Dr. G. Ballet has reported several instances of this disease, in which the symptoms of exophthalmos were accompanied with epilepsy, or with epileptic attacks, or with paralytic phenomena, either hemiplegic or paraplegic, or some decided weakness of certain voluntary movements, or with hemianæsthesia, or even with aphasia. He speaks, too, of polyuria, glycosuria, and albuminuria being occasionally coincident with these symptoms of exophthalmic goître. Dr. Pierre Marie considers the morbid phenomena to be due, not to alteration of this or that nerve, not even to a neurosis of the vagus, or of the great sympathetic, but to a condition having the character of a general neurosis. Thus there are found, coincident with them, symptoms that point to a disturbance of the functions of the central nervous system, such as paroxysmal diarrhœa, with sudden onset and equally sudden termination, violent attacks of sudden hunger amounting to boulimia, uncontrollable vomiting, præcordial pain simulating angina pectoris, rapid breathing, frequent

dry cough, abundant sweating, with a subjective sensation of heat, cutaneous lesions like vitiligoo pigmentation, loss of eyebrows and eyelashes, urticaria, &c.

An important symptom is tremor, generally more or less present. This tremor is somewhat peculiar. It may be general or confined only to the extremities, especially the upper extremities, often enough to prevent the use of the needle or the pen. It sometimes can only be recognised if the arms are extended. It is more rapid than in senile trembling, or paralysis agitans; the oscillations amount to $8\frac{1}{2}$ per second, whilst in the latter condition the oscillations are only 5 or 6 in the same lapse of time. This tremor was especially remarkable in a case of Dr. Guéneau de Mussy, and was accompanied with pigmental patches on the face, particularly of the temporal fossæ.

The possible central origin of this disease is also seen experimentally, as the palpitation, the goître, and the exophthalmos have been produced by cauterisation of the upper fourth of the restiform bodies. But although these three symptoms can be thus induced by experiment, they do not make up exophthalmic goître, as described by Basedow and Graves. Over and above these phenomena there is a peculiar nervous condition, of which the tremor is one of the most constant symptoms. On only one occasion has experiment induced in the same animal the three cardinal symptoms; but Filehne, by the operation on the restiform bodies above-mentioned, implicated the vagus, and thus caused acceleration of the heart's

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action by paresis of the inhibitory nerve. He was able also to induce exophthalmos, usually more pronounced on one side than on the other. It occurred even if the sympathetic had been previously divided in the neck, thus excluding the possibility of its being due to spasm of the musculus orbitalis. Filehne formulates the following propositions :—

1. Basedow's disease may be produced by paralysis of certain nerve-regions, which are controlled by the medulla oblongata. The points traversed in common by the nerve-paths concerned are the restiform bodies.

2. Under such circumstances the exophthalmos and the goître depend on dilatation of the blood-vessels.

3. The increased rapidity of the heart's action is brought about by diminution or abolition of tone in the vagus.

4. That Basedow's disease in human beings depends on the same physiological relations is highly probable, but must first be established by proofs afforded by pathological anatomy. In this investigation attention should be directed not only to the medulla oblongata, but also to the condition of the trunk of the cardiac portion of the vagus.

Warner has described a case of Grave's disease, complicated by ophthalmoplegia externa, as well as by bilateral paresis of the seventh and fifth nerves, and by tremor of the legs. The association of the phenomena of Graves's disease with symptoms evidently of a central origin render probable the central origin of the exophthalmic goître. Féréol has ob-

served the following symptoms (certainly pointing to a coarse brain lesion) come on six months after the onset of Basedow's disease in a man forty-one years of age; pain in the head, vomiting, giddiness, tremors, reeling gait, with a tendency to fall to the right; subsequently diplopia, due to paresis of the right fourth nerve, and in addition, on the right side diminished motor power, with hyperalgesia, and on the left, analgesia.

The localisation of the lesions of Graves's disease in the medulla oblongata is almost precisely that which has been marked out by Eckhard on the vasomotor centre, puncture of which will lead also to glycosuria.

Instances, too, of central lesions have not been wanting in several cases. In one, recorded by Morell Mackenzie, Lockhart Clarke found, post-mortem, that the substance of the cerebral tissues was not unnaturally soft, there was no clot or embolism anywhere, but the corpora quadrigemina and the medulla oblongata, particularly on its posterior part, were very soft, and on minute examination displayed the usual appearance of common softening.

Dr. Cheadle has published a case in which, although the brain and spinal cord seemed perfectly normal to the naked eye, microscopical examination showed very great dilatation of the vessels of the medulla oblongata and cervical region of the cord. The cervical sympathetic showed no abnormality whatever. This affection of the vessels Dr. Cheadle considered as probably a result of the disease, and he says that the condition of the nervous system is

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probably more one of disorder than of organic morbid change; but the results of this autopsy induced him to suggest the localisation of the lesion in the vasomotor and cardio-inhibitory centres.

It seems generally agreed that the right eye is usually affected with exophthalmos before the left, and that the right side of the thyroid gland is enlarged more frequently and more early than the left. It is interesting to remember (looking at the morbid phenomena as due to paralysis rather than irritation) that the right pneumo-gastric is the one almost wholly concerned in the cardiac inhibition, the left having little to do with it. The palpitation is in most cases the earliest symptom. It is by far the most important; and in those rare cases in which it is absent, it is where the left side, both as to eye and thyroid, is attacked, and where therefore presumably the paretic affection of the left vagus, which sends so slight a connecting link to the cardiac plexuses, interferes but little with the ordinary nervous antagonism of the heart. At any rate irritation of the cardiac branches of the cervical sympathetic does not explain these exceptional cases of left exophthalmos without palpitation. Dr. Fitzgerald's theory that the right vagus inhibits the heart almost wholly, because in the embryo the heart early projects towards the right side, may or may not be true; but both experiment and pathology go to prove that cardiac inhibition is one of the main functions of the right vagus, whilst the left has little or nothing to do with it.

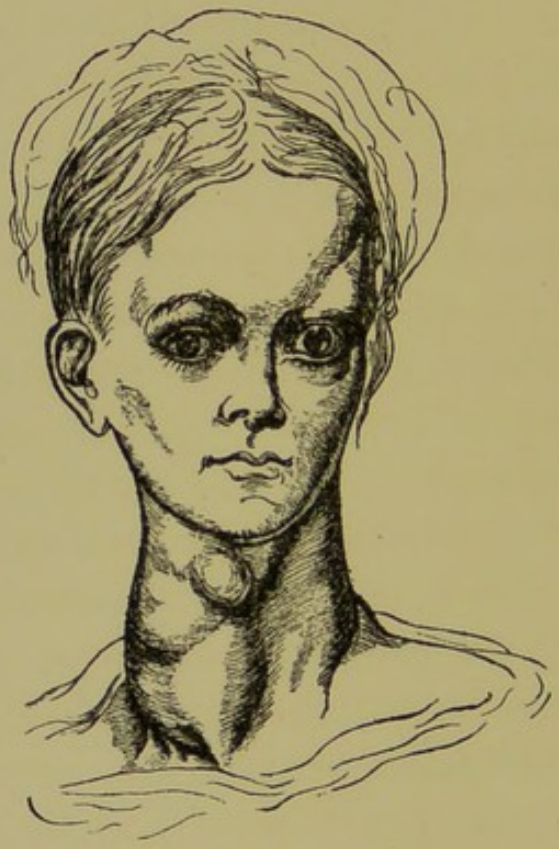
The two phenomena that there is special difficulty in explaining on the theory that some affection of the cervical sympathetic is the cause of the disease are—(1) the impairment of the consensual movement of the upper eyelid in association with the eyeball; and (2) the abnormal widening of the palpebral fissure, due to retraction of the upper lid, and the incompleteness and diminished frequency of the act of involuntary winking. The defect of co-ordinate movement between the upper eyelid with the eyeball is an early symptom, and is wanting in protrusion of the eyeball from any other cause. Whatever may be the true explanation of the normal condition, that when the globe is rotated downwards, the upper lid follows it, it is necessary that there should be an inhibition of the levator palpebræ. In whatever way this inhibition is interfered with in exophthalmic goître, it is more reasonable to suppose that it is a paralytic action than a spasmodic one; and, if paralytic, then certainly central. In one very remarkable case, where the exophthalmos was unilateral, this phenomenon of Graefe's was present on both sides. Sattler says, with reference to this central origin of Graefe's phenomenon, 'there is no doubt whatever that the movements of the lids in association with the raising and lowering of the level of fixation, that is to say, the consensual action of the levator and orbicularis on the one hand, and of the ocular muscles which rotate the eyeball about a horizontal axis on the other, are presided over by a definite co-ordination centre, just as much as are the associated movements

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of both eyes!' But it is probable that Dr. W. A. Fitzgerald is right in saying that it is exceedingly doubtful whether the orbicularis takes any part in the movements of the eyelids in association with the eyes. All that seems necessary for the descent of the upper lid is an inhibition of the levator palpebræ, and this is probably brought about by the action of an associated centre for looking down, which simultaneously causes contraction of the muscles which rotate the eyeball downwards. One of the main objections to the sympathetic origin of these phenomena is that spasm of the unstriped muscular fibres of Müller in the upper and lower lids would mean permanent irritation of the sympathetic, a condition that is against all analogy.

Sattler thinks also that in an analogous manner are to be explained the two other lid-symptoms, the increased gaping of the palpebral aperture, as well as the diminished frequency and incomplete character of the act of involuntary winking. It is well known that the width of the palpebral aperture, and also the completeness and number of the involuntary descents of the upper lid, occurring in a given time, stand in a reflex relation on the one hand to the amount of light, which stimulates the retina, and on the other to the amount of stimulation applied to the sensitive nerves of the anterior surface of the globe, which latter is of course under normal conditions very slight. And this is illustrated in the most striking manner by the narrowing of the aperture, which invariably occurs on exposure to dazzling

PLATE VIII.



Crossed Exophthalmic Goître.

Dr. BURNEY YEO's case.

light, by the characteristic and involuntary position of the lids, which is often noticed with corneal nebulæ, by the marked sinking of the upper lid, which accompanies even the slightest irritation of the conjunctiva or cornea, and so on ; and again by the abnormal width of the aperture, which impresses such a peculiar character on the gaze of amaurotic patients. There clearly exists here a similar reflex relation to that between the size of the pupil and the amount of light stimulating the retina.

He sums up the whole question thus. 'We have now seen that the essential, and to a certain extent constant, symptoms, which make up the comprehensive picture of Basedow's disease, and which show themselves in such various and widely distributed organs, can be explained in an uniform manner by the assumption of a lesion of certain centres, which leads to the impairment or abolition of the functions presided over by these centres, and in fact, more precisely expressed, of a lesion implicating the tone in the vagus centre which regulates the cardiac movements, or the peripheral and still unmixed nerve-paths proceeding from it ; further, the vasomotor centres for definite regions of the body, especially of the head and neck, and finally the centres for certain co-ordinated movements and reflex actions.'

The plate, used by the kind permission of Dr. Burney Yeo, is that of a case in which, with profuse perspiration, and constantly recurring diarrhœa, the exophthalmos implicated the left eye only, whilst the right side of the thyroid was enlarged. This crossing

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of the symptoms seems to Dr. Yeo, and probably with great truth, to prove the central origin of the lesion.

It has been seen, therefore, that the pathology of this disease has been constantly a subject of controversy. Each of the three theories are supported by influential authority; each more or less successfully accounts for the main phenomena of exophthalmic goître. Some observers hold to the opinion of irritation of the cervical sympathetic; others to paralysis of the cervical sympathetic; others again to the central origin of the disease, the lesion being situated in the vasomotor centre in the medulla oblongata, and implicating the vagus nucleus and parts in its immediate neighbourhood, the lesion being a paralysing one. The possibility of the symptoms being due partly to irritation, partly to paralysis, seems such a contradiction in terms, that it may be dismissed.

It is probable that under any one of the three theories the phenomena might be induced by reflex action.

The theory of irritation of the cervical sympathetic is wanting in the following points:—1. That persistent and continuous irritation for a period of many years is practically unknown. 2. That even under long-continued irritation there would be a certainty of nerve exhaustion, causing temporary diminution or complete remission of symptoms. 3. That the paralysis of the levator palpebrarum is not accounted for. 4. That under this theory, the vagus is left normal, the inhibitory function of this nerve on the heart remains in full activity, and would be capable of antagonising the effects of irritation of the cardiac

sympathetic branches. The theory of paralysis of the cervical sympathetic is deficient, in that paralysis of this nerve would lead to retraction of the globe of the eye rather than exophthalmos, and the explanation of protrusion of the eyeball being induced by the pressure of dilated paralysed vessels behind it insufficient. This theory will explain the thyroid enlargement; but even here it would be remarkable that there is seldom any coincident vascular paralysis of the face and ear, whilst there is a difficulty under this theory of explaining those cases in which the goître is absent. Here, too, the palpitation has to be accounted for by a strange idea, viz., that paralysis of the accelerator branches to the heart induces increased cardiac action by the passive dilatation of the cardiac vessels, allowing a larger amount of blood to circulate in the structure of the heart, so stimulating the ganglia of the heart, and intensifying their action. Here again, with an uninjured vagus, it is difficult to believe that such increased functional activity of the cardiac ganglia would overcome the inhibitory antagonism of the vagus. Nor does paralysis of the cervical sympathetic, any more than its irritation, account for the condition of the levator palpebræ.

There remains then the question of a central origin. As a matter of experiment exophthalmos has been found to be a result of section of the restiform bodies, and therefore may be expected to manifest itself from any destructive lesion of that, if not of other portions of the medulla oblongata. Lesion

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also of a portion of the floor of the fourth ventricle would account for any interference with the independent or co-ordinate action of the levator palpebræ, as one portion of the origin of the fourth nerve can be traced into a grey nucleus at the upper part of the floor of the fourth ventricle, close to the origin of the fifth nerve.

Without going so far as to demand the presence of vaso-dilator nerves to explain the size of the vessels of the thyroid, and the consequent tumefaction of that organ, it is enough to know that the lesion of a small portion of the fourth ventricle on each side of the middle line will include the chief vasomotor centre of the body, and that an extensive lesion of this region would lead to paretic dilatation of all the vessels of the body. The weak point in this theory of central origin seems to be that there is so seldom any dilatation of other vessels beside the thyroidal. Paralysis of the vagus, a necessary sequence of any lesion of the vagus nucleus, would explain the dilatation, simply from the action of the accelerator nerves and the cardiac ganglia uninhibited by the proper functions of the vagus.

Under this theory, therefore, the phenomena of exophthalmic goître are more or less satisfactorily explained ; and a somewhat paralytic lesion or lesions of the medulla oblongata (such lesions being possibly mere disturbances of innervation of the part from deficient nutrition, &c., and even under certain circumstances being set up by reflex action from a distant organ), would be more likely to be associated

with other paralytic or semi-paralytic phenomena. This theory of central origin, too, does not militate entirely against the implication of the sympathetic. The lesion is in great part one of the chief vasomotor centres. The very fact that section of the restiform bodies leads to exophthalmos and to goître is proof positive that the fibres that rule the constriction of the vessels of the thyroid, and behind the globe of the eye, are paralysed by this section. Such fibres can only pass down the upper portion of the cervical cord, and thence to the superior ganglia of the sympathetic. The frequent absence of oculo-pupillary phenomena in this disease can be easily understood by the fact that these fibres are given off to the superior cervical ganglion before the oculo-pupillary centre in the cord is reached. The presence of this vascular dilatation in the orbit and in the thyroid gland, whilst flushing of the face is only exceptionally met with, is only explicable on the view that this negative, this paralytic condition of nerves from the medulla oblongata affects only certain of the cells of the superior cervical ganglion. It is reasonable to believe that each cell in a ganglion has its own special function, and rules some particular region. Of those which are vasomotor cells, each one probably regulates the calibre of a single vessel, or even of a single branch of a vessel. There is no greater difficulty in believing this than in receiving the teaching both of experiment and pathology that the same ganglion comprises cells with oculo-pupillary and vasomotor functions.

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Thus, whilst the central origin theory gives the only reasonable explanation of the palpitation, and of Graefe's phenomenon, it accounts for the other important symptoms of exophthalmic goître by the lesion being of a vasomotor centre, the effects being modified and specially localised by the influence of the cervical sympathetic. The variation in the presence of the leading symptoms is illustrated by the following cases, taken almost hap-hazard from the writer's case-book :—

1. Mr. E. S., 28.—Ill five years with goître, exophthalmos, and palpitation. Four years ago the goître was large; now all thyroid swelling is gone. Diuresis.

2. Miss A. D., 15.—Eyes prominent. Thyroid body has been large for some years. Palpitation only during the last four months.

3. Miss S., 30.—All the symptoms well marked. Is much depressed, and thinks she will be lost eternally.

4. Mrs. B., 47.—For several months has collapsed in strength. Palpitation on slight exertion. Tremor, especially on any voluntary motion. Thyroid much enlarged ; intense thrills in it, and loud bruit all over it. No exophthalmos.

5. Mrs. T.—Ill six years. Goître. Palpitation. No exophthalmos.

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CHAPTER VI.

HEADACHE—HEMICRANIA.

PATHOLOGY — Headache

LEAVING the subject of hemicrania for later consideration, the symptoms of headache have so much to do with the condition of the circulation, that it naturally must be mentioned when diseases or disorders of the sympathetic are spoken of. No classification of headaches is entirely satisfactory. It is difficult to avoid what are logically cross-divisions in their arrangement. Still it is feasible to speak of—

1. Hyperæmic headache, active and passive.
2. Anæmic headache.
3. Neurasthenic headache.
4. Rheumatic headache.
5. Syphilitic headache.
6. Toxic headache.
7. Sympathetic.
8. Hysterical. It is in these two latter divisions that the classification is apt to intrude upon former varieties.

1. The pain in the head resulting from active hyperæmia is accompanied with flushing of the face and redness of the conjunctiva with pulsations of the

arteries, especially of the carotids, with dizziness, muscæ volitantes and tinnitus, with aggravation of the pain on stooping or bowing down the head Alcoholic stimulants, excessive use of tea and coffee, inordinate sexual excitement, may induce it. It is closely connected with the angio-paralytic variety of hemicrania, only that it is not confined to one set of vessels only. It is often met with after sexual excitement without satisfaction, a kind of 'mental onanism.' The frequent repetition of the hyperæmic condition leads to an habitual dilatation of the arteries of the head. The pupils in this variety are generally constricted. Headache from passive hyperæmia is often associated with a cyanotic discolouration of the face and ears, and is met with in many of those affections in which a partial venous stasis in the head is a marked symptom, as in certain diseases of the heart, in tumours of the neck pressing upon the jugular vein, &c.

It seems at least doubtful whether headache depends upon any vascular changes in the brain itself, except in pressure on the nucleus of the fifth nerve.

2. The anæmic headache is constantly met with in practice. In loss of blood at parturition or after operation, or after profuse fluxes of various kinds, diarrhœa, hyper-lactation, or in a more chronic form after prolonged residence in a damp district, whether malarial or otherwise, the well-known phenomena of anæmia make their appearance, with dragging pain in the head, sometimes accompanied by the sensation

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of hammering within the skull, and with vertigo and tinnitus. The character of the headache is more like that of active than of passive hyperæmia.

3. Here again in neurasthenic headache the difficulty of classification appears. It might be well to speak of this variety as a form of the passive hyperæmic headache. Long continued brain-work, especially such as is characterised by monotonous sameness, anxiety over a sick bed and prolonged night-watching, depressing emotions or venereal excess, all such causes induce not only a condition of nervous debility, but a peculiarly intense form of headache. Hypochondriacal feelings are often coincident with this variety. Frequently the vessels of the head are hyperæmic, apparently from paresis of the vasomotors. It is a form that is especially benefited by brain food, such as fatty and farinaceous materials, by guarana, the salts of zinc, strychnine, ergot, and the bromides.

4. The rheumatic headache is met with after chilling the sweating scalp or the nape of the neck, but it may occur under very various conditions—simply as the effect of a chill, and accompanied by stiffness, the pain being intensified on any movement of the occipito-frontalis muscle, and often associated with muscular rheumatism in other parts of the body. This form is benefited by external warmth, and scarcely needs internal remedies at all, though in some cases either alkalies or the salicylate of soda may be usefully exhibited. Or headache shows itself as a symptom accompanying acute rheumatism, and due

sometimes to the pyrexia, sometimes to the reflex effect of pericarditis, and then frequently associated with acute delirium. Or again, in a very intense form, more often seen in gout than in rheumatism, with considerable hyperæmia of conjunctiva and flushing of face, without heart disease, without paralysis, without convulsion, but not seldom ending in coma and death, the lesion being either pachymeningitis or arachnitis.

The headache of gouty people is usually frontal, and is often associated with vertigo, but the form just mentioned seems to implicate the whole convexity of the head.

5. The syphilitic headache may often be a warning of syphilitic affection of brain, but in secondary syphilis it may mean periosteal affection of the skull-cap, or it may exist without any coarse lesion, and accompanied with some swelling of the cervical glands. The greater intensity by night is often characteristic, but, if it is not treated, the intervals become smaller and smaller, no less by day than by night, leading even to delirium and mania.

6. The toxic headache may depend on the circulation of poisonous substances in the blood, or upon the effects of these poisons on other organs. Some people suffer thus from alcohol, from chloroform, carbonic oxide, sulphuretted hydrogen, and even under the poisonous effects of lead. Very many complain of headache after opiates. It is occasionally a symptom of uræmia.

7. The sympathetic headache may depend entirely on a morbid condition of the vasomotors of the

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membranes, due to an excitability or a paresis of some vasomotor centre, but most usually it is the result of reflex action, and depends on the original irritation of a full stomach, of indigestion in its various forms, on the pressure of parasites in the alimentary canal, or the irritation of hemorrhoids. In much the larger number of cases the irritation has its starting-point in the stomach or intestines. But there is no reason against lesion in any organ being the origin of this sympathetic headache. Uterine lesions often are.

8. The hysterical headache can often scarcely be separated from the sympathetic. It is frequently intense and very real. It may be hyperæmic, or anæmic, or set up by reflex irritation, or be symptomatic of brain-starvation from insufficiency of brain-food, either in quality or quantity. With many it is associated with much photophobia, with tinnitus and vertigo.

It is a true neurosis depending on abnormal conditions of the arteries. It is not unusually accompanied by neuralgia of the fifth nerve, and may be effectively treated by guarana, gelseminum and arsenic.

In many hysterical patients, in whom headache is a prominent symptom, quinine is not well borne.

The association of the sympathetic with these headaches is mainly vasomotor.

Of all forms of headache, the most difficult to explain is the one that attacks accurately one side of the head only, hemicrania. It is a constitutional anomaly. If it occurs after twenty-five years of age, it often

warns of the gradual approach of general paralysis, or of some other severe cerebral lesion.

Does the study of hemicrania show definite abnormality of the sympathetic? The peculiarity of pain referred to only one side of the head is associated with very opposite vascular conditions. Du Bois Raymond thought the symptoms were due to a tonic narrowing of the vessels of the head. Müllendorf, on the contrary, thought they were due to a paralytic dilatation of these vessels. Both were right from a certain point of view. The difficulty of explanation is increased by the fact that the two forms may exist one after another in the same individual during the same attack, or may alternate in different attacks.

Seeligmüller says 'the nature of the disease is the pain.'

1. In tonic or spastic hemicrania, the prevailing phenomenon is tetanus of the muscular coat of the arteries on the affected side of the head, in the region supplied from the cervical portion of the sympathetic nerve. That this is the condition of the vessels is shown by the hard cord-like temporal artery, the pale and sunken face, the small eye, the diminished temperature. The pupil is dilated during the height of the attack from increase in the tonic excitation of the dilator fibres, which arise from the cilio-spinal centre, and follow the course of the cervical sympathetic. The extreme action of these fibres overcomes abnormally the contracting power on the iris of the ocular-motor nerve. The subsequent contrac-

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tion depends on a secondary diminution in innervation, akin to fatigue, corresponding to the condition of the vasomotor fibres. Thus, too, towards the end of the attack, the ear becomes red and warm, and the conjunctiva, from relaxation of its vessels following the tonic spasm, becomes injected. Increase of saliva, with a certain tenacity of the secretion, is met with in this form. Eulenberg and Guttman consider that the pain is caused by tonic spasm of the unstriped muscles of the vessels (as in cramp and colic); that is, from pressure on nerves of sensation distributed within the muscular tissue. But it is not quite certain which branches of the fifth nerve are affected by the pain. Some localise the pain in the frontal branches, but it is more probable that the branches implicated are those which supply the dura mater, coming from all three divisions of the fifth nerve.

2. The other form, hemicrania-neuro or angio-paralytica, is characterised by relaxation of vessels, arterial hyperæmia, and increase of temperature on the affected side. The state of this side of the head and face is not unlike that of an animal in which the cervical sympathetic has been cut.

But the explanation of the pain in this variety is not straightforward. It is said to be due to irritation or compression of the nerve elements by the temporary increase of the blood pressure, and the greater quantity of blood in the small arteries and veins. But pain is not usually felt from congestion, at least pain of the acute intensity of hemicrania. And although in the later stages of the spastic form, when

the vascular spasm gives place to dilatation, the paretic congestiveness does not perhaps equal the marked hyperæmia of hemicrania angio-paralytica, yet it is remarkable that in these later stages of the spastic form the pain diminishes *pari passu* with the spasm, whilst in fact the vessels are getting more or less into the same condition that is said to cause the pain in the paralytic form. The symptoms, however, of each form of this affection, especially the oculo-pupillary phenomena, point to the cervical sympathetic, or to the corresponding half of the cilio-spinal region of the cord as the part specially implicated, and the success of caffeine, quinine, guarana and ergot in the paralytic variety, and of nitrite of amyl, carbonic acid inhalation, hot drinks, &c., in the spastic, points to the same conclusion.

The frequent occurrence of nausea, vomiting, muscæ volitantes, tinnitus, and foul taste, are all points directly or indirectly associated with the sympathetic; and not only is tenderness met with on pressure over the last cervical and four first dorsal vertebræ, but deep pressure over the cervical ganglia excites pain.

Dr. Latham's dictum is good for the spastic form, that the disease is characterised by a morbid activity of the sympathetic nerve, in consequence of a defective control or inhibition by an exhausted or enfeebled cerebro-spinal system.

In hemicrania alternans, when the patient is affected sometimes by one form, sometimes by the other, the law of paretic fatigue, following inordinate

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action, may afford the explanation of the paralytic form. In other cases the paralytic form may owe its causation to depressing influences affecting the whole system, and especially the nervous centres, such influences being particularly those of climate, of mental strain, or of venereal excess.

The neurotic origin of hemicrania is rendered more probable by the fact that it is sometimes congenital, or at any rate hereditary. The pain, though unilateral, is not always strictly so, nor is it so intense as neuralgia. When confined to one side, it does not affect the whole of that side of the head equally. There is generally a special parietal point of pain. On the affected side there may be a special acuteness of the sense of touch.

Some observers, even at the present day, look upon hemicrania as a pure neuralgia of the fifth nerve, independent of the abnormal vascular condition. With reference to this, it must be said that in hemicrania the branches of the trigeminus that go to the cerebral dura mater are the parts affected, and that these arise from all the three divisions of that nerve, viz. 1, the nervi tentorii of Arnold from the first division; 2, a branch running with the arteria meningea media from the second division; and 3, the nervus spinosus of Luselda from the third.

It is somewhat peculiar that certain branches of each of the three divisions of the trigeminus should be thus affected, without further implication of any other branch from either division of the fifth. It would be unusual even if the lesion were one of the

nucleus of the fifth itself. It is pretty certain that lesion of a nucleus of the fifth may induce very intense neuralgia. In a case lately seen, a gentleman aged seventy-eight, with very advanced arcus senilis and diseased vessels, was the subject of most aggravated neuralgia of the fifth nerve on the right side of the face, that from its mode of coming on, the conditions (nutritive and other) of its origin, and the best methods of relief, seemed to depend on defective innervation of the fifth nucleus in the floor of the fourth ventricle. The pain, or the lesion that induced the pain, set up a reflex influence on the facial nucleus of the same side, causing a most intense spasm of the facial muscles. It cannot therefore be said absolutely that the neuralgic theory of hemicrania cannot be entertained. But to call it a neuralgia does not explain the phenomena. The most violent neuralgia need never be associated with a spastic, or with a paralytic condition of the vessels, such as is seen in hemicrania.

Still less does the neuralgic theory explain those cases of hemicrania alternans that are not very uncommon. Opposed to this is the sympathetic theory above mentioned, by which the origin of the phenomena is placed either in the cervical sympathetic or in the bulbo-spinal vasomotor centres. It is evident enough that such a theory accounts for both the tetanus of the muscular fibres of the vessels of the head, and also for their paralytic condition. It explains the oculo-pupillary phenomena, the increased quantity and altered quality of the saliva, the frequent connection of migraine with epilepsy,

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the paralytic state of the vessels of the choroid and retina in the paralytic form, the fact that the phenomena may be set up reflexly by irritation from distant organs, especially from the abdominal and pelvic viscera so largely supplied from the sympathetic system, and irritation from which is mainly carried by the sympathetic. But neither theory adequately explains the pain, nor the slow pulse which is often found. How is it that section of the cervical sympathetic, followed as it is by paralysis of the vaso-constrictors and dilatation of the vessels of the face and head, the eye, and the ear, is not accompanied by any pain whatsoever? If pressure of dilated vessels on sensory nerves induces the pain in the paralytic form of hemicrania, why does not experimental section of the cervical sympathetic own a similar state of pain as a necessary sequence? It is probable enough that in the spastic form the pain may be caused by tonic spasm of the unstriped muscles of the vessels, that is, from pressure on nerves of sensation distributed within the muscular tissue; but that the acute pain of hemicrania results in the paralytic form from simple congestion seems impossible.

Judging therefore from the associated phenomena in this latter form of hemicrania, it seems reasonable to believe that the same lesion that antagonises in the medulla oblongata the action of the vaso-constrictors of the head, irritates also the nucleus of the trigeminus and excites the nucleus of the vagus. The slowness of the heart's action proves conclusively that the inhibitory action of the vagus is in the ascendant. The

same lesion (itself possibly one of the circulation only) may easily irritate the nucleus of the trigeminus or some of its cells, and thereby cause localised pain. But as in this confessedly difficult neurosis all theories fail somewhat in giving an adequate explanation, so this one is difficult thus far, viz. that a lesion that irritates the trigeminus and the vagus might be considered as paralysing the vasomotors. Can the same lesion be paretic to one set of nerves and stimulating to others? Some perhaps would allow this to be the case. But it seems more in accordance with physiological action to deny the probability. But the lesion of the vasomotor centre may be irritative throughout, and may act as stimulating vaso-dilator nerves—nerves which usually do so little to antagonise the vaso-constrictors that the very possibility of their existence is even forgotten. Under abnormal conditions of stimulation, however, they may assert their existence and their functional activity, as decidedly as the nervi erigentes of Eckhard, and, if this be granted, the full distended arteries, the pain, and the inhibited action of the heart will all find their origin in an irritative lesion of the medulla oblongata.

If the incurability of a disease may be tested by the vast array of remedies recommended for its treatment, so the obscurity of the causation of an ailment may be gathered by the very various opinions held concerning it. Thus, as to hemicrania, some observers think it the result of gastric disturbance.

Niemeyer and Allbutt consider it is due to hepatic lesion. Hasse calls it a neuralgia of the fifth nerve,

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especially of the branches to the meninges and the cranial bones. Anstie thought it due to irritation (molecular atrophic) of the roots of the fifth. Piorry placed it on the common ground of the fifth nerve and the sympathetic, in dependence on the ophthalmic ganglion, the nervous network of the iris. According to him it is an irisalgia, and he bases his view on the fact that the globe of the eye is the seat of special pain, and that the patient complains of subjective luminous sensations. Taking into consideration the apparent deep seat of the pain, and its association with the nerves of special sense, Romberg and Calmeil look on the disease as a neuralgia of the brain itself, a cerebral hyperæsthesia. Bois Raymond, himself a great sufferer from the spastic form of the disease, considered it a consequence of an exaltation of the cervical sympathetic, with or without a morbid state of the cilio-spinal region of the cord, showing contraction of the temporal artery, pallor of face, dilated pupil, and retraction of the ocular globe. He experienced pain on pressure of the spinous apophyses of the corresponding vertebra. He thought the pain of the disease was caused by a cramplike pressure of the sensory filaments in the muscular walls; and attributed the nausea, the vomiting, the sparks before the eyes, to modification in the intra-cephalic vascular tension. He compared the pain and its causation to the uterine colic during parturition. Müllendorf, on the other hand, finding the retinal vessels dilated, the corresponding side of the head covered with sweat, and that pressure on the carotid

artery lessens the malady, believes that the lesion is a temporary paralysis of the cervical sympathetic. Jaccoud tries to reconcile these two statements by saying that in migraine two periods succeed one another with regularity, one of excitation and the other of paralysis. He believes that the observations of the two preceding authors were taken at different periods of the disease. Latham adopts Jaccoud's views, chiefly on the ground that the initial pallor is succeeded by redness of the cheek and injection of the conjunctiva; coincidently with this the urine becomes very clear and abundant, whilst the saliva becomes gluey and disagreeable, just as is the case after section of the splanchnic nerves and the salivary branches of the sympathetic. He describes also the visual disturbances that precede, in some people, the peri-orbital pain. These consist in a partial obscurity of the field of vision, and in the appearance of a luminous, flickering, sometimes coloured zone, surrounding the point of obscurity. This obscurity seems to indicate that at the commencement of the symptoms there is a partial constriction of the vessels of the retina. The views of Eulenberg and Guttman have been already given. They have sometimes observed dilatation of pupil at the beginning of an attack, and very frequently a constriction of this orifice towards the end. They believe that the tendency of hemicrania to decrease with age is owing to the fact that in advanced life the vessels lose their contractility. Vulpian believes that some of the symptoms are certainly connected with the cervical

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sympathetic, but only when the migraine occupies the frontal and orbital regions, and not even always then. He considers that gastric disturbances have much to do with the causation, but that the mechanism of the affection cannot be explained with precision. Leveing considers the affection to be a true volcanic discharge, by which the nervous system is cleared of an excess of tension. This seems more a mode of formulating an expression of symptoms than of explaining them. He believes that the first impulse to this discharge occurs in the isthmus of the brain.

Hervez de Chégoin combines the idea of nervous plethora with the vascular mechanism, that is to say, he localises this plethora and its manifestations in the sympathetic. But he neglects, or does not admit, the period of the tetanisation of the vessels. He places the pain in the peri-vascular network; and to justify this localisation he shows that the pain of migraine is very different from that of neuralgia of the fifth. The latter is intense, of short duration, intermittent, striking like lightning the anatomical course of the branches of the fifth; whilst the pain of migraine is continuous, and, like a wave, passes over the distribution of the arteries. According to him there is an abdominal crater, that, coincidently with the brain crater, vomits forth the excess of tension that torments the sympathetic.

Poincaré believes that the phenomena of this disease have to do with more departments than one of the nervous system. One thing seems certain, that as regards the state of the vessels of the head,

there are two phases closely related to each other, one of pallor, the other of redness. The first is exceedingly transitory, and may pass unperceived. He agrees that the pain in the first stage is located in the perivascular network, and is engendered by the vascular spasm. In the congestive stage the aspect of the pain changes, taking on the form of neuralgia of the fifth. Probably the fifth is then affected, either by the erethism attacking it in its turn, or by being irritated by the dilatation of vessels, and congestion both of its nucleus and its branches. So the other symptoms fall naturally under the heads of these two stages. At the moment of pallor the patient distinguishes objects with difficulty. A kind of mist separates him from his surroundings. There are numerous lacunæ in the field of vision, a semi-amaurosis due to the retinal anæmia that results from vascular constriction. Sound is perceived vaguely. The poverty of the cerebral circulation serves to explain the sort of intellectual incapacity that manifests itself at the beginning of the malady. During the congestive period it is not muscæ and mist that impede vision. The retina is hyperæsthetic. The patient closes his eyes because the light gives him pain. The congestion of the retina and of the cerebral seat for the perception of visual sensations induces subjective luminous phenomena. So, in the ear, noise is insupportable, and buzzing and tinnitus occur. Intellectual work is active, but fatiguing. In many, nausea and vomiting are met with, without any disturbance of stomach. Poincaré thinks, and

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with reason, that they may be the consequences of changes in the intracranial tension, or of vascular modifications in the brain, of central origin, and resembling those seen in meningitis.

But it is not proved that the cervical portion is the only region of the sympathetic affected. The coronary plexuses of the stomach may be similarly involved; and a spasm of the muscular coat of the stomach, thus originating, may irritate branches of the vagus, causing thereby nausea and vomiting.

Judging by what patients say, it seems probable that coitus may prevent an attack, whilst in continent persons the disease is more frequent and more intense. No doubt, as has been before mentioned, the phenomena may be induced by reflex action from distant organs and from emotion.

Dr. Day says: 'It is important to recognise the fact that migraine is purely neurosal, and not dyspeptic, in its origin. Put aside the liver, and the stomach, and the intestines as the origin of evil, and seek its explanation in some excitement or other alteration of the cerebral ganglia, for it is essentially cerebral. All successful treatment must be based on this understanding. The intimacy between nervous and neuralgic headache is so close, that we have however to remember that nervous headache, which may be entirely frontal for years, does frequently become, with the lapse of time, trigeminal or one-sided.'

Other observations tend towards a central origin. Thus Dr. Saundby has reported a case of migraine in a young woman which had occurred at intervals ever

since she was twelve years of age, and had always been accompanied by complete paralysis of the left third nerve. This paralysis passed off in a few days, but there remained a slight degree of permanent ptosis, complete paralysis of the superior rectus, slight dilatation of pupil, and vision = $\frac{1.5}{1}$. The patient had enjoyed otherwise good health, was well developed and well nourished, and had never suffered from fits.

Some very interesting notes on migraine of the eye by Galezowski were translated some years ago by Dr. Stanford Morton. The symptoms which characterised this affection are these:

1. The complaint generally attacks those who have for several years been subject to ordinary migraine; this latter ceases, and is replaced by visual nervous symptoms, which, however, may come on without being preceded by any other nervous symptoms.

2. Migraine of the eye is more frequent in females than in males, and occurs at all ages; but Galezowski has only seen it twice in individuals as young as thirteen or fourteen.

3. The onset of the attack is not always the same. In some cases the ocular migraine is preceded by headache; whilst in others (and these are more frequent) the visual trouble comes on quite suddenly, and is characterised either by hemiopia or central scotoma.

4. The hemiopia is either monocular or binocular. The former is sometimes lateral, and at other times it occupies the upper half of the visual field. In the

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binocular form the field of vision is obscured laterally, either in the right or left half of both eyes; the sight is completely lost on the half of the field of vision; nevertheless, the acuity of vision remains almost normal. This hemiopia is only temporary, lasting from twenty to fifty minutes, and then disappearing completely. Sometimes, however, Galezowski has seen it pass into complete blindness, lasting for a brief period; or again, in other cases, it is followed by a slight indistinctness of vision for the remainder of the day.

5. Central scotoma is more rarely the chief symptom of the malady, which maintains this form throughout; but three times it has been seen to be transformed into hemiopia.

6. Flashes of light and rainbow colour in zigzag forms generally accompany ocular migraine. These phenomena are perceived by the patient in the obscured part of the visual field, becoming gradually more and more indistinct, and finally disappearing altogether. Three of Galezowski's patients said they observed thousands of luminous muscæ and silvery spangles darting about in the darkened field of vision.

7. There may be more or less violent attacks of vomiting preceding or accompanying the migraine of the eye; but these are frequently altogether absent.

8. After the disappearance of all the ocular symptoms, giddiness, more or less intense, comes on, and continues some hours, or even days.

9. The scintillating hemiopia is almost always followed by headache, which continues all the rest of

the day, either in one half or over the whole of the head.

10. The eyeball is often painful and tender, the patient experiencing a sensation of weight and tension at the back of the orbit, and occasionally the eye is red and watery.

11. Periodic hemiopia generally recurs at long intervals, once or twice a year only; but in certain cases it is more frequent, and may recur as often as once a month, once a week, or even sometimes twice or thrice a week. In these latter cases there supervenes a disturbance of sight and a sort of asthenopia, which is almost permanent, and renders all work impossible.

12. Ocular migraine is often observed in those troubled by dyspepsia, but this last symptom is not constant: when it exists, it almost always indicates the presence of gout, which, as Troupeau and Charcot have well demonstrated, often predisposes to migraine.

13. Periodic hemiopia is observed sometimes in pregnant women, but then it is accompanied neither by scintillations nor by headache. On the contrary, in pregnant women one sees cerebral troubles characterised by a sort of aphasia, and by encephalopathy, which may continue for half an hour or longer.

14. Analogous cerebral derangements may also be found in migraine of the eye, but they are relatively rarer and of shorter duration than in the preceding case.

15. Ocular migraine does not present any gravity,

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and disappears of itself under the influence of a tonic strengthening regimen. The sulphate and bromohydrate of quinine have seemed to Galezowski speedily to relieve the symptoms. The employment of ferruginous preparations, the cold douche, and avoidance of all exciting aliments, such as coffee, liqueurs, spices, and such like, may also act efficaciously in accelerating the cure of migraine of the eye.

Dr. Simpson Craig strongly recommends the strong resinous tincture of the gelseminum sempervivens.

With reference to the treatment of migraine in general, it has been already stated that the treatment of the two forms is diametrically opposed. In the spasmodic, the sympathico-tonic form, whatever tends to diminish the flow of blood to the painful region, as, for instance, compression of the carotid on the affected side, increases the intensity of the symptoms; while the pain and other morbid phenomena are lessened by anything that increases the quantity of intracranial blood, as by pressure of the opposite carotid, causing a diversion of blood through the contracted artery. It is partly on the fact that the object of treatment in this variety is to relax muscular spasm, that the claim of this disease to be in great measure a disease of the sympathetic rests. To this end, Hammond, among others, speaks of the value of galvanism, one pole being applied over the cervical sympathetic and the other over the solar plexus, and a current from about fifteen cells being allowed to pass for two or three minutes. Still better is the administration of a

quarter of a grain of morphia hypodermically, followed by a drop of a 1 per cent. solution of nitro-glycerine every fifteen minutes in pill or on sugar. Specific etiological complications require special modifications of this treatment; thus, if the migraine own a malarial origin, a large dose of sulphate of quinine should be given instead of the morphia. During the intervals, the bromides should be administered persistently for months; and during the same period, glonoin (one drop of the 1 per cent. solution three times a day). In the paralytic form, compression of the carotid on the affected side lessens the pain, and whatever tends to increase the amount of intracranial blood intensifies all the symptoms. Cold to the nape of the neck is useful. Thirty or forty grains of guarana, or ten grains of caffeine, or even strong tea or coffee, will often cut short a seizure; but better still is a large dose—a hundred grains—of sodic bromide. In these doses the bromides give tone to the vessels whose coats are relaxed.

Treatment during the intervals should consist of the bromides, ergot and strychnia. If the pressure of undigested food in the stomach or intestines should be acting either as a cause or a complication, an emetic or a purgative may be necessary.

Dr. Hammond speaks not only of the two forms alternating in the same person, but of their being present coincidently, one side of the head suffering from the tetanic form, the other from the paralytic. This, however, is exceedingly rare.

It seems therefore, from the effects of remedies as

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well as from a study of the phenomena of the disease and of its occasional complication, that some portion of the sympathetic is primarily affected; probably not the cervical ganglia themselves, but a portion of the vasomotor centre in the medulla oblongata, acting by and through both the sympathetic centres in the cervical cord and the sympathetic cervical ganglia, but that over and above this seat of lesion the nucleus of the vagus is generally implicated, and the nuclei of other nerves, pre-eminently the fifth, are likewise involved. The disease is central in its origin, though mainly of a sympathetic centre; and the state of central circulation, of which often the lesion consists, may be caused by reflex irritation from distant organs.

Consult—

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CHAPTER VII.

INSOMNIA.

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THAT many kinds of insomnia depend upon changes in the vascularisation of the brain is simply a matter of daily experience. It is very generally accepted that sleep is due to a decrease in the size of the cerebral arteries, inducing a partial anæmia of the brain. Chapin relates an interesting experiment tried by himself with reference to this question: 'Hearing it suggested,' he says, 'that if sleep is dependent on cerebral anæmia, amyl nitrite, which promotes the circulation in the brain, should waken sleeping persons, I tried it one evening. With the aid of my nurse I very carefully applied a few drops of the drug to the nostrils of ten or twelve patients who were sound asleep, and in every case they awoke in less than one, or at most two, minutes. This was repeated on several evenings and on different patients, but with a uniform result. Lest it might be simply the odour of the drug or its irritation of the fifth nerve, or my presence near the bed, I tried, on other occasions, bisulphide of carbon and oil of peppermint, but succeeded in awakening not a third of those on whom they were employed. Hence we conclude that sleep can be induced, directly or reflexly, through the sympathetic

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nerve, which has under its control, partially at least, the vessels of the encephalon.'

Vulpian objects to the experiments of Durham and of Hammond that they were made under the influence of anæsthetics, and therefore that the sleep was not a natural one. To Fleming's experiments, in which sleep was induced by compression of the carotids, he objects that the result was not sleep, but the loss of consciousness not unlike that seen in plugging the cerebral arteries. Other authors, like Cappie, explain sleep as caused by venous congestion and pressure. One fact rather militates against Durham's views, viz. that in anæmic individuals, whether the anæmia be caused by hæmorrhage by chlorosis or by cachexia, insomnia is a prominent phenomenon instead of somnolence; whilst in plethora, or after excess in eating or drinking, the somnolence is accompanied with suffusion of the face and injection of the conjunctiva, and presumably, therefore, with congestion of the brain. Experiments, too, on the lower animals, by compression of the carotids and vertebrals, do not result in causing sleep, although necessarily the brain must be in a state of anæmia. Experimental faradisation of the central sympathetic on both sides, though leading to constriction of cranial and intracranial vessels, causes no tendency to sleep.

Brown-Séquard states as his opinion that in many nonepileptic persons sleep resembles a slight attack of epilepsy. In speaking hereafter of epilepsy something may be said on this point. But the French savant goes on to say that whatever be the nature of sleep,

it is certain that it is in part a condition of sub-asphyxia, and in this relation the daily loss of consciousness resembles an attack of epilepsy.

Vulpian believes that the state of the circulation has little to do with the causation of sleep. His expression is as follows: 'Pour ne parler que des animaux, il est beaucoup plus probable que ce sont les éléments anatomiques eux-mêmes; ceux, par exemple, de la substance grise de certaines parts de l'encéphale, chez les animaux supérieurs, qui s'engourdissent pour ainsi dire, et cela primitivement d'une façon indépendante.' He thinks that the vascular and cardiac modifications are only accessory, and play no essential part in the physiology of sleep. His idea seems to be, that at the early commencement of sleep the vessels of the brain are congested, but that by and by the respiration becomes calmer and slower, the heart beats with less energy, and thus the cerebral circulation is somewhat modified, the early congestion giving place to slight anæmia; that, from the fact of the relative repose of the cerebrospinal centres, the vasomotor system becomes slightly predominant, and as a consequence of this there is a feeble increase in tone of the various vessels of the body, and so amongst others of the brain; but that the vasomotors are certainly not to be credited with any share in the production of sleep.

In Vulpian's experiments too with anæsthetics the vascular changes in the brain have been slight. The pallor induced in the vessels of the cerebral pia mater under chloroform and under ether was very slight,

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under opium nil. Under the influence of this drug, or of chloral, there was seen no modification of the pia mater circulation.

'The salient feature of sleep is the cessation of the automatic activity of the brain; it is the diastole of the cerebral beat,' as Mr. Foster says.

'That sleep is a slackening of molecular activities, especially of the brain, is an expression of a fact. That this depends on exhaustion is probable, but is an opinion only. It is, however, analogous to what is found very largely in the animal and the vegetable world, a period of activity using up more or less the force capable of being generated, and succeeded by periods of inactivity in cycles. It cannot be that this period of inactivity is caused by the accumulation in the protoplasm of the brain of the products of protoplasmic activity, that the presence in the cerebral tissue of an excess of the products of nervous metabolism is the cause of sleep.' If so, why should the period of sleep ever terminate? Michael Foster thinks that more may be said in favour of the conception that during the waking hours the expenditure of oxygen exceeds the income, and that the quiescence, which we call sleep, comes from the exhaustion of the body's store of intra-molecular oxygen. But taking the general results of experiments and of pathological observation into consideration, it must be concluded that in sleep the brain is more anæmic than in the waking hours, even if this anæmia is an accompaniment rather than a cause of sleep. The very fact that to induce sleep we should withdraw the brain as

much as possible from the influence of all extrinsic stimuli is a proof of this, since any stimulus of sight or sound, for instance, at once necessitates for its perception a condition of centre that is the opposite of anæmia. There is an interesting case on record of a lad, whose connection with the external world was, from a complicated anæsthesia, limited to that afforded by a single eye and a single ear, and who could be sent to sleep at will by closing the eye and stopping the ear.

But the ordinary experience of the laity even would tend to show the same thing. The somnolent effects of digestion, in which the alimentary canal makes so great a demand on the circulation; the good results of inducing sleep in hard-worked brains of intellectual people, in whom insomnia is so frequent, by insisting on their taking a small meal at midnight; the advantages of a warm bath before going to bed, or at least a prolonged foot-bath; the almost certain success of a long narrow sinapism down the whole length of the spine, and the occasional benefit of remedies that cause an intestinal flux—all point to the same conclusion, that when the brain is anæmic, molecular activity is difficult; molecular inactivity, sleep, is more probable. The somnolence of old age, with atheromatous cerebral arteries, shows the same thing.

Do the vasomotors ever sleep? In that cycle that brings periodical inactivity to the rest of the body, is there any evidence that this system of nerves partakes in this inactivity? Digestion proceeds during sleep, and secretion goes on in all

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glands: the heart acts, though not so strongly as during waking time; there is little, if any, passive arterial dilatation to be recognised anywhere throughout the body. There is nothing, therefore, strange in believing that the sympathetic may be in full activity whilst the brain is asleep; the strangeness, indeed, would be all the other way. If the vasomotors were paretic during sleep, there would be some arterial dilatation. No observer has seen this. Most physiologists speak of the opposite condition of the cerebral vessels. And even if sleep is not caused primarily by anæmia of brain, there seems no doubt that the duration of molecular inactivity may be at least prolonged by a constricted state of the arteries of the brain. Dr. Mortimer Granville's dictum, that 'general sleep is the aggregate of independent, though normally correlated, sleeps, induced in various parts of the system,' may be accepted as true in the main. But one may join issue with him as to visceral sleep. How is it that a rectum, empty on going to sleep, is full in the morning? that the urinary secretion is going on through the deepest sleep? that the sense of epigastric weight from a heavy meal overnight has passed away on awakening? that in eruptive diseases a skin clear in the evening may be covered with rash in the morning, to say nothing of the points referred to above? Do not all these point to the fact that vasomotor and sympathetic action persists when the rest of the body is asleep? It has been truly said, that a very common cause of insomnia in certain of its most troublesome forms, namely, those accompanied

with mental restlessness and worry, is such vasomotor disturbance or debility (it may be either or both) as prevents the conversion of the jactitatory or pulsating current of the blood into a continuous and steady flow before it reaches the capillaries. When this state of affairs exists, relief may occasionally be obtained from a moderate use of some stimulant in the form of a 'nightcap.' But this is a mere expedient for the service of the moment, and does nothing towards permanent cure. The rational remedy for this form of insomnia is undoubtedly a tonic treatment, acting as directly as possible on the vasomotor centre or system. Sedatives do harm. In other words, help the vasomotor system from a state of partial inactivity to one of renewed activity, and you procure sleep. Such a statement—and it is one that all medical men must be constantly verifying—is incompatible with the view that this system is at rest when the brain and muscles are asleep.

Suppose that some degree short of perfect rest have been attained, and that the patient sleeps, but with constant terrifying dreams. Perhaps he may wake with great palpitation of the heart; or after a restless, tossing night may get up with a feeling of exhaustion. Surely this means that molecular inactivity has ushered in the first going to sleep, but that from some one of many causes, especially from long-continued thought, or study, or anxiety, the vessels had become so paretic, so dilatable, so imperfectly under the influence of the vaso-constrictors, that an influx of blood to the brain occurs, too large for true

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rest, and brain activity results, uncontrolled by reason but largely under the influence of emotion.

A convenient division of types of insomnia has been made by Dr. Sawyer, of Birmingham, into psychic, toxic, and senile. He holds that in natural sleep the brain is relatively anæmic, but that there are causes of insomnia which act primarily in sustaining cerebral activity, and with it, and in consequence of it, relative cerebral hyperæmia; whilst any agent which sustains cerebral hyperæmia, or any morbid condition which impairs the contractility of the cerebral arteries, may prevent wholly or in part the occurrence of such a degree of cerebral anæmia as is required for the production of sleep. In such a complex condition as conscious cerebral activity, where thought implies increased blood-flow, and increased blood-flow implies thought, we cannot in any given case allow, with strict accuracy, entire causal precedence to either of the factors which are essential to the common result. In some cases of sleeplessness, as in the psychic group, undue cerebral activity is the primary vice; in others, as in the toxic and senile varieties, relative cerebral hyperæmia is the initial error, and cerebral activity its direct consequence. This form may come on from sudden shock, or from long-continued mental strain, the same part of the brain having been kept continuously upon the rack. But in either case sleeplessness did not occur until there arose from exhaustion partial or complete vasomotor paralysis of the intracranial vessels, until the arteries of the brain, worn out by a sustained erethism,

could no longer, even when the brain most needed it, find the force for that contraction of their calibre without which sleep is impossible. It is a kind of insomnia in which unnatural excitation of the cerebral cells is the initial fault.

'In toxic insomnia a poisonous agent maintains cerebral vascularity at such a height that conscious cerebral activity, that is, "wakefulness," is an inevitable consequence. Tea and coffee, and perhaps especially excess of tobacco, are frequent causes of toxic insomnia. Tobacco particularly, if pushed to undue limits, will induce cerebral vasomotor debility or paralysis, and as a consequence persistent conscious thought. In the insomnia of delirium tremens, and still more in the imperfect, broken, unrestful sleep of the subjects of chronic alcoholism, the lesser arteries of the brain are paralysed by alcohol, and sleepless cerebral activity is the inevitable consequence. Another form of toxic insomnia is seen in gouty persons, in cases of chronic albuminuria, in persons in whom there is found a pulse of high tension, or the direct physical evidences of the characteristic cardiac hypertrophy which accompanies chronic interstitial nephritis. Insomnia in such cases is due to the maintenance of a state of high tension in the cerebral arteries. Again, there is the senile form of insomnia. The sleeplessness from which many old persons suffer is mainly, if not solely, the result of senile degeneration of the smaller cerebral arteries. Those vessels are less elastic and less contractile than in health, and their weakened walls

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often lead to their permanent dilatation. They are physically unable to adapt themselves fully to the condition of relative arterial anæmia which is requisite for healthy sleep. The tendency of this condition of the blood-vessels of the brain to prevent or to diminish sleep is probably, to a great extent, counteracted by the cardiac feebleness which so frequently and so fortunately coexists with the vascular changes.'

In thus calling special attention to Dr. Sawyer's excellent lectures on insomnia, it must be remarked that the position of cerebral hyperæmia in the causation of sleeplessness is all important in the toxic and senile forms, whilst in the psychic it takes a less prominent part. In this variety, as has been mentioned above, a general tonic treatment is more beneficial than a sedative. A change of work and of scene, good food, exercise, absence from books, ergot, digitalis, and in some cases a little alcohol, will be better than the use even of chloral and the bromides. General tonics may be advisable; opium never, or scarcely ever. The concentration of attention upon some simple act or thing may induce sleep by exhaustion of this faculty. The good effects of cold to the body in many cases of psychic insomnia depend probably on the reflex effect causing constriction of the cerebral arteries. The treatment of the toxic variety is a matter of common sense, when the insomnia depends on the introduction from without of toxic substances. Diminish or give up the poison, and gradually the effects of the poison on the cerebral

arteries will vanish. But it is otherwise when a gouty constitution or the effects of renal mischief have to be taken into account. It may suffice to say that all methods of diminishing arterial tension are to be employed. In gouty insomnia (excluding, of course, the sleeplessness due only to pain) opium is counterindicated, or at least should be withheld until the high arterial tension has been overcome.

In the senile form the cause lies so much in the domain of pathological histology, and the diseased cerebral arteries are in most cases so far from being amenable to treatment, that drugs are often useless. Still, even here, ergot will sometimes do good, and chlorodyne and the milder sedatives may be given. The important point is to avoid much stimulant or excess of food, or hurry—anything, in a word, that stimulates the heart—always remembering that the safety of such patients, and even their measure of sleep, largely depend on a quiet, equable, unexcited circulation.

From the whole consideration, therefore, of the causation of sleep, and of the various forms of insomnia, it must be admitted that if in one variety the vasomotor system takes a secondary place, yet in all it is exceedingly important, and in some forms primarily so.

Consult—
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CHAPTER VIII.

EPILEPSY

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In epilepsy much may be said about vasomotor influence. Meynert believes that in epilepsy the hippocampus major is a vasomotor centre, irritation of which causes spasm of vessels, and so epileptic convulsions. Nothnagel considers vascular cramp an essential factor in all epileptic seizures. Binswanger says that in a typical fit, excitement of the convulsive centre and of the vasomotor centre are co-ordinated. If the excitement of the vasomotor centre exist alone, there is 'le petit mal'; if excitement of the convulsive centre exist alone, we have those rare cases of motor epilepsy, convulsion without loss of consciousness. Most frequently the centres are excited together.

The influence of emotion, especially the terror at the sight of another person in an epileptic fit, seems so prominent in this disease, that this alone points to a vasomotor basis.

The three views most in vogue as to an epileptic fit are these:

1. Epilepsy is simply a disease of the vasomotor centre in the medulla oblongata, setting up vasomotor spasms affecting particular arteries, and thus causing local cerebral anæmia, which induces the discharge from the hemispheres. This theory is held by few.

2. Convulsion depends on a discharge of motor or convulsive centres in the medulla oblongata, while loss of consciousness is the result of arterial spasm in the hemispheres, due to the action of the vasomotor centre in the medulla.

3. The view of Dr. Hughlings Jackson, that the local discharge in the brain excites at the spot arterial contraction, and thus determines the spread of the discharge.

To this Dr. Gowers objects that the pallor of the face is often absent; that, when present, it is no proof of anæmia of the brain, but is probably due to a reflex contraction of peripheral vessels excited by the discharge in the brain; and that convulsion is not usual in cardiac syncope: that this third view is not needed, and is opposed to the fact, proved by experiment, that functional debility causes reflex dilatation, and not contraction of vessels. He would say that the phenomena of epilepsy depend on instability of resistance, rather than on any primary change in the energy-producing action of the cells. It seems open to question whether this somewhat negative theory suffices to explain all the various forms of epilepsy; whether, particularly, it demonstrates the mechanism of 'le petit mal.' On the other hand, Echeverria has recorded twenty-six cases of epilepsy, in almost all of which there was found sclerosis, or fatty degeneration, or amyloid degeneration, or pigmental infiltration of the cervical sympathetic, and often two or more of these changes united; sometimes also a similar condition of the solar and

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other abdominal plexuses. The writer, however, has frequently found such sympathetic lesions without epilepsy.

Again, although the extraordinary high temperature in the status epilepticus is not a proof of the implication of the sympathetic, and may be caused merely by paralysis of the inhibitory heat centre in the brain, yet this great heat can hardly arise without some vasomotor change, even if this very paralysis of heat inhibition be not caused by anæmia of the centre from reflex contraction of its vessels.

The numerous instances of true epilepsy, caused by reflex irritation from distant organs travelling upward by way of the sympathetic, are not wholly explicable by the theory of instability of cells. The epileptic condition consequent on irritation of the uterus and its appendages, the gastric epilepsy in men, of which Pommay speaks as answering to uterine epilepsy in women, are only some of the examples of this reflex condition.

The difficulties are the greater, inasmuch as all theories must be more or less hypothetical. It seems likely that the convulsive centre in the medulla is a minute corpus striatum for collecting, modifying, radiating convulsive motor phenomena from the cerebral motor area: that epilepsy with convulsion may depend on direct or reflex irritation of this centre, but far more frequently on some condition of cells in the cerebral motor area that may well be termed 'instability;' that these lesions and their consequent phenomena may be wholly independent

of vasomotor disturbance, but that loss of consciousness, occurring either as an early symptom of the convulsive form of epilepsy, or as an independent phenomenon in 'le petit mal,' owns as its cause anæmia of a portion of the non-motor area of the brain, an anæmia depending on vasomotor irritation.

Brown-Séquard's experiments, showing that compression of the cervical sympathetic was a valuable means against 'le petit mal,' points to the truth of this latter proposition.

Benedikt believed that the epileptic attack is caused by a sudden spasm of the vessels, and presents the most complete analogy to neuralgic attacks; only that the irritation affects chiefly the vasomotor nerves, and so leads directly to anæmia of the brain.

In 112 cases noted some years ago by the writer, a definite aura could only be traced in ten. Even in what used to be called 'eccentric epilepsy' an aura is not a necessary phenomenon. The large number of attacks that depend upon intestinal irritation occur without any aura, just as in convulsions due to the irritation of teething; and probably the majority of afferent nerves are not sensory; but the difficulty of finding the starting-point in many cases of convulsions, joined with the fact that even a sensory starting-point may be unheeded by the patient until his attention is called to it by another person after close investigation, would be sufficient evidence against the term 'eccentric epilepsy'; whilst in many cases, and notably in such as depend upon venereal excess or self-abuse, it is often an open question as to

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whether the fit owes its origin to an eccentric physical exciting cause, to a moral cause, which also may be eccentric, or simply to the circulation of vitiated blood in unstable nervous centres. And yet, although a patient may seldom be aware of any sensory affection of a nerve amounting to an aura, and the physician may constantly be unable to detect any exciting cause from the periphery, or connected with any internal viscus, yet a large number of patients give an account of some sensation that warns them of the approach of a fit.

This warning differs from an aura, and seems often to be the commencement of the fit before the loss of consciousness occurs. It takes various forms, according to the portion of the nervous centres first attacked by the arterial spasm. In one case a shaking of all the limbs preceded the unconscious state; in another, a violent tremor of the whole body; in a third, in whom also a distinct aura existed in the shape of pain commencing from the bottom of the bowels and travelling upwards, a similar tremor was invariably noticed for twelve or even for twenty-four hours before the fit; in a fourth this warning occurred in the form of vertigo for a minute or two before the fit. In this case the attack was followed by partial motor paralysis of the right hand, affecting only the flexor muscles; the extensors drew the fingers backwards almost at right angles to the hand. Another, whose attacks seemed at first to depend upon the irritation of ascarides, had warning of the attack half an hour previously by partial

trismus, which prevented her from speaking. Another was both giddy and tremulous. In this case the attempt to read a book would always induce an attack. In two others vertigo almost always gave them notice of the immediate approach of the fit. In two more the warning was by previous headache, and this is very frequently the case, the headache being in such cases anæmic, and caused by arterial spasm rather than by any deficiency in the heart's action, and therefore, although closely connected with the headache from hæmorrhage or excessive lactation, it is not under the control of the same remedies. In another case the warning was described as a great darkness that came over her for a moment or two, before she lost her senses; a sudden and temporary amaurosis, caused by the entire cutting off of the supply of blood from the retina. Another always knew the fit was approaching by heat coming all over the body. In this case the fit first came on after a severe scalp wound, caused by a blow.

Another case opens up different ground. She has no warning herself, but those about her know for twelve hours previously, from her being tiresome. May we not put down this irritability to a want of co-ordination in the various portions of the brain, owing probably to imperfect blood-supply to some parts? In another case the fit was always preceded by giddiness and pain behind the left ear, and was succeeded by partial and temporary paralysis of the left facial muscles.

Another gave the following account of herself.

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Susan L., aged 34, has been married ten years, and has miscarried eleven times. Dark hair, light eyes. Her father suffered from fits. Had no infantile convulsions. No known cause for the fits. She knows when an attack is coming on by feeling giddy and sick and becoming very sallow. Her first fit was when she was twenty-two, her third during her first pregnancy. In the last three years the fits have been more frequent, occurring almost once a month, generally in the night. Does not bite her tongue. The fit is succeeded by headache, drowsiness and vomiting of bile. Bowels confined. She was only three months under observation, at the beginning of which time she miscarried for the twelfth time: but during that short period, under purgatives and bromide of potassium, she had no attacks. This probably was not a case of pure epilepsy, but it is mentioned for the symptoms that preceded the attack.

In another case the fit was invariably preceded by shivering.

In a child, aged 7, who had had fits for twelve months, the first having occurred after a violent fright, the attack was always preceded by six or eight minutes of excessive laughter. This symptom, like the manifestation of bad temper in a former case, is probably due to a want of harmony and control between the emotional and intellectual portions of the brain, caused by partial loss of blood-supply; this loss is due to the commencement of that arterial spasm which, when carried to a farther degree, produces entire loss of consciousness.

In another case, in which the first attack seemed to have been excited by the patient having seen a man in a fit in church, the convulsion was preceded by a great shaking of both arms; and in a boy the fit is always preceded by intense cramp in the left arm.

Most of these warnings are evidently due to local arterial spasm, either of the retinal artery, the anterior and middle cerebral arteries, or of these arteries on one side only, or of the vessels of the medulla oblongata; and the nature of the warning sensations seem to vary according as the loss of blood-supply occurs first in the interior or the posterior portions of the encephalon.

And this leads to another interesting fact in the consideration of the subject. May not epilepsy occur without loss of consciousness, or rather may not phenomena, which we have been accustomed to look upon as epileptic, take place without that loss of intellectual power and of sensation which has generally been considered the main feature of the attack? These conditions differ much from each other, and are met with in all varieties, from convulsions with absolute clearness of intellect to circumstances in which, with many of the cerebral symptoms of the attack, the consciousness is never entirely lost. These latter are the connecting links between this disease and true epilepsy.

Thus Mrs. A. R., aged 28, who has been ill for two years, and has several fits a week, states that in the attack she feels slightly confused and falls,

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but never loses consciousness, has some clonic convulsions of the extremities, but gives no account of tonic spasm. She does not suffer from headache and drowsiness subsequently. Her memory is beginning to be impaired.

A second case was under observation for more than two years, E. P., aged 61, a widow. Every day, and many times a day, for six months before she came under observation, she suffered from a severe convulsive condition. She never loses consciousness in the least, and does not even feel confused; but has most violent clonic convulsion of the mouth, jaws, eyelids, and all the facial muscles. The head is shaken from side to side with intense rapidity; the arms and hands are in a similar state of clonic convulsion. The legs are not always affected, and when they are not, she is able to stand through the whole of the attack. In all respects the convulsions of this case closely simulate epilepsy. She has no tonic spasm, no subsequent headache and drowsiness, no loss of memory or of any mental faculty, no paralysis. She has never had any other kind of fit. The only effect of these attacks is the fatigue consequent on the violence of the convulsions. As these attacks have been often witnessed during a medical visit, it is certain that there is no loss of consciousness. It may be well to state that under the bromides the convulsions have diminished in frequency and intensity, and for some months past have scarcely troubled her at all whilst she has persisted in the remedy. They recur as before whenever she attempts to leave it off.

A third case has its connecting links with the former one and its points of difference.

J. P., aged 22, a coal miner, has been ill two years. Has about eight attacks in the twenty-four hours, in which there is sometimes little convulsions, sometimes a great deal; and in the latter case the attack may last fifteen minutes. He has only lost consciousness five times altogether. In this case memory was deficient; but the man reckoned well, and was sensible enough. Although he improved under treatment, there is no doubt he will become a confirmed epileptic, and that the occasions on which he loses consciousness will increase.

Another case had the first and only fit six weeks before coming under observation. She lost consciousness and was convulsed. Since then she has had several attacks, in which the left hand has been violently convulsed, and in which no loss of consciousness occurred.

Do not all such cases depend simply on the portion of brain affected by arterial spasm? The nervous centres that rule motion may be thus affected, the great motor area on each side of the fissure of Rolando, &c. and the centres of consciousness be quite free, and vice versâ.

This seems to be the case in those numerous instances of 'le petit mal' in which loss of consciousness is the prominent symptom. This form of epilepsy owns many varieties. In some cases it is difficult even for people on the watch for the attack to recognise it in the affected individual. The patient

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may be eating, walking, or occupying himself in any way, and will suddenly lose consciousness for half a minute or so, with scarcely any change in his external appearance, except perhaps that there is a slight vacancy of countenance. Not only is there no convulsion, but there is not even any loss of motive power. He does not fall and he goes on eating, and even walking, as it were, automatically. If he is engaging in conversation he will cease speaking, and will lose the thread of the conversation to some extent; and if he is performing any action that requires considerable co-ordination of muscles, such as writing, painting or playing upon any instrument, he will not continue such movements during the momentary loss of consciousness. Many other acts that demand less conscious combination, such as walking, he will continue to perform, but he will have no recollection of having performed them. The only account he can give of the attack is that he has lost a moment of the day, and that, although he may have felt giddy just before he lost consciousness, his memory is an entire blank as to the attack itself. More commonly, however, the loss of consciousness is accompanied by a sudden paralysis of limbs, and the patient falls. There is, however, no tonic spasm, no clonic convulsion, no subsequent headache or drowsiness. Almost immediately after the patient falls consciousness is regained and the ordinary occupations of life are resumed. This course of things may recur fifty times a day.

Another variety, which may be the connecting

link between this form of epilepsy and the so-called epilepsia gravior, manifests itself by a sudden loss of consciousness and a fall; and these phenomena are immediately followed by some tonic spasm of the glottis and temporary asphyxia consequent thereon. Such cases constantly suffer from headache and sleepiness after the attacks, and are only distinguishable from cases of epilepsia gravior by the absence of clonic convulsion. These cases are closely allied to what nurses call 'inward convulsions' of infants, in which there is probably loss of consciousness and very evident glottidean spasm; and when the attack does not proceed to clonic convulsion, there may often be traced a general tremor that passes over the whole body. If the patient lives, the loss of consciousness is pretty sure sooner or later to be followed by clonic convulsions, and the attacks will become those of convulsive epilepsy; but the slighter attacks may occur alone for a number of years, as in the following case:

Harriet H., aged 19, light hair and eyes, was frightened when she was three years old, and has had attacks of 'le petit mal' ever since. If she is at dinner, she will go on eating during the attack; and if she is walking, she goes on, and does not fall. Has had four severe fits with convulsions (the first when she was fifteen years old), in one of which she has bitten her tongue. This case was brought to the infirmary by the mother, who stated that she was most anxious the girl should be frightened again, as she thought it might cure her; since a younger child,

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a boy, had been frightened at Christmas, and had had similar attacks up to September, and then was frightened again, and had had no fit since.

It is very questionable whether these attacks of epilepsia mitior should not be considered as serious as, or rather more serious than, the form of epilepsy with convulsive phenomena.

In those cases in which the two forms are united, the attacks of 'le petit mal' may occur many times a day, and the convulsive fits only once a day, or once a week, or once a month. It is comparatively common that the non-convulsive form should occur as often as once daily; whilst it is comparatively uncommon for the convulsive form to occur more frequently on the average than once in several days. So great is this difference in the frequency of the phenomena of the two forms, that we are almost tempted to look upon the cerebral congestion of these convulsive fits that are accompanied by laryngeal spasm as absolutely preservative against very frequent repetition of such attacks. It is easy, therefore, to realise that the mind is more likely to become rapidly impaired when its blood-supply is cut off very frequently than when this lesion takes place once in two or three weeks, and seldom once a day. Amidst all the difficulties of the subject, one point alone seems at once established and capable of explanation, viz. that impairment of the mental faculties takes place in direct ratio with the frequency of loss of consciousness.

There is some objection to the view that chronic

convulsions depend on the asphyxia and the accumulation of black blood in the encephalon and spinal cord.

Brown Séquard founded these views upon some experiments in which clonic convulsions were set up by injecting venous blood into the system. It seems, however, more than doubtful whether these experiments bear out his conclusions. For instance, in Kusmaul and Tanner's experiments, the prevention of the entrance of red blood into the brain, when the veins were left perfectly free, induced violent convulsions. Again, in an animal bled to death, and in which, therefore, no accumulation of black blood can have existed, the uterus expelled its contents precisely in the same manner as in an animal into which venous blood has been injected. It would seem, therefore, more accurate to hold that clonic convulsions depend on the want of arterial blood in the spinal cord and the medulla oblongata, caused partly by the further extension of the first excitation (contraction of arteries), and partly by the asphyxia inducing a condition of venosity through the whole system.

It may also be open to question whether the cessation of the convulsion depends on exhaustion of the nervous power. If the view is correct that the clonic convulsions are induced by a want of arterial blood, their cessation would be the consequence of a restored supply of this blood. The convulsion thus might be an evidence of want of nervous action, and its cessation would be consequent

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on a restored nervous action. If it is allowed that loss of consciousness is occasioned by arterial spasm in the cerebral lobes (frontal), and that the tonic rigidity and clonic convulsions are the consequence of arterial spasm, similar in kind if not in degree, in the motor area of the brain, and perhaps, too, in the medulla oblongata and spinal cord, it is difficult to believe that in the first case the function of the part is temporarily annihilated, whilst in the second it is intensely exalted. It is scarcely reasonable to suppose that the same condition which, when acting on the frontal lobes, causes destruction of all cerebral functions, should, when acting on the motor area, or upon the spinal cord, cause an intense exaltation of motor power. Nor can we believe that a fresh supply of arterial blood, which, acting on one portion of the brain, causes it to resume its functions, would, when acting on another portion, almost in a similar ratio, lower and depress the functions of this nerve centre.

In illustration of the theory that clonic convulsion may not be the consequence of excitation of the nervous centre by venous blood, but rather a manifestation of diminished supply of arterial blood, may be mentioned an experiment performed by Vulpian, and recorded in his 'Leçons sur la Physiologie du Système nerveux,' p. 455: 'J'ai vu chez un lapin la compression des artères carotides et vertébrales déterminer une suspension des fonctions encéphaliques: mais, chose bien remarquable, la respiration spontanée continuait, le bulbe rachidien

ayant échappé plus ou moins complètement à l'anémie encéphalique. Les mouvements spontanés et réflexes avaient entièrement disparu dans la face et les yeux ; le tronc de l'animal vivait encore en supportant une tête physiologiquement morte. Eh bien, au bout de deux ou trois minutes, les moyens de compression ayant été enlevés, il se produisit d'abord des mouvements convulsifs assez violents, qui ne durèrent que peu de temps : puis toutes les manifestations de la vie, tous les mouvements volontaires et autres reparurent peu à peu dans la tête : l'animal recommença à marcher et revint bientôt à son état normal.'

Here the convulsions were the connecting link between apparent death or paralysis of the muscles of the head and face, and their restoration to voluntary movement. Entire loss of arterial blood from the part was followed by loss of all movement ; the very commencement of restored arterial flow was followed by convulsion before the full circulation was restored ; and a full restoration of artificial circulation was the immediate precursor of normal life in the part.

Again, the explanation of these convulsive conditions which simulate epilepsy, but without loss of consciousness, seems particularly difficult if we accept the theory that the accumulation of venous blood at the base of the brain is necessary for their production. In the second case of this kind, mentioned above—that of E. P.—the patient seemed to have no stage of tonic spasm preceding the clonic convulsion. Neither was there the slightest headache or sleepiness following the attacks, the extent of which

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is a fair test of the amount of venous congestion in the brain. From a condition of ordinary healthy life, she was wont to be taken with the intense clonic convulsion previously described, and the moment the convulsion ceased she was herself again. The observation of all such cases seems to point to partial arterial spasm of the motor area of the brain as the proximate cause of the convulsion. Several cases of a similar nature are collected from various sources in Dr. Reynolds' work on epilepsy. It is true enough that some tonic spasm may exist, and yet be very difficult of detection, and Trousseau would seem to look upon its non-existence as an impossibility. He says, 'The tonic always precedes the clonic stage, but the duration and violence of the latter are by no means proportionate to the duration and violence of the former. Thus, very violent clonic movements often succeed a slight tonic contraction, and, reciprocally, an excessively powerful tonic contraction may be succeeded by very moderate clonic movements. Thus, the length of the first stage is sometimes so short, and the second stage comes on so quickly, lasting for a more or less prolonged period, that an observer who is not on his guard, or not very attentive, might think that the convulsions were clonic at the outset. . . . From what I have said, this remarkable fact follows, that rigidity seems to be an essential obligatory element of all convulsion. It is never absent, and can even be alone present, whether it constitutes the convulsion by itself, as in idiopathic contractions, or whether the convulsion is

incomplete, as in eclampsia, where the clonic stage is absent, whereas clonic movements never, perhaps, come on from the first.' This is a strong statement. In a case before mentioned, the woman has more than once been talking in the out-patients' room, when the first symptoms of the attack have commenced in rapid twitchings of the facial muscles, followed immediately by clonic convulsion of the limbs; and with the first twitching she has said, 'Now, sir, the fit is coming on.'

It is difficult to conceive how any person could speak with any laryngeal spasm sufficient to cause cerebral congestion; and if this venous blood theory be right, how is it that the true spasm and the clonic convulsion do not bear a direct ratio to each other? A slight tonic stage should be followed by slight clonic convulsion, instead of the two stages bearing, as they often do, almost an inverse proportion to each other. How, too, can the period of rigidity exist by itself, unfollowed, as is sometimes the case, by clonic convulsions? It seems, then, probable that clonic convulsion is an evidence of partial arterial spasm in the motor area of the brain, in the medulla oblongata, and in the spinal cord; that it plays no part in the causation of the subsequent headache and sopor, except by sometimes adding the element of exhaustion, when the convulsion has been very intense and protracted; and that when such convulsion has followed the injection of venous blood, it has been from the venous blood inducing contractions of the arteries, as the blood in the exanthemata sometimes does.

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The attacks, which most frequently consist only of clonic spasm, are, as Dr. Gowers has written, slight fits, of partial distribution, beginning in and often confined to one limb. 'The most perfect type of these attacks are those which are sometimes produced by organic brain disease, and Hughlings Jackson has proposed to designate them "Epileptiform convulsions," as distinguished from ordinary epileptic fits. But such local attacks of clonic spasm sometimes occur in cases in which there is no other reason to believe that there is such a disease as tumour, and in which the variable position of the local fit and other symptoms preclude the supposition that the symptoms are due to any fixed coarse organic disease. These local clonic convulsions are especially common in the upper limb, beginning in the hand. First the fingers begin to twitch, then the elbow is jerked and the shoulder. The attack may then cease, or the face or leg may be jerked in the same manner. Consciousness is often not lost, if the spasm is confined to a single limb. Less commonly these attacks begin in the face, and still less commonly in the leg.'

The convulsive phenomena of an attack of epilepsia gravior may well be termed the effect of a discharge from a morbid nerve-centre. By some this term may be considered rather the expression of a fact than an explanation of it. But if such discharge be wholly independent of the state of the vessels, and therefore of the vasomotor system, what explanation can be given of the prevention of an attack by tying

a ligature, blistering, &c., at some spot between the apparent focus of origin of an aura—that is, the spot to which the sensation of the aura is reflected, and the centre of discharge. It is under all circumstances difficult to give a reason why this method does in most cases delay or prevent the attack. But it is in analogy with the effect of similar treatment in other parts of the body that its action is a reflex one on local internal circulation, and as the effects of this mode are either on the sensory nerves of the limb with which vasomotor fibres are largely commingled, or on the vessels of the limb innervated as they are by the vasomotor system solely, it seems easy to understand that the reflex effect will have a primary influence on vasomotors at a distance, and that a fit is prevented by alteration in the calibre of vessels. In describing the pathology of a fit, Dr. Brown-Séquard, in one of the latest articles that has been written on epilepsy, repeats in the main his belief of more than twenty years ago. The course of a fit is as follows:

Cause.	Effects.
1. Excitation of certain parts of the excito-motor organs of the nervous centre.	1. Contraction of blood-vessels of the brain and face; tonic spasm of muscles of the eye and face.
2. Contraction of the facial blood-vessels.	2. Facial spasms.
3. Contraction of the blood-vessels of the cerebral lobes.	3. Loss of consciousness; congestion in the base of the brain and the spinal cord.
4. Extension of the excitation in the excito-motory organs of the nervous centre.	4. Tonic contraction of the laryngeal, the cervical, and some respiratory muscles (Laryngismus and Trachelismus).
5. Tonic contraction of some respiratory and vocal muscles.	5. Epileptic cry.

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CAUSE.	EFFECTS.
6. Further extension of the excitation in the excito-motory organs.	6. Tonic contraction, reaching most muscles of trunk and limbs.
7. Loss of consciousness alone, or with tonic spasm in trunk and limbs.	7. Fall or precipitation forward or backward to the ground.
8. Laryngismus, trachelismus, and rigid spasm of some respiratory muscles.	8. Insufficient breathing; obstacle to entrance of blood into the chest and to its issue from the cranio-spinal cavity.
9. Insufficient breathing, rapid consumption of oxygen, and detention of venous blood in the encephalon.	9. Increasing asphyxia.
10. Asphyxia and perhaps pressure by accumulated serous blood in the base of the brain.	10. Clonic convulsion elsewhere; contraction of the bowels, the bladder, the womb; increase of secretions; efforts to inspire.
11. Exhaustion of the nervous power generally, and of the reflex excitability especially; return of regular respiratory movement.	11. Cessation of the fit; coma or fatigue; headache and sleep.

Brown-Séquard makes the alteration in these views, that the loss of consciousness is too rapid, too complete, to be due only or chiefly to a contraction of the blood-vessels of the cerebral lobes. It is owing, he thinks now, to an inhibition of the activity of cerebral nerve-cells that this symptom appears. On what this inhibition depends, or how induced, he does not say. Is the sudden loss of consciousness in an attack of syncope due to an inhibition of the activity of cerebral nerve-cells? Without doubt. But in syncope so grandiose a form of expression is not considered necessary. It is enough to say that the nerve-cells are inactive because they are cut off

from their basis of supply, the arterial circulation. It is not so very uncommon to see an attack of syncope as sudden, as rapid, as the loss of consciousness and the fall in epilepsy.

It does not seem unreasonable to believe that conditions so closely allied own one and the same causation, a cessation of arterial blood-supply, though in one case the *modus operandi* is deficient *vis à tergo*, in the other arterial spasm.

It is possible that the seat of origin of epileptiform convulsion is not the same as the centre of true epilepsy; that in the former case the purely motor area in the immediate neighbourhood of the fissure of Rolando is implicated, in the latter the frontal lobes.

Putting aside for the moment the question of true epilepsy, and speaking only of epileptiform convulsion, is it necessary, is it indeed possible, to insist on a morbid instability of cells of this or that convolution, which from its very nature must have been making gradual progress for some time before the so-called discharge? How, then, were animals rendered subject to epileptiform attacks, if not to true epilepsy, by the section of the sympathetic in the abdomen? How was a similar effect produced by M. Westphal by slight percussion on the head? How are the symptoms of an epileptic attack induced by Ferrier by submitting the surface of the cerebral hemispheres, in a rabbit or a cat, to the influence of a pretty intense faradaic current? How even are these cells rendered so rapidly unstable by the essence

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of absinthe, as in M. Magnan's experiments? How, too, are animals rendered liable to epileptiform seizures under the influence of experimental lesions of the spinal cord, or of the cutaneous nerves?

If the instability of a cell means that it is deprived of its blood-supply—if its less power of resistance is explained by saying that a cell at once begins to lose its normal function when the process of interchange between cell and vessel comes to an end by the abnormal constriction of the latter, then the reflex effects of the experiments given above find a suitable explanation. Irritation of the sympathetic in the abdomen may be transmitted either directly from ganglion to ganglion, until it reaches and influences the cells of the cervical sympathetic, or it may be carried through the spinal cord itself to the cervical ganglia; irritation of the sympathetic fibres contained in the sciatic or crural nerves, or in the spinal cord itself, may also find a similar pathway through the cord to these cervical ganglia. But this may not be the only region of transmission. The existence of vasomotor nerves, destined for the vessels of the brain, and reaching them neither through the cervical cord of the sympathetic nor through the upper cervical ganglion, has been proved by the experiments of Nothnagel. When in rabbits the upper cervical ganglion has been destroyed, this observer has seen that galvanisation of sensory nerves—the crural, for instance—causes constriction of the arteries of the cerebral pia mater, and that this effect is produced more easily on irritation of the terminal

expansion of these nerves than of their trunks; and although other observers have not always met with the same success, yet Vulpian, after extirpation of the superior cervical ganglion, has found faradisation of the sciatic nerve to be followed by dilatation of the pupil, and by constriction of the vessels of the ear.

It will be easily recognised how loth the writer is to give up the *rôle* of the sympathetic in the causation of at least some of the important phenomena of epilepsy. In epileptiform convulsions, such as seem to be set up by extrinsic irritation, the irritation of the vasomotors may be sufficient in itself to start the convulsive symptoms; although even here patients, the subjects of such convulsions, are not the strongest people in the world, and may have derived some hereditary neurosal tendency from a former generation.

But in true epilepsy it is probable, from various causes—hereditary, insanatory, abnormal—the nutrition of the brain, or of certain portions of it, has gradually been seriously modified. In such persons, such modification of nutrition may show itself by an irritative state, that impresses itself primarily on the vasomotors of the vessels of the region so affected. This impress may find the centre of its reflex arc in one of the ganglia in or on the vessel itself, causing partial constriction of vessel over a very localised area, and so un-inhibited convulsive movement of a single limb, for instance, or portion of a limb. More usually the eisodic influence is carried to the medulla oblongata, and may constrict all vessels deriving their

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direct vasomotor influence from thence, or through the cervical cord to the superior cervical ganglia, from which most of the cortical vessels derive their innervation. From so extended a region of constricted cerebral vessels result, first, loss of consciousness, and, secondly, tonic spasm. The clonic convulsion is rather the effect of the gradual yielding of the vasomotor constriction, allowing at first more blood to enter the arteries than during the period of tonic spasm, but yet far less than is necessary for controlled movement or for rest; and the headache depends on the various congestions, the consequences of laryngeal spasm, the coma, and then the prolonged sleep, partly on fatigue from exhaustion of the nervous powers generally, but mainly to lesser phases of the cerebral congestion, the result of the fit; which congestion passes away as the blood is enabled to return to the right side of the heart, and as respiration returns to its normal condition.

The part, therefore, taken by the vasomotors is, primarily, that they have had something to do in rendering the nutrition of the brain-cells imperfect; but also that they feel the first effect of this malnutrition, and, as a consequence of this irritative effect, induce, by their constriction of vessels, loss of consciousness and tonic spasm of muscles.

Dr. Mussen Young has drawn attention to cases which illustrate what he calls 'an epileptic condition of the centres which control the nutrition of the body.' The following case is a typical one: A. is now 27 years of age, and has been an inmate of

the Suffolk County Asylum for nine years. In order to understand her condition, it will be best to begin by describing her as she appears under the most favourable circumstances, when her general physiognomy is very good, and her appearance somewhat prepossessing, with a florid complexion, but rather epileptic expression. Her mental condition is at all times weak, but when in the condition just described she is not inclined to be violent, understands most of what is said to her, and gives fairly rational answers when questioned. This comparatively satisfactory state lasts for some time, when suddenly a marked change, more especially physically, takes place, and within twenty-four hours a decided deterioration is observed. This degeneration progresses very rapidly, but not uniformly, as during the course of an attack she frequently exhibits apparent signs of improvement, or at least of arrest of the attack. Any hopes, however, of a permanent change for the better are soon dissipated by a relapse into a worse state than formerly; and at the end of a fortnight her general appearance has so much altered that she would not be recognised as the same person, unless special attention with that object were directed towards her. Although she may be thus reduced to a kind of living skeleton, and may appear to be literally dying of starvation, her appetite remains unimpaired, and she takes her food well, and with no abatement of relish. Coincidently with this alteration in her physical condition, her mental state gradually becomes more unsatisfactory, until at last

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she is restless and troublesome, incapable of understanding any conversation, or of herself speaking otherwise than in a most incoherent manner. This condition continues until she seems to approach a state of collapse, when an improvement as rapid as the original degeneration sets in, and in a very short time she regains her former satisfactory state. This, however, again proves to be of only temporary duration, and the same sequence of events follows. The quiescent period which elapses between any two seizures is very variable, and does not bear any relation to the menstrual function.

'The periodicity of the attacks in this case, together with the course they run, present (says Dr. Young) many points of analogy to those cases of epilepsy in which there are considerable intervals of entire immunity from fits, followed by periods during which the seizures are so frequent and severe as to leave the subject of them quite prostrate for a time, until they gradually pass off and another interval of repose ensues. The resemblance is still more marked by considering what might be called the convulsive character of the physical degeneration, as shown by the temporary intervals of improvement or arrest in severity of the attacks. The intense degree of malnutrition cannot be owing to a defective supply of nutritious matter, because the appetite is always very good, and there are never any signs of gastric or intestinal derangement. The entire course of events may fairly be accounted for by supposing that the cerebral centres which govern the nutrition of the

body may be acted upon by the same conditions as those which affect the motor centres, occasioning ordinary epilepsy. In favour of the idea that the blood-supply of the nervous centres is at fault, it may be urged that, considering the extreme vascularity of nervous tissue everywhere, a slight derangement either of the supply or of the quality of the blood would be likely to seriously interfere with the proper fulfilment of the functions of any part of the nervous system. On the other hand, when it is remembered what an important part the vasa vasorum play in the nourishment of the larger blood-vessels, and that these vasa vasorum are ultimately in great measure controlled by branches of the sympathetic, it is open to supposition that the small nervi nervorum have a considerable share in the regulation of the nutrition of the nervous centres, and that the morbid phenomena described above may be due to their nervous force periodically acquiring, as it were, a state of unstable equilibrium.

'Why such a condition of things should occur, and be manifested in such a manner, is as inexplicable as why miasma should affect the body in the periodic manner witnessed in ague.'

Consult—
Eulenberg and Guttman, op. cit.
Dr. Gowers, 'Epilepsy.'
Binswanger, 'Med. Record,' vi. 467.
Brown-Séquard, 'Lancet,' 1866, i. 141.
Nothnagel, 'Med. Record,' vi. 480.
Echeverria, 'Epilepsy.'
Dr. Young, 'Brit. Med. Jour.' 1882, i. 615.

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Dr. Pommay, 'Med. Record,' x. 138.
Chapin, op. cit.
Dr. R. Reynolds, 'Epilepsy.'
Vulpian, 'Leçons sur la Physiologie du Système nerveux.'
Trousseau, 'Clinical Medicine.'
Quain's 'Dict. of Medicine,' article—'Epilepsy.'
'Comptes Rendues de la Société de Biologie,' 1872.
Vulpian, 'L'Appareil Vasomoteur.'

CHAPTER IX.

SPINAL CORD LESIONS—PROGRESSIVE MUSCULAR ATROPHY—TABES DORSALIS.

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No more than the ordinary vasomotor influence can be traced in most of the other morbid conditions to which the cerebro-spinal nervous system is liable. In hæmorrhage, in cerebral softening, in sclerosis of the brain and cord, in inflammation of the cells of the anterior horns or in atrophy of the same, the sympathetic has not much to do directly. In one disease just mentioned—progressive muscular atrophy—the fact that the lesion is due to a malnutrition of cells of the anterior cornua, induced by a condition of the nutritive vessels rendered morbid by an abnormal state of the vasomotor nerves, is probable, but has certainly never been demonstrated. It has, however, been already mentioned that Sir Charles Bell thought the sympathetic was concerned in this disease, and that Jaccoud shares his views; and that, besides the lesions of the anterior horns, the cervical ganglia of the sympathetic have been sometimes found converted into fat; whilst the raised temperature in the early stages, diminished later, the increased perspiration, the atrophy of the layers of skin, the

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painful swelling of joints, and sometimes the contraction of one pupil, all point to some sympathetic connection. The affection of the sympathetic, which is present in some cases, is a combined affection, characterised clinically by its special group of symptoms (oculo-pupillary phenomena).

Remak holds that progressive muscular atrophy is a disease of the sympathetic ganglia, and also of the cervical part of the spinal cord.

The question is, Is the sympathetic really affected at all? And if it is, is it through centripetal conduction of the primary muscular disease to the peripheral nerves, the spinal roots, and the rami communicantes; or, contrariwise, is the sympathetic affection the primary one, spreading centrifugally to the peripheral nerve-trunks and muscles, and centripetally to the spinal centres?

In Schneevogt's cases the cervical cord of the sympathetic was almost converted into a cord of adipose tissue, in which the nerve fibres were replaced by flat cells containing crystals. The cervical ganglia had almost completely degenerated into fatty tissue, whilst the thoracic part of the sympathetic likewise contained some fatty matter.

If among the disputed positions of the vasomotor centres in the spinal cord, the smaller cells of the anterior cornua be assigned as their proper place (and by mere process of exhaustion it is difficult to make any other choice), it is scarcely to be wondered at that some cases of progressive muscular atrophy are associated with vasomotor phenomena.

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In pseudo-hypertrophic paralysis, one case has been attributed to paralysis of the sympathetic; but from the study of the physiology of the sympathetic, the course of the disease, the post-mortem appearances, rendering the nervous origin of the disease more than doubtful, and the sufficiency of other explanations of the lesions, the conclusion is almost necessary that the sympathetic has little or nothing to do with this disease.

It is quite another question whether progressive muscular atrophy has not some influence on the sympathetic. Myosis, in this disease, is rare; but when it exists, it may depend in some sort on the cutting off more or less of the influence of this nerve from the ciliary ganglia, so that, its inhibitory effect on the contraction of the pupil being removed, the oculo motor acts with great intensity. But this and all other sympathetic phenomena in progressive muscular atrophy seem to be secondary in point of time to the disease itself.

The same thing may be said of other spinal diseases, and even of spinal injuries. It is probable enoughthat in chronic myelitis, from compression or otherwise, if absolutely localised to regions that are known to contain special sympathetic centres, the vasomotors of the head and face, or at least of one side of them, may be affected, or there may be seen certain oculo-pupillary phenomena, or in any region the vasomotor connected with that portion of the spinal cord may be at first constricted from irritation and afterwards dilated from paralysis. This would

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be a not improbable effect of the localised lesion of the cord. As a matter of clinical observation, however, it is exactly in such lesions that the sympathetic ganglia, cervical, dorsal, lumbar, &c., manifest their independent action, and by keeping up the circulation in the various parts of the body as well, or almost as well, as when the cord is intact, show that, over and above the force derived from the centres in the spinal cord, they possess an inherent power of their own, that under certain conditions suffices for the requirements of the system. As a matter of experience also myelitis is not diffused. It may implicate the whole of a segment of the cord horizontally, but it is not usual for it to spread very far vertically, and the secondary degeneration of Turck's column in a descending direction, of Goll's column in an ascending, are not such as would be likely to have any effect on the sympathetic nerves.

Trousseau, however, speaking of the congestive phenomena in tabes dorsalis, says that 'these congestive phenomena show themselves in other diseases acknowledged to belong to the class of neuroses, such as hysteria, asthma, and exophthalmic goître; and they belong, in my opinion, to the same category as those which in his experiment Professor Claude Bernard produces at will by dividing the sympathetic. They point to some disturbance in the functions of that nerve, of which we neither know the nature nor the cause.'

That the sympathetic may be somewhat involved in tabes dorsalis is seen by the altered condition of

the pupils, by the gastric, intestinal, and vesical crises, by the permanent acceleration of the pulse which has been sometimes observed, by the alteration in the secretion of the alimentary canal, by cutaneous ecchymoses and eruptions, and, according to some, by the affection of the nails and joints. Certain alterations have also been seen in the functions of the sweat-glands.

Twenty years ago, Duchenne, of Boulogne, stated that in a certain number of cases of this disease the cervical sympathetic must be regarded as its starting point. This he inferred from the frequent occurrence of the well-known oculo-pupillary symptoms, and from the various vasomotor disturbances on the face and elsewhere. He believed that the sympathetic is only functionally affected, and that as a result of this there is a neuro-paralytic state of the vessels of the posterior columns, resulting finally in the changes found here on autopsy. The sympathetic has been found structurally healthy by adequate observers in this disease, but this does not invalidate Duchenne's views, as he only speaks of a functional disturbance. Rosenthal says that the symptoms above mentioned are due to an affection of the centres in the cord from which the sympathetic fibres arise, the centrum cilio-spinale superius and inferius, the centrum genito-spinale, and the vasomotor centres scattered along the whole length of the cord.

Professor Fürstner and Dr. Zacker, of Heidelberg, have recorded an unusual case, in which the most marked lesions consisted in the formation of cavities

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in the spinal cord. The disease was associated with peculiar vasomotor disturbances and some morbid condition of the cervical sympathetic; and the theory is advanced that perhaps this latter lesion was the causation of the abnormal changes found in the spinal cord and in the brain. In the early stages there was contraction of the right pupil, much sweating on slight exertion over the right half of the face, with slightly lessened temperature on the right side. On touching the skin, pricking it, stroking it with a finger-nail or with a key, there was seen, on the part touched, first a pale colouring of the skin, framed by two red streaks, then in place of the pale colouring there occurred a light red colour, and the parallel streaks grew pale. In one or two minutes, in the whole region of the reddened part there were raised clear blisters, which became confluent, corresponding to the early red streaks, forming a channel filled with serum, quite overtopping the level of the skin, remaining several hours and then vanishing completely. On more mechanical irritation there occurred in the whole region a bright rose colour, which rapidly became pale again. These and analogous phenomena were confined to the right side of the body. Later on, profuse lachrymation occurred on the right side only; with injection of the right eye, and much redness and swelling of the mucous membrane of the lids, and dilatation of the left pupil. Besides these sympathetic phenomena there were found right analgesia; trophic disturbance of the right hand; failure of deep and cutaneous

reflexes on the right side; later on, failure of left patellar tendon reflex; certain cerebral phenomena, especially disturbance of speech. Without mentioning all the particulars of the lesions found, it may suffice to speak of glia-proliferation and chink-formation in the right medulla oblongata; formation of cavities in the spinal cord; degenerative changes in the nuclei of the hypoglossal, vagus, spinal accessory, and ascending root of the fifth nerve. In the cervical region there was destruction of the right posterior horn, of the posterior part of the grey commissure, of great part of the left posterior and right anterior horns, and only slightly of the left anterior horn. In the dorsal region, there was destruction of the whole of the right anterior and posterior horns, in great part of the left anterior horn, and all the left posterior horn. In the lumbar region, there was destruction of the right posterior horn in great part; less of the right basal substance and grey commissure. The upper and lower cervical ganglia were submitted to a close investigation, and showed decided differences between the right and left sides. These consisted specially in the fact that on the right side signs of extreme hyperæmia and inflammatory changes were found, which on the left side seemed far less distinct. In the former the mass of visible capillaries turgescent with blood was of such a kind that the coloured section retained a yellow colour. Besides, the arterial vessels showed pretty considerable changes of their walls, especially of the adventitia, which were partially and pretty richly permeated with

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cellular elements. On the left side these changes were less. The accumulation, too, of pigment within the ganglion cells seemed to be more intense on the right side than on the left; yet we must scarcely lay any particular weight on this point, as the accumulation of pigment in the cells of the sympathetic, even when normal, may be considerable. The peculiar vasomotor disturbance was bilateral, though chiefly of the right side, in unison with the pathological anatomy. It was evidently a paretic condition of the whole of the vessels of the skin, exclusive of the face, and was only a higher degree of what is seen in congestive roseola and meningeal tâches, and was a specimen of angio-paralysis, and probably of spinal origin. We refer to this the profuse secretion of sweat of the right half of the face, the headache, tinnitus, and perhaps the lachrymation repeatedly observed in the right eye, connected with the great injection of vessels of the mucous membrane, and the formation of bladders of the upper lid, though these latter phenomena might be referred to the pathological function of the trigeminus. The later extensive paresis of the vessels of the skin should be referred to functional changes in the central vascular centres, presumably spinal.

This injury of the vasomotor centres could have influenced the vessels of one side of the cord, and perhaps those of the brain on the other side.

These observers think that the autopsy in this case throws light on the three following cases, in which the post-mortem records are not so complete:

1. Westphal's case. Male, 37 years old. First phenomenon three or four years ago. Atrophy of left hand and of fore-arm, deafness, formication; ice-cold condition of right arm, loss of coarse power in both sides. Right-sided headache. Transient difficulty in swallowing and of speech. Hoarseness. Giddiness, connected with anxiety. Numbness over the whole body. Among the latter phenomena disturbance of circulation and of respiration. Increase of temperature. Death.

Post-mortem.—Swelling and formation of cavities in the spinal cord. In the cervical region a part of the anterior horns, and larger or smaller remains of the posterior horns persisted. Lower down the substance of the anterior and posterior horns only dislodged. One posterior horn thinned. Nowhere destruction of the whole grey substance. Unilateral myelitis in the medulla oblongata (at least of the hypoglossal nucleus). Fatty degeneration of the hypoglossus, vagus, and glossopharyngeus. Flattening of one olive without further change.

2. Schuppel's case. Anæsthesia, paralysis of the sense of temperature, trophic disturbances of the upper extremities. Gradual anæsthesia, and analgesia of the trunk and lower extremities. No paresis. No muscular atrophy.

Post-mortem.—Central spinal formation of cavities. Lesion extended from level of first cervical nerve to lumbar swelling. Destruction of posterior horns, anterior and posterior commissures, posterior column, and partially of posterior roots. Slight destruction of anterior horns.

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3. Schultze's case. Female, 37 years old. First phenomenon four years before death. Formication, fibrillary tremor of upper extremities. Formation of bladders on the right hand. Atrophy and paresis of both arms. Analgesia and partial anæsthesia. Diminution of left tendon reflex. Disturbance of speech. Paresis of tongue. Bulbar phenomena.

Post-mortem.—Glia-proliferation and formation of cavities in spinal cord. Chink-formation in right half of medulla oblongata at level of hypoglossus nucleus, corresponding to the direction of the vagus. Sclerosis and atrophy of left olive. Degeneration of ascending root of fifth cervical region, partial destruction of all the anterior and posterior horns. Dorsal region, destruction of all the left half of the posterior horn, less of the right. Lumbar region, destruction of left posterior horn, and of the anterior section of the posterior column.

Dr. Alexander Hackin, of Belfast, has drawn attention to that portion of the dorsal cord covered and limited by the fourth and fifth dorsal vertebræ. He had observed in every case of hysteria, chorea, neuralgia, facial paralysis and other neuroses in females, the co-existence of pronounced spinal tenderness on pressure over the fourth or fifth, more frequently the fourth and fifth dorsal vertebræ; with this practical result, the almost invariable and rapid cure of those maladies by remedies applied over these vertebræ alone. He soon after recognised a similar condition of the spinal column in males so affected. Still further observation elicited the un-

recognised fact that this local spinal peculiarity, which at first had been looked upon as a morbid symptom and the heritage of enfeebled constitutions, was in reality the normal condition of every healthy individual as well, of every age and class.

It is interesting to note the very various ailments that in Dr. Hackin's experience have yielded to counter-irritation over this region. 'Among the ailments,' he says, 'that have yielded so rapidly to the counter-irritant treatment were trigeminal neuralgia, facial paralysis (Bell's), acute hysteria, dysmenorrhœa, the reflex vomiting, the neuralgic toothache, and the pruritus pudendi of pregnancy, gastralgia and other neuroses;' and when vesication was the method adopted, he was generally enabled to assure the invalid that in five hours, coincident with the formation of the blister, all painful symptoms would permanently take their departure. By neuralgia he means the typical disease, of remittent character generally, but not always departing at night, to recur at the same hour every morning, or vice versâ, and not that hybrid ailment, principally rheumatism, so often miscalled. Chorea has almost always yielded to this plan of treatment, with some important exceptions, including the chorea of pregnancy and that form depending on organic cardiac disease. The dysmenorrhœa of neuralgic type yields readily, and he has not once failed for many years, by a single vesication over these vertebræ, to put an end at once to the sickness of pregnancy for the whole remaining period of gestation, no matter at what

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stage he was consulted. The neuralgic toothache and the pruritus pudendi of the puerperal condition yield as readily, and to one application.' And the remarkable point in this observation is, that after the healing of the blister and the cure of the disease, the hyperæsthesia of this spinal region remains the same.

Mr. Pridgeon Teale had before this expressed his belief that there exists an etiological relation between an irritable state of the spinal cord and sympathetic ganglia, and neuralgic troubles in remote organs; but his proposition refers chiefly to phenomena exhibited in those remote parts to which the nerves from the diseased portion (cord or sympathetic ganglia) are distributed. They do not explain the difficulty that counter-irritation of an area unconnected by nervous filaments with the seat of pain or paralysis will also as rapidly stifle the disorder. Dr. Brown-Séquard has announced as a general principle that general or local anæsthesia can be produced in the lower animals by applying to their peripheral nerve-distribution a strong counter-irritant, like a few drops of chloroform, and that the anæsthesia was explicable on the theory of inhibitory action.

Nothing is more easy (or more unsatisfactory) than to carp at therapeutical results recorded by others, and in the present case it may scarcely be permissible to wonder whether similar universally good results will invariably follow the same simple mode of treatment under other conditions of climate, race, &c. As a matter of experience the great hyper-

æsthesia of this spinal region has never been recognised by other neurologists, by whom, for purposes of diagnosis, careful percussion of the spine is practised daily.

But in taking the fact as somewhat less universally true, it is necessary to look for the explanation in the considerations—(1) that each segment of cord is inhibitory of the parts below it; (2) that in many cases the ganglia, from which proceed the accelerator nerves of the heart, frequently derive some of their spinal origin as low as the region above named, and that these ganglia rule not only the heart, but the vascular tone of the whole of the upper part of the body; and (3) that from the same region are supplied the ganglia that give off branches to form the great splanchnic nerve, the most important strand of the sympathetic in the body.

It will be remarked that most of the ailments found to be controlled by counter-irritation of this spinal lesion are due to modifications of blood-supply, and therefore are such as may be benefited by any change of the blood-supply. Such an action may not unreasonably be supposed to be the indirect result of counter-irritation in this region. The explanation of its influence on the reflex vomiting of pregnancy is less easily to be explained, except it be that in reflex vomiting of pregnancy the eisodic course passes up the spinal cord to the origin of the phrenic, and the vagus, and that counter-irritation at this point for the time being prevents the passage of the irritation through this portion of cord. It is

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probable, too, that the irritation may sometimes travel by sympathetic paths, and only enter the cord through the splanchnic nerve origins. If so, this same region would be again the bar to further progress.

Whether a blister at this spot acts like Dr. Buzzard's blisters over the course of an epileptic aura, or whether it acts by modifying the circulation in this segment of cord, thereby preventing the function of the cord at this spot, it is difficult to determine.

A case has been recorded by that excellent neurologist, Mr. W. H. Kesteven, in which there had been some pre-existent disease, which had caused the destruction of the body of the seventh dorsal vertebra. About three months before death occurred, the patient had received a blow on the back, which had caused some displacement of the diseased part and rupture of some blood vessels, thus giving rise to an effusion of blood into the spinal canal, and consequent pressure on the cord. Some of the blood had also escaped anteriorly, forming a hard tumour in the posterior mediastinum, which had pushed forward the sympathetic ganglia, and caused destruction of the rami communicantes of the seventh ganglion on each side. The heads of the seventh and eighth ribs on each side were also diseased. The symptoms which had been noticed during life pointed chiefly to pressure on the spinal cord produced by the hæmatoma, and to damage also caused to the sympathetic by pressure. Mr. Kesteven, in a note to the writer, explains that the

symptoms during life were such as would be caused by loss of tone in the hepatic vessels, e.g. diminished secretion of bile; the same condition in the kidney, as shown by scanty urinary secretion; sluggish or impaired action of the circular muscular fibres of the intestines, resulting in imperfect peristalsis; impairment of bladder, as shown by inflammatory symptoms in that organ, and loss of muscular power in its walls. These phenomena would result from the destruction of the rami communicantes of the seventh ganglion, whereby the nervous power of the ganglion on each side would be diminished, this morbid condition being conducted through the splanchnic to the solar plexus, and there upsetting the balance between the sympathetic or vaso-constrictor action and the cerebro-spinal or inhibitory action, with the results as above.

There are a certain number of cases on record in which dilatation of pupil has resulted from irritative lesions of the cervical cord, especially of a traumatic origin. Occasionally this symptom is the result of a non-traumatic lesion of this portion of cord. Paralytic myosis has very occasionally been observed as the result of traumatic lesion of the cervical cord.

Crushing accidents to the upper dorsal region between the seventh cervical spine (first dorsal nerve) and the fourth dorsal spine (sixth dorsal nerve) may, among other phenomena, occasionally lead to priapism, in which, with irritation of the nervi erigentes, there will be a paralytic condition of the vaso-constrictors.

Lesion of the lower dorsal region, between the

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upper border of the fifth dorsal spine (seventh dorsal nerve), and the lower border of the tenth dorsal spine (space below the twelfth dorsal nerve) may lead to any or all the lesions that would follow either irritation or paralysis of the splanchnic nerves, deficient peristalsis, constipation, scantiness of bile, scantiness of urine, vesical weakness, or congestion of spleen, of liver, of kidneys (possibly with albuminuria), of stomach, and much of the intestines, with abnormal secretions of the alimentary canal, diarrhœa, even intestinal hæmorrhages and congested mucous membrane of bladder. Lesion here, too, by its influence on the spermatic plexus, may influence the formation of semen.

Lesions of the lumbar cord, between the lower border of the tenth dorsal spine (just below the twelfth dorsal nerve), and the upper border of the second lumbar vertebra, may lead to complete paralysis of the bladder and rectum, with tendency to inflammation and ulceration of the former organ.

The enormous number and importance of the sympathetic ganglia and nerves of the uterus render this organ less closely dependent on the spinal cord than many others. Any crushing accident, however, to the lumbar cord, of a nature to irritate the nerves that indirectly come off from it to the uterus, would cause contraction of that organ, and, if in the pregnant state, expulsion of the fœtus.

Disease of the cervical cord may involve augmentation of temperature in the body generally (hyperpyrexia).

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In concussion of the spinal cord, the temperature will probably be at first depressed, as the result of shock, though subsequently a febrile elevation may continue for some days.

In punctured or gunshot wounds of the spinal cord, there is found on the side of the motor paralysis a vasomotor paralysis also, which carries with it as a consequence (1) an elevation of temperature from $1\frac{1}{2}°$ to 2° Fahr., and (2) a hyperæsthesia for all modes of sensibility (owing in part to hyperæmia in limb and cord and nerve-roots).

Of course, with reference to thermometric observation, the rapid sequence of spinal meningitis or myelitis must be borne in mind. Sudden crushing lesion of the spinal cord, with fracture or dislocation of vertebræ, show variations in respect of the temperature of the parts below the lesion that depend on its nature, the intensity of shock, &c. The general course would be—(1) Diminution of temperature, as the result of irritative constriction of vessels from shock. (2) Increase of temperature from vasomotor paralysis, which, though not complete (the sympathetic ganglia to some extent supplying the vaso-motor nerve-force, which is lost from the spinal lesion), is sufficiently distinct to lead to some increase of heat. (3) If the patient lives, a coldness of the paralysed region occurs, partly from paralytic inactivity of the limbs, partly because all nutrition in a part below so severe a lesion of cord is carried on imperfectly. This coldness is delayed if the original

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lesion sets up more or less localised meningitis and myelitis.

In a crushing lesion of the lower cervical region marked hyperpyrexia is sometimes met with, the temperature rising to 108° and even to 112°. Perhaps this means that the lesion being of a region above the segment from which the heart derives its spinal innervation, the nutrition of the body, and chemical action everywhere goes on, and is not controlled by the action of the inhibitory heat-centre above. It is similar to what is seen so often just before death, where the functions of the medulla oblongata are being gradually paralysed. The difficulty about the explanation of this hyperpyrexia is that most of the animal heat in the body is supposed to be generated by muscular action; in such a lesion, the paralysis of all the limbs is usually more or less complete, and thus the muscular action that remains is confined to the movements of the heart, of the vessels, of the diaphragm, of the muscular coat of the intestines, and, if it has escaped paralysis, of the bladder. So high a temperature under such circumstances seems to prove that animal heat is derived from other sources besides muscular contraction—from the friction of the blood in the vessels, from the formation of all secretions, probably from every intellectual action, if not from the perception of the sensations from all the organs of special sense. If the lesion affect the very lowest portion of the cervical region, the innervation of the cardiac accelerator nerves of the sympathetic may be interfered with,

and from the function of the vagus being inefficiently antagonised by the influence of the ganglia of the heart itself, this organ beats slowly, or with irregularity and frequency. The vasomotors supplying the vessels of the head and face may be paralysed and paretic, and the congestive phenomena here apparent may be more conspicuous on one side than on the other.

A similar condition of the upper cervical cord gives time for no vasomotor phenomena; the paralysis of the diaphragm, as well as of the other muscles of respiration, causing either immediate or rapid dissolution.

In spasmodic spinal paralysis there are usually no vasomotor disturbances in the limbs.

In these crushing lesions of the cervical spine there are usually two conditions connected with the sympathetic system (besides the state of the temperature), the small pupil and priapism. After this kind of paralysis the pupil is not contracted, but is simply unable to dilate. Thus, when the patient lies with his head in shade, the pupils are as small as if they were exposed to bright light, but no smaller. Mr. Hutchinson regards the priapism under these circumstances as essentially paralytic and passive. The erectile tissue of the penis is unable to contract, and blood is pumped into it. Whenever it is present we may feel sure that vasomotor paralysis exists; and when it is extreme, the probability is great that the heart's action is vigorous. As regards temperature, the usual rule would be that cited above—(1)

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some coldness from shock (contraction of arteries); (2) in the greater number of cases vasomotor paralysis, and consequent rise of temperature; (3) after a time the temperature either becomes equalised, or the limbs affected are rather colder than in health. But exceptional cases occur of crushing of the cerebral spine, in which, at an early period, but after all shock has been recovered from, the surface of the paralysed region is decidedly cold, even though the colour of the cheeks and lips is rosy, and this, too, when the persistence of well-marked priaprism afforded evidence of vasomotor paralysis. The comparative coolness of paralysed limbs that have been abnormally hot in the stage of vasomotor paralysis may be explained by gradually decreasing heat action, by inactivity of paralysed muscles, possibly by a torpid state of cell-life unfavourable to the production of heat; and to this may be added the fact that very often the vasomotor paralysis passes away, the sympathetic ganglia taking on an independent action. But it is more difficult to explain coldness of paralysed limbs during the period of vasomotor paralysis. Such cases are rare, but they exist; and if even one such case had been carefully observed, there must be a physiological reason for the phenomenon.

In Mr. Le Gros Clark's case the temperature fell to 82° Fahr. The main factor in the explanation is the depressed condition of the heart. Vasomotor paralysis with good heart action is accompanied by rise of temperature, until such time as the function of vascular innervation is in these spinal-cord lesions

taken up by the ganglia themselves. But vasomotor paralysis, attended with very enfeebled action of the heart, is not accompanied by a rise of temperature, but, on the contrary, by a fall; and although, in the observations hitherto made, no explanation has been found why, in this case, the heart retains its power, in that, it does not, the lesion of the cord being equal in extent in the two cases, yet the difference lies in the fact, that so few observers have recorded the rupture or the preservation of the rami communicantes connecting the cord with the cervical and upper dorsal ganglia. Rupture of these connecting links between the spinal cord and the cardiac accelerator nerves brings into play the inhibitory action of the vagus, slows the heart, and thus enfeebles the circulation, causing coolness of the surface, even though the arteries may be in a condition of vasomotor paralysis.

The conclusions that may be drawn as to the connection of the sympathetic with lesion of the spinal cord are—

1. That most, if not all, the cases in which changes of temperature, oculo-pupillary phenomena, alterations in the movements, the secretions, and the vascular tone in the abdominal cavity through the splanchnic nerve, can be best explained by lesion of the sympathetic centres in the cord itself primarily.

2. That in all inflammatory spinal lesions it is very possible that the alterations of nutrition and the vasomotor congestion, the early stages of inflammation, may be derived from the ganglia outside the

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cord. The nutritive deficiencies necessarily associated with and dependent on vascular alterations may owe their origin to or may base their starting-point in the sympathetic centres of the cord, or, as in the case of descending lateral sclerosis, be the first effect of cutting off a strand or column of the cord from the higher regions of the brain, from which it is the normal conductor of voluntary impressions. In either case the vasomotor nerves play a certain part in the causation of the inflammatory state. But that in any more direct way lesion of any portion of the sympathetic system affects the spinal cord is yet unproven. Certainly the few observations in the case of progressive muscular atrophy that seem to imply any causative relation between the sympathetic ganglia and this lesion, are far outnumbered by the many instances in which there was no such relation, and in which the disease was unaccompanied by any ganglionic lesion, or by any symptom that could be referred to the sympathetic system.

Consult—

Eulenberg and Guttman, op. cit.
Leyden, op. cit.
Dr. Dowse, 'Lancet,' 1879, ii. 686.
Pierson, op. cit.
Chapin, op. cit.
Trousseau, 'Clinical Medicine.'
'Med. Times,' 1884, i. 980.
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Dr. Hackin, 'Lancet,' 1884, ii. 56.
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CHAPTER X.

SUNSTROKE. THE SYMPATHETIC IN HEMIPLEGIA. LESION OF NERVES. GENERAL PARALYSIS.

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In sunstroke, the primary condition is twofold—paralysis of the inhibitory heat-centre and paresis of the chief vasomotor centre in the medulla oblongata. The loss of tone in the small arterioles, and the consequent congestion, is especially seen in the lungs, and form an important element as to the peril of the patient. Hæmorrhages have been found in the ganglion cervicale supremum.

The symptoms depend on excessive heat. Perhaps when this heat is that of the sun, it is apt to affect the vasomotor centre in the medulla oblongata especially, by striking on the unguarded occiput and neck; but the same symptoms arise when there is no direct influence of the sun upon the person attacked. Even in this country we recognise not only grades in the intensity of this affection, but some variety in its forms. This variation has been formulated by Sir Joseph Fayrer under three divisions: 1. The syncopal form, heat-exhaustion, on which there is depression of nerve force and prostration of muscular power. The skin is pale, cold and moist, and the pulse is

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quick and feeble. Death may occur rapidly in the state of collapse from failure of the heart. 2. The asphyxial form, sunstroke proper. The brain and nerve centres, especially the respiratory, are overwhelmed by the sudden elevation of temperature, and respiration and circulation fail, the failure of the latter being probably due to the inhibitory influence of the vagus. Whether the coagulation of the cardiac myosine is an ante-mortem or a post-mortem change, the heart's action is brought to a close by the heat in the same manner, as it has been shown by Claude Bernard and Lauder-Brunton, that the effect of high temperature in animals is first to accelerate and finally to stop the heart, and especially the ventricles in a state of contraction. 2. The hyperpyrexial form, heat-fever, coming on by night as by day, in the shade as in the sunshine, and affecting persons whose nerve power has been depressed by a variety of causes—by fatigue, overcrowding, bad hygienic surroundings, dissipation, previous illness, &c. Fayrer's description of this form is peculiarly graphic: 'The temperature of the body rises to 108°–110° Fahr., or even higher. The brain, medulla and cord, the nerve-centres generally, and especially the respiratory, suffer from overstimulation followed by exhaustion. Respiration and circulation fail; there is dyspnœa with hurried gasping breathing, great restlessness, thirst and fever, frequent micturition and a pungent burning heat of the skin, which is sometimes dry, sometimes moist. The pulse varies; in some it is full and laboured, in others quick and jerking. The face, head and neck are

congested to lividity, and the carotid pulsations are visible. The pupils, contracted at first, may dilate widely before death. Delirium, convulsions, frequently epileptiform in character, coma, relaxation of the sphincters and suppression of urine come on, and are frequently the precursors of death, which is due to asphyxia. Recovery not unfrequently partially occurs, to be followed by relapse and death, or secondary consequences, the result of overheating, end in meningitis or cerebral changes, which may destroy life or intellect at a later period, or permanently compromise the whole health or that of some important function.'

The after symptoms, as detailed by Sir Ranald Martin, are of extreme interest, and they, as well as the more chronic and subdued influence of insolation, throw much light on the pathology of the disease, and confirm the opinion of Dr. Handfield Jones, that heat-apoplexy essentially consists in a functional paralysis of one or more great nervous centres.

These symptoms are:

1. Some mental depression with sense of weariness along the spine.

2. Impression of the sun being always shining on the person, with tinnitus aurium.

3. Distressing formication, sometimes accompanied by a peculiar and general eruption and desquamation of the skin.

4. Deafness, more or less severe, with impaired vision and inability to use the eyes in reading and writing.

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5. Various paralytic affections, more or less general, as hemiplegia and local palsies; as of the eyelids, cheeks or upper extremities, with and without loss of sensation. Heavy and difficult breathing.

6. Distressing hysterical states of the nervous system, with absence of self-control in laughing or crying, the paroxysms being followed by great prostration of nervous power.

7. Interruption to natural sleep with incapacity for any kind of business.

8. Impression of alarm on any sudden movement of the body, and upon the occurrence of sudden noises.

9. Sudden epileptiform seizures, without loss of consciousness, followed by great nervous prostration.

In India we are familiar, besides, with the acute sequelæ to sunstroke, as ardent fever with acute delirium, remittent and intermittent fever complicated with dysenteries, hepatic inflammations and congestions.

These statements need the most careful consideration, not only from the light they throw on all the nature of heat-apoplexy, but from that which they shed on the pathway of a multitude of nervous disorders. Dr. Handfield Jones remarks that any man of experience in the manifold disorders of jaded and exhausted nervous systems will recognise at once how close is the resemblance between the results of tropical heat and those produced by the ordinary causes in operation among the struggling multitudes of our large towns.

It may be observed—1. That the most abiding results of heat-apoplexy are almost all referable to impaired functional energy of the cerebro-spinal system. 2. That this impairment shows itself either in motor paralysis, sensory paralysis either of common or special sensation, hyper and dysæsthesiæ of the nerves of common and special sense, in debility, and undue excitability of the emotional centres, and in similar states of the cerebral hemispheres and spinal cord. 3. The earlier sequelæ appear as the well-known consequences of vasomotor nerve paralysis.

To those sequelæ of sunstroke may be added two others—the extreme sensitiveness of a patient who has once suffered from sunstroke to the rays of the sun or to excessive heat ever afterwards, and the effect exercised on them by alcohol. Both these symptoms, consecutive to the affection, point to the unstable condition of the great vasomotor centre in the medulla oblongata. The writer's case, already published, of recovery by hypodermic use of quinine, illustrates the theory of the vasomotor affection in the disease. In many cases it is enough to reduce the excessive heat of the body ; in others, it is necessary to rehabilitate (so to speak) the inhibitory heat-centre in the brain, and to give tone to those great organs that rule the vaso-motors ; and the chief centre, the one that controls all the vasomotors in the body, or at least that influences all the vasomotor centres in the spinal cord, lies within the medulla oblongata.

As paralysis on one side of the body from cerebral

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lesions will be either from cerebral hæmorrhage, embolism or thrombus with consecutive softening, softening without embolism or thrombus, and generally owning as its cause a diseased condition of cerebral arteries and narrowing of their lumen, syphilis of surface of brain or tumour in certain situation, it is easy to understand that the sympathetic phenomena likely to accrue from hemiplegia would be vasomotor only.

The difference of opinion that seems to have existed on the temperature of paralysed parts has depended, like the variations shown after spinal injuries, on the period the paralysis had existed when the thermometric observations were taken. Thus, as regards paralysed parts, Schmitz has observed a slight diminution of temperature; Bärensprung also, in four cases, found the temperature of the paralysed parts less in three cases, and a trifle higher in one case, than in the healthy part. Nothnagel found in the hollow of the hand of a paralytic arm a temperature of 3.6° Fahr. lower than in the healthy side. Such statements are quite unsatisfactory, as leaving unrecorded the period of the hemiplegia.

On the other hand, Folet, quoted by Wunderlich, made continuous observations on hemiplegic patients and came to the following conclusions:

1. In the immense majority of cases the commencement of the hemiplegia is accompanied by an increased temperature on the affected side; both sides are very seldom alike, and a diminished temperature on the diseased side is hardly ever noticed.

2. The rise of temperature varies between .54° and 1.62° Fahr., but seldom exceeds 1 8° Fahr.

3. The presence or absence of contractions has no influence on the thermometric results.

4. The thermometric difference may be greatly augmented by various primary causes.

5. The original cause of the hemiplegia has no effect upon the result.

6. Recovery from the paralysis tends to equalise the temperature again, and if the paralysis continues the height of the temperature varies greatly, and in one case may return to the normal in a few months, in others, may continue unequal for even years together.

7. Undoubtedly paralytic atrophy necessitates depression of temperature.

8. In an old hemiplegia, when the affected side exhibits a high temperature, and when the other side becomes paralysed at a later date, either the two sides become equalised in temperature, or the side last paralysed now becomes considerably hotter.

9. The general temperature of hemiplegic patients is not usually above the normal, but exhibits an average height of 98.6° Fahr., except in the last hours of life, when it generally rises.

10. Lepine states that he has found—

(*a*) That in a recent case of hemiplegia the paralysed limb is at first warmer than the healthy one, and on exposure to a certain amount of cold loses more heat than the sound one; with a still greater

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degree of cooling, however, it loses less heat than the other.

(*b*) On the other hand, in a very old case of hemiplegia, the paralysed limb appears colder than the other, but remains relatively warmer than the healthy one when exposed to cold, and with artificial heat becomes less warm than the sound limb, thus exhibiting less extensive fluctuation either upwards or downwards under external thermic influences.

There is no doubt, if hemiplegia arises from cerebral hæmorrhage, that Folet does not go far enough back for his observations in many cases. The first phenomena of cerebral hæmorrhage are those of collapse, though in some instances the pulse, respiration and temperature remain normal. But when these physiological effects are abnormal, as they often are, the face is pale, the pulse slow and laboured, 60, or under, the respiration shallow, the temperature in the axilla 96° or lower. In the second stage, the stage of reaction, the pulse and respiration quicken, and the temperature usually rises, for instance, to 101° or 103°. Shortly before death it may rise to 107° or more. These phenomena are general, and affect both sides of the body equally. As the stage of reaction subsides, in cases in which life is prolonged and hemiplegia is persistent, then the affected side may remain slightly warmer than the other for a considerable time, even two or three months, and the temperature here only falls below that of the healthy side in the chronic stages of the affection. Dr. Bastian's quotations from Bourneville on the temperature in cerebral hæmor-

rhage include all that is best known on the subject. Most of the views thus formulated can be verified by any hospital physician. He says: 'Looked at from the point of view of the variations in temperature that are to be met, Bourneville divides cases of apoplexy due to cerebral hæmorrhage into four categories: 1. Cases produced by very large cerebral hæmorrhages, or in which two or more bleedings rapidly succeed one another. Here death often occurs in an hour or two, and during this period the temperature of the body is decidedly lowered. 2. Cases in which death takes place in ten to twenty-four hours, and where the temperature is at first lowered, though the temporary depression is soon followed by a rapidly increasing rise of body heat. 3. Cases terminating in death at the end of a few days. Here then (*a*) are initial lowering of temperature, (*b*) a stationary period in which, after regaining its physiological standard, the temperature of the body undergoes slight oscillations above and below this point, and (*c*) an ascending period in which the temperature steadily rises. 4. Cases in which the patient recovers. Here we have an initial lowering of temperature, as in the other divisions, followed by a temporary rise, and succeeded by a pretty speedy return to the normal standard. Now taking as one type the cases which belong to the third category, I shall be able to lay before you the facts ascertained by Bourneville concerning the several thermometric stages and the modification of pulse and respiration which they entail, or which at all events accompany them. These

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details refer to temperatures taken in the rectum, and they have to be studied for each of the three periods —for the period of initial lowering, for the stationary period, and for the ascending period of body heat.

(*a*) Period of initial lowering. When the temperature is taken about a quarter of an hour after the outset of the apoplectic attack, it is usually found to be already below the normal standard (probably at about $97\frac{1}{4}°$), though there may be no notable alteration in the character of the pulse or respiration. Occasionally the fall of temperature takes place a little later, but in either case it may sink as low as 96° Fahr. Any recurrence or continuance of the bleeding into or upon the brain causes the temperature to remain for a longer period at this low point, or will again depress it should it have begun to rise. During this period, all the more familiar apoplectic symptoms are well marked—coma, resolution of limbs, stertor, with or without occasional vomiting and relaxation of the sphincters, are to be met with.

(*b*) Stationary period. In an hour or two, should the patient survive, we may begin to get more distinct evidences of the hemiplegic condition. The state of coma passes perhaps into one of stupor, in which the patient is less profoundly insensible. The alteration in temperature, however, now to be described, may still supervene, even where the coma continues without diminution. In this stage, then, the temperature returns to the normal $99\frac{2}{5}°$, or it may rise to 101°, and it continues to oscillate rather irregularly between these two points for two, three, or

four days. During this time the respiration is not sensibly altered in frequency, and the fluctuations of the pulse are slight and irregular, the rising and falling in the number of pulse-beats being by no means always harmonious with the variation in temperature.

Now in the most severe cases of apoplexy the patient dies in less than two hours—whilst the temperature is still low ; and when the patient is destined to die in from ten to twenty-four hours, the comparatively stationary period of body-heat just described is also absent, the primary depression of temperature being followed by a steady and continuous rise, till it reaches at the time of death some point ranging between 104° and 108°. On the other hand, in instances where the stationary period of body-heat is present, if the patient is destined to recover from the immediate effects of the already existing brain lesion, and if no visceral disease of an inflammatory nature (in the lungs for instance) should complicate the case, the temperature after two or three days of slight elevation sinks to the normal grade and there remains. Where already the brain lesion is such that the patient is likely to die in the course of a day or two more, the stationary period is followed by another change in the temperature of the body.

(*c*) Ascending period. A rise of temperature of a rapid and continuous character is just as unfavourable a sign, when it sets in after a stationary period as when it occurs immediately after the period of initial lowering.

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As a rule, death is then not far distant, and the temperature may rise, as before stated, to any point between 104° and 108°, the maximum being attained at the time of death, or very shortly afterwards. During this stage, too, the pulse and the respiration are much more constant in their character than they are during the earlier stage of the malady.

The pulse becomes small and very frequent—120 to 136 per minute. The respirations increase in frequency (48 to 64) and are also more laboured, noisy, and often complicated with laryngo-tracheal râles.

At the same time the extremities become dusky, especially on the paralysed side, whilst the face is red, swollen, and covered with an abundant clammy sweat. Finally, there is the most absolute muscular resolution in all parts of the body, even where more or less rigidity had previously existed, which continues till death closes the scene.

Bourneville found that, with the exception of a few altogether unusual cases, the period of initial lowering of temperature commonly met with in cerebral hæmorrhage is either absent or much less slightly marked, when we have to do with cerebral softening. But after the first two hours, in a case of softening, the temperature may suddenly rise to 102° or even 104°, though it soon again descends to the normal standard and afterwards exhibits altogether irregular oscillations—sometimes remaining the same for two days, and sometimes presenting evening or morning remissions of nearly 2°.

Amongst the exceptions to this order of events

in cerebral softening we have to include most instances in which the pons varolii is thus affected, this being a part of the brain which is known to contain most important vasomotor centres. The writer has seen a case of pons hæmorrhage with temperature of 110°. Again, in the so-called 'stationary period,' the oscillations of temperature are more regular and slighter in cases of cerebral hæmorrhage than in those of softening. Such a period lasts for a variable time in instances of cerebral softening, though, when one of these cases is about to prove fatal, the temperature-wave becomes characterised by an ascending period. Even here, however, in the majority of instances, the rise in temperature takes place more slowly than where we have to do with the terminal period of a case of cerebral hæmorrhage. It will be seen, therefore, that Folet's fifth axiom is not in accordance with experience, viz. that the original cause of the hemiplegia has no effect on the result, the fact being that the thermometric difference shown in cerebral hæmorrhage and cerebral softening are such as to enable the physician to make a differential diagnosis.

Although the paralysis in uræmic coma is not exclusively confined to one side, and cannot be termed hemiplegic, yet it is interesting to note the great thermometric differences in this condition, compared with cerebral hæmorrhage and softening. The temperature of the body begins to fall with the onset of uræmic coma, and continues to sink as long as this condition persists, so that it may fall as low as 90° Fahr.

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in fatal cases. There is no reason to believe that the sympathetic nerve or centres have anything to do with the condition of the pupil in hemiplegia from any cerebral cause.

In old cases of hemiplegia, even where there is no secondary descending degeneration of spinal cord and contraction, the paralysed leg, and less frequently the arm, may be mottled and œdematous, owing to a partial paresis of the vasomotors.

It is not just plain sailing to attempt an adequate explanation of the fall and rise of temperature in these cerebral lesions. It must be conceded that abnormally high temperature depends on a paralysis of a temperature-regulating centre, which inhibits the centres in the spinal cord, and which in the normal state controls the combustion of protoplasm. The high temperature that occurs after severe injuries to head and spinal cord, and even sometimes after fatigue, must be due to this paralytic pyrexia. In Mr. Teale's case, where the temperature varied for nine weeks from 108° Fahr. to 122° Fahr., the pyrexia was due to meningo-myelitis.

When, too, in experiments in the upper part of the cervical cord, or at the junction of the pons and medulla oblongata, Quineke found that division of the cervical cord caused high temperature, these pyrexial effects could be prevented by the administration of quinine, the quinine acting presumably by checking the combustion of protoplasm, as is seen by the diminished excretion of urea from the use of this drug. Over-exertion will cause raising of tempera-

ture, both by the extra combustion of tissue and by the fatigue consequent on it having a paralysing effect on the inhibitory heat-centre.

Of course all heat in the body must depend primarily on chemical action, and a regulating centre is one that controls this chemical action. If the centre be paralysed, combustion goes on in excess. This paralysis is, in the case of a chill, a reflex act, possibly by cutting off the blood-supply by vascular contraction.

2. In toxic conditions—as, for instance, in zymotic disease—it is caused by the circulation of blood containing microbes, or by the direct action of the morbid organism itself on the inhibitory heat-centre.

3. In insolation, the paralytic effect is produced by the rays of the sun, or by the heat of the external environment, acting mechanically. It is probable that this paralysis, whether caused by one of these modes or another, is increased by its own efforts, i.e. by the circulation of hot blood. This combustion of protoplasm is so closely connected with the vaso-motor system, that the effects of its abnormal increase, hyperpyrexia, may be considered in large measure as pertaining to the sympathetic. These effects are manifold. In the nervous system, confusion, delirium, coma may be met with; as connected with the heart, irregularity and paralysis. A granular degeneration of the muscles is one of the results; a diminution of the red corpuscles of the blood; a tendency to ecchymosis, and extravasation from the vessels; a fatty degeneration of the parenchymatous organs; an

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interference with the general nutrition of the body, caused partly by the loss sustained by the uncontrolled combustion, partly by difficulty of nutritive supply; and this latter cause may perhaps be somewhat brought about by the absence in hyperpyrexia of the pancreatic secretion, this absence preventing the absorption of fatty and starchy matters. From the same cause various modes of death occur, in which the high temperature acts fatally in very different ways. Thus—1. By increased cell-growth and its results, e.g. the ulceration of typhoid or tuberculosis in the bowels, the stuffing of the lungs in phthisis or in pneumonia, the blocking of the larynx in croup, &c.

2. By degeneration of the cardiac muscles, causing paralysis of heart and death by syncope.

3. By lethal diminution of the red corpuscles of the blood.

4. By the effects on general nutrition from deficient assimilation.

5. By interruption of the due proportions between endosmosis and exosmosis in the tissues, with special reference to the migration of white corpuscles.

6. Paresis of the pneumogastric and of the cardiac sympathetic, and so death by asphyxia or by syncope.

In these remarks on hyperpyrexia nothing has been said upon an important factor, viz. the derangement of the regulations by loss. This has been touched upon in considering the phenomena of fever.

In the view that heat is generated not solely by

the non-nitrogenous portion of the food, but by combustion of all the tissues in the body, the statements of Professor M. Foster are especially clear. He says: 'We may indeed at once affirm that the heat of the body is generated by the oxidation not of any particular substances, but of the tissues at large. Wherever metabolism of protoplasm is going on, heat is being set free. In growth and in repair, in the deposition of new material, in the transformation of lifeless pabulum into living tissue in the constructive metabolism of the body, heat may be undoubtedly to a certain extent absorbed and rendered latent; the energy of the combustion may be, in part at least, supplied by the heat present. But all this, and more than this, viz. the heat present in a potential form in the substances themselves, so built up into the tissues, is lost to the tissue during its destructive metabolism, so that the whole metabolism, the whole cycle of changes from the lifeless pabulum through the living tissue back to the lifeless products of vital action, is eminently a source of heat.'

Early in the century Mr. Earle published a paper entitled 'Cases and Observations illustrating the Influence of the Nervous System in regulating Animal Heat.'

Case 1.—A sailor had paralysis of the left arm after an injury. The temperature of the right (uninjured hand) was 92° Fahr. Of the left paralytic hand only 70° Fahr. After electricity for ten minutes it rose to 76° Fahr. On another occasion, the temperatures were:

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		Before electricity	After electricity
Paralysed limb	Hand	71°	77°
	Arm (at elbow)	80°	83½°
	Axilla	92°	93°
Healthy limb	Hand	92°	92°
	Arm (at elbow)	95°	95½°
	Axilla	96°	96°

Case 2.—Excision of portion of ulnar nerve (about one inch) for neuralgia. Two years after, the cleft between little and ring fingers of that hand = 57°, other parts of the hand 62°. Between little and ring fingers of other hand = 66°, other parts of the hand 62°, as before.

More recently Mr. Jonathan Hutchinson has published a series of accurate observations on temperature after injuries to nerves in the 'London Hospital Reports.' In his third case, one of injury to the median nerve supplying the fore-finger, there was a difference of 10° Fahr. between that and the adjacent thumb. In Case 4 there had been section of ulnar and median nerves of right hand, the latter partial only. Fourteen weeks after the accident the following remarkable difference in temperature was noted: Cleft between the little and ring fingers, left 83°; right (paralysed) 64° = 19° Fahr. difference. Twenty-one months after there was still 10° difference in this situation, and 5° between the right and left hands in the cleft between the fore and middle fingers, the paralysed parts being that much colder.

In Case 5, there was division of the ulnar nerve and vessels, and of the median nerve. Anæsthesia of the parts supplied. Inflammation of the tips of

three fingers unattended by sensation. Diminution of animal heat in all the parts paralysed. Increase of heat during inflammation, but still not up to the normal standard. Three weeks afterwards, the temperature of the hands was as follows:

Right fore-finger (paralysed) . . .	side 79°	front 78°
Left „ (sound) . . .	„ 90°	„ 87°
Right little finger (paralysed) . .	radial side 80°	ulnar side 79°
Left „ (sound) . .	„ 89°	„ 89°
Right ring-finger (paralysed, but inflamed)		front 89°
Left „ (sound)		„ 91°
Right middle finger (paralysed, but slightly inflamed) . .		87½°
Left „ (sound)		91°

Mr. Erichsen has recorded a case in which, twenty-one weeks after section of the ulnar nerve, the fourth cleft was 9° less than the corresponding part of the other hand. It is not at all easy to account for this depressed temperature after nerve section. Vaso-motor paralysis is followed at first by a rise of temperature, and although as time goes on, if the paralysis persist, the temperature gradually recurs to the normal point, or even a little below it, yet this effect of nerve section goes very much farther. This low temperature after section of nerve contrasts well with the temperature of an inflamed region in a hemiplegic patient.

Thus, Joseph Weir has been affected with left hemiplegia for ten years. His shirt caught on fire and he suffered extensive burns on the back, chiefly on the left side, and also of the left arm, but not in the axilla. No shock or pain at first. No pain until the inflammation had reached beyond the middle line of the back, and the patient had no constitutional

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symptoms until the suppuration became excessive. No pain was ever complained of on the hemiplegic side, though tactile sensibility was, if anything, increased in the hemiplegic arm and leg. The temperature of the burnt hemiplegic side was always higher than of the other.

Thus:

Left Axilla Evening temperature	Right Axilla Evening temperature
°	°
$100\frac{2}{5}$	$99\frac{2}{5}$
100	$98\frac{2}{5}$
100	$98\frac{2}{5}$
$100\frac{1}{5}$	99
100	$98\frac{2}{5}$
100	98
$99\frac{1}{5}$	$99\frac{3}{5}$
100	99
99	$97\frac{3}{5}$
101	$98\frac{2}{5}$

Mr. Hutchinson's hints at an explanation are well worth noting, though not wholly satisfactory. He says: 'It is to be noted that none of my cases were accurately observed soon after the accident, most of them not until the wound was healed. Many conditions make trials of temperature at early periods after the injury liable to mislead; such, for instance, as the fact that the vessels as well as nerves are usually cut, that inflammation is in progress at the wound, and that the part is protected by dressings. Respecting the state of things some weeks afterwards, I find no exception to the assertion that there is always a remarkable loss of heat in the part which has lost sensation. The amount of comparative loss

will vary with the external conditions, for it is a remarkable feature, as regards the heat of paralysed parts, that it is so much at the mercy of external influences. All my patients agreed in the statement that, in cold weather, the anæsthetic fingers become very blue and cold, and in many I was able to demonstrate this fact. In the warmest temperature, however, their heat could never be raised up to that of adjacent unparalysed parts. Nor does even the existence of active inflammation raise the part to the normal standard, although it much increases it. In Case 5, there were ten degrees of difference between the little finger in the hand from which the nerve-supply had been cut off and that of the other. The ring-finger was paralysed and inflamed, and its temperature was increased by inflammation, until it came within two degrees of that of the other hand, but still it did not quite reach it.

'In Case 4, we put the two hands into hot water (118°), and the result was that both hands gained in temperature about ten degrees, and that the difference between them remained as great as at first. It would appear, therefore, that while a paralysed part can be cooled to almost any extent, it cannot be raised by artificial heat beyond a certain point, and that point much below the maximum of its uninjured counterpart. To what are we to attribute this remarkable loss of power to sustain the normal temperature and to guard against the depressing effects of external cold? To say that it is the result of mere disease is unsatisfactory, for we find it just as marked, when

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one finger is contrasted with the others on the same hand, as when the whole hand is affected. Case 1 is almost crucial in this respect, for the man had but a finger and thumb, and used neither of them; yet the finger deprived of nerve influence registered ten degrees lower temperature than the thumb which yet enjoyed it.

'To allege that it is due to dilatation of the smaller arteries and the consequent slowness of the circulation is scarcely more satisfactory, since paralysis of the vasomotor nerve, and consequent dilatation of the vessels, is well known to lead to increase of temperature. Yet it is quite certain that the capillary circulation is in some way greatly disturbed.

'If you chill the part it becomes not merely pale but livid; if you warm it, it becomes not of a bright pink flesh tint, but of a peculiar dull brick-red colour. The most plausible conjecture seems to be, not that the nerve control over the vessels is at fault, but that the vis a fronte is itself much diminished.

'One way in which this must be reduced would, no doubt, be by the atrophy of those parts of the skin which endow it as an organ of tactile sensibility. No doubt but that, after a nerve of sensation has been long cut, you will find the papillæ of the skin wasted and shrunken. The nerves themselves will also no longer maintain their own nutrition, and thus a much diminished demand for blood will result.

'I cannot but think it probable, however, that there is something more than this, and that the endowments of every single cell in a part no longer connected by

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nerves with the great centres undergo an alteration. If we may suppose that the cells of a part no longer possess the same energy of growth and nutrition, no longer attract the blood, take from it what is needed and transmit it forward, then we can at one and the same time account for the loss of heat, the manifest slowness of the capillary circulation, and the occasional changes in structure which may be considered as lesions of nutrition. If such an hypothesis be admissible, we may perhaps be permitted to speak of paralysis of the vis a fronte of the circulation as the real cause of the loss of temperature after division of nerves.'

It seems certain, at any rate, that this depression of temperature is more or less independent of the vasomotors. A limb with its nerves cut, and thus separated from its nerve-centres in its motor, sensory and trophic relations, would be very imperfectly in a position for carrying on even a tolerable combustion of protoplasm. As a matter of fact, such a limb dies. In the cases above mentioned there was only incomplete nerve loss, only partial cutting off of the vascular supply. In spite, therefore, of somewhat dilated vessels from paralysis of the vasoconstrictors, there must have been necessarily but little combustion of tissues. Increased heat following vasomotor paralysis presupposes normal vis a tergo from the heart's action and a normal connection of the motor and sensory nerves with the nervous centres.

The conclusion of the matter seems then to be :

1. That in high temperatures, from some cause or

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other, reflex or direct, there is complete or partial paralysis of the inhibitory heat-centre.

2. That this inhibitory heat-centre is probably situated in the brain itself, certainly above the junction of the medulla oblongata with the pons; although Fischer places it in the anterior fibres of the cervical portion of the spinal cord.

3. The paralysis of this inhibitory heat-centre allows an abnormal combustion of protoplasm in all the tissues.

4. That one factor towards this increased combustion is found in the paretic state of the vasomotors, often coincident with paralysis of this inhibitory heat-centre taken in connection with an otherwise normal circulation. The overheated blood moreover exercises a paretic influence on the vasomotors.

5. That in injuries to the cervical spinal cord, the first thermometric effect may be a depressant one, from the influence of shock, but this is shortly followed by hyperpyrexia, because, from the position of the lesion, the function of the inhibitory heat-centre is in abeyance, and because of vasomotor paralysis.

6. That the hyperpyrexia gradually wanes in these cases if life is prolonged, partly from the fact that the cervical ganglia of the sympathetic take on an independent action and remedy the vasomotor paralysis.

7. That in cerebral hæmorrhage, after the first effect of shock has passed off, a considerably heightened temperature is the rule, and this because the crushing

of the brain by hæmorrhage destroys, or at least presses upon and renders inert, the inhibitory heat-centre.

8. That the coldness of a limb, partly separated from its attachments to its nerve-centre, has little to do with the condition of the vasomotors; although their paralysis and the consequent dilatation of vessels may lead to an abnormally rapid loss of heat, the coldness is mainly due to the fact that almost all contraction in the limb is put an end to by cutting off from it the influence of its nerve-centre.

The influence of the sympathetic on the nutrition of a part and on its circulation render it, to say the least, one of the chief factors in inflammatory disease of every organ of the body. The vasomotor supply of the cerebral and meningeal vessels brings the brain and its membranes into close relationship with the cervical ganglia. This is seen in general paralysis of the insane. The lesions found post mortem are many and various, but in all cases there may be found cerebro-meningeal hyperæmia that has often proceeded to emigration of leucocytes, distension of vessels, impediments (chiefly thrombotic) to the circulation, irritative overgrowths of the connective nuclei of the walls of the vessels, and probably also of the neuroglia. This is only a somewhat more modern expression of Calmeil's dictum, that the pathological lesion is chronic inflammation of the brain, especially of the superficial part of the convolutions, the grey substance and the meninges. In addition to the 'délire ambitieux'

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which is sometimes wanting, and which, occurring in a few other morbid states of brain, cannot be considered pathognomonic of general paralysis, early inequality of the pupils, a sense of fatigue that yet does not overcome the tendency to wakefulness, irritability of temper, slight paralytic phenomena of speech, of prehension and of locomotion, with great facility in the formation of bed-sores, would be some of the more remarkable symptoms of this condition. Not only does the inflammatory character of the disease connect it with the sympathetic, not only can the intermission of the phenomena be explained in no other way, but the inequality of the pupils has been thought by some to point to the same fact. This however is seldom, if ever, the case. The asymmetry of the pupils is due to the want of symmetry in the rate of progress of the central lesion; and depends on the irritation of the third nerve on the side of the brain first attacked. This irregularity is also found at times in chronic mania and melancholia. On examining, however, on one occasion all the subjects of general paralysis in the Bristol County Lunatic Asylum, a few cases were noted where the inequality of pupils was only occasional. Here it is probable that varying conditions of the sympathetic may have induced a phenomenon which would not be transitory if it depended on persistent structural lesions. But with reference to the exaltations and intermissions (never remissions) in general paralysis, it may be said with Vulpian: 'Que toutes les attaques apoplectiques de la paralysie générale ne sont pas

dues nécessairement à des congestions cérébrales, et que, parfois, elles peuvent être déterminées, au contraire, par de l'anémie de parties plus ou moins étendues de l'encéphale, et que cette anémie est alors le résultat de phénomènes vaso-constricteurs réflexes, provoqués par l'irritation dont les parties où siège l'inflammation sont atteintes.'

Consult—

Sir J. Fayrer, Quain's 'Dict. of Med.': 'Sunstroke.'
Wunderlich, op. cit.
Handfield Jones, op. cit.
'Lancet,' 1876, ii. 444.
Dr. Hughlings-Jackson, Reynolds' 'System of Medicine,' ii. 548.
Dr. Bastian, 'Paralysis from Brain Disease.'
Prof. M. Foster, op. cit.
Mr. Earle, 'Med. Chir. Trans.' 1816.
Mr. Hutchinson, 'London Hosp. Rep.' iii. 305, 321.
Vulpian, op. cit.

CHAPTER XI.

EPHIDROSIS.

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Unilateral ephidrosis

In diseases accompanied by pyrexia, there is no necessary connection between the state of the cutaneous vessels and the sweat-glands. In the third stage of ague the vessels seem in the same state as in the second, and yet sweating occurs. Probably in the normal state, fibres from the sympathetic exercise a moderating action on the secreting work of the sweat-glands; when these fibres are paralysed, hyperidrosis occurs.

Paralysis of the vasomotor centre in the medulla oblongata by injury causes decreased production of heat, depending on the fact that the medullary centre dominates the vessels all over the body; and that general dilatation of all the vessels produces a sluggishness in the movements of the blood in all parts of the body. Increased heat of blood cannot, therefore, depend on this general paresis of all the vasomotors, but must be due to affection of the controlling inhibitory heat centre or centres, that have been found by experiment to lie above the medulla oblongata. What is known about the influence of the sympathetic on sweating has been mainly observed

in cases of unilateral hyperidrosis. Eulenberg and Guttman have remarked that after section of the cervical sympathetic, in one case of the left sympathetic, there were very varicose and dilated vessels, which perhaps, when full, pressed on some of the sympathetic nerve elements, and so paralysed them; also that by galvanisation of the cervical sympathetic the secretion of perspiration in the arm is increased. They think that this is due to currents entering the brachial plexus or the spinal cord, and has nothing to do with the sympathetic proper. Unilateral ephidrosis is sometimes seen in exophthalmic goître. Schwabach records a case in which pressure on the cervical sympathetic was associated with heat and redness of the right side of the face, and unilateral sweating on the least exertion. In Séquin's case there was normal perspiration on the left side of the face, whilst the right side was absolutely dry, and here the right sympathetic was adherent to the sheath of the vessels. In Ebstein's case of unilateral sweating, sometimes hæmorrhagic, there were very dilated and varicose vessels in the ganglia of the affected side, and the inferior cervical ganglion especially presented brown dots and lines visible to the naked eye, and consisting of cavities filled with blood-corpuscles, and lined by a distinct endothelium. The walls were thick and contained many spindle-shaped nuclei; the vessels very dilated, sometimes varicose. The ganglion cells were pigmented; on some the nucleus was quite concealed by dark-coloured granules. On the right side the nerve cells presented similar

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appearances, but there were no cavities, and the vessels were much less dilated. Ebstein believed that transient interference with the function of the ganglion cells may have resulted from occasional temporary over-distension of the dilated vessels with blood, consequent on hindrances to the escape of venous blood. To such transient paralysis of the ganglion cells he ascribes the hæmatidrosis.

Analogous changes in the ganglion have been found by Lubimoff in several examples of various disorders, as puerperal fever, &c.; but they were much slighter than in Ebstein's case.

In Seeligmüller's case of a woman who had had right ephidrosis during the whole of life, and in whom all the symptoms of paralysis of the right cervical sympathetic were manifested, there was found after death sclerosis and fatty degeneration of the right cervical sympathetic. In a case in the Bristol Royal Infirmary, a woman, aged 46, showed the interesting association of symptoms of enlarged thyroid, palpitation, no exophthalmos, contraction of the left pupil only, and left ephidrosis; whilst in another case a difference of symptoms was seen, in that the ephidrosis was on the left side of the face, whilst the left pupil was larger than the right.

It has been already stated that nerve fibres have been traced directly into the secretory cells of the sweat glands, but that there is a difference of opinion as to whether the nerves that influence the secretion of sweat are spinal or sympathetic, and also as to the position of the sweat centre. According to one

observer, the sudoriferous nerve fibres for the anterior limbs run in the upper part of the thoracic portion of the sympathetic, and this view is confirmed by the experiments of Luchsinger, and still further by those of Vulpian, who found, however, that a feeble secretion of sweat could still be produced after section of the thoracic trunk. He noticed also that the sudoriferous nerve fibres, which arise directly from the spinal cord, were more numerous in the sciatic than in the brachial nerves.

It seems probable the cervical sympathetic cord contains but few fibres, if any, which excite the secretion of sweat, and the variations in its physiological condition act upon the sweat glands, only in a more or less indirect manner, by the modification of the capillary circulation, which in their turn influence the activity of the anatomical elements. The sweat-exciting nerves of the skin of the face are derived either from the sympathetic fibres which accompany the vertebral artery, and are ultimately derived from the inferior cervical ganglion, or else they come from portions of the sympathetic which proceed from the medulla oblongata and the pons. These fibres accompany the various cutaneous nerves; they are perhaps numerous in the cutaneous branches of the fifth, and experiment shows that when the facial nerve in man is paralysed and has lost its irritability, if pilocarpine is injected the sweat appears later on that side of the face than upon the other. Although no sweat centre is known with certainty, it has been thought by Adamkiewicz that

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the nervous apparatus which presides over this secretion has its probable origin at the surface of the brain, from whence nerve fibres pass to the secreting ganglia, situated probably in the anterior horns of spinal grey matter; that the secreting fibres leave the cord with the motor nerves, and are distributed to the same regions as these nerves. Dr. Saundby gives cases of sweating occurring only at certain periods of the day, in which this phenomenon was probably excited by reflex action, having indigestion as a starting point, or else it was set up by the toxic influence of some product of imperfect digestion circulating in the blood. One of the factors in night sweats, so common in many exhausting diseases, seems to be the slowing of the heart's action during sleep, leading to peripheral congestion, a condition not sufficient in itself to lead to sweating, but taking rank as an important auxiliary, whilst from the same cause the amount of carbonic acid is increased. The night sweats of phthisis were thought by Dr. Brunton to be occasionally due to the stimulation of the sweating centre by increased temperature. The same observer considered that the increase of carbonic acid in the blood stimulates the secretion of sweat, as shown in the cold sweat on the foreheads of dying persons.

Pathology seems to bear out the view of sweat-centres in the brain. In Dr. Ringer's case, there were right hemiplegia, hemi-anæsthesia, and hemiopia, the patient not being able to see to his paralysed side. There was unilateral sweating over

the right half of the scalp, face, neck, and chest, to a less extent over the right arm, and still less again over the right leg. This right side of the body was frequently quite moist, whilst the left side remained perfectly dry. The perspiration was more intense on the right side of the face than elsewhere. In this case, when the upper lip was shorn, the hair scarcely grew on the right half of the lip. The lesion was probably in and around the posterior end of the optic thalamus.

Dr. Russell records an interesting case, in which unilateral congestion and sweating occurred, only during epileptiform paroxysms, over the left side of the face, head, and neck. The flushing and perspiration were intense, but only during the fits. Dr. Russell doubted whether the stimulus, which usually contracts the vessels in the epileptic fit, had produced paralysis of the external vessels by having been rendered inhibitory, either through exaggeration of its power, according to the views of Mr. Lister, or through a morbid state of the nervous tissue on which it operated, according to the opinion of Dr. Handfield Jones, or whether an influence totally different in its character was concerned.

In another case in which the phenomena of epilepsy and chorea were curiously intermingled, but where the right side was affected first and mainly with the choreic movements, a very copious perspiration occurred over the left hand, and it was invariably observed that whenever the movements occurred with more

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severity than usual, the sweat poured off her left hand, the right hand being only moist.

Unilateral ephidrosis is not a very common phenomenon, but some interesting examples are on record. Thus Ollivier speaks of a young man, aged 21, who suffered from congenital and constant sweating of that part of the skin of the face which is innervated by the right superior maxillary branch of the fifth nerve, viz. the lower eyelid, the right half of the nose, the cheek and upper lip of the right side. On the bridge of the nose sweating extended a little beyond the middle line towards the left, evidently from the interlacing of the terminal filaments of the contiguous nerves of the two sides. The reaction was acid. Warmth, alcohol, exercise and especially mental emotion, increase the secretion. When the sweating is most active the skin becomes injected.

The patient's maternal grandfather suffered in the same way all his life, but did not die until 82. His mother's sister and her only daughter suffer (and this in the same nerve district), while her two sons are free.

Speaking of neuralgia, Professor von Pitha records a peculiar case. 'Another objective symptom, found in certain cases of neuralgia, is a local cold sweating at the seat of pain. I met with this symptom, he says, very strongly marked, in a case of neuralgia of the median nerve, occurring as a sequel to a gunshot wound in the person of a young officer, whose own father believed him to be simulating; but I verified the occurrence during each paroxysm of cold sweat-

ing in the palm of the hand, extending upwards along the course of the median.'

In a case of Dr. M'Call Anderson there was hemiplegia of the right side, anæsthesia of the left, and unilateral sweating of the left side. The patient, a man, aged 49, had very slight paralysis of the left side, with rigidity and increased patellar tendon reflex, partial left anæsthesia to touch, pain, and temperature.

There was probably a double lesion, affecting on the right side the optic thalamus, the peduncular tract in its ascent through the internal capsule, and the optic radiation of Gratiolet: on the left side, the corpus striatum and its immediate neighbourhood. Dr. Anderson considered the unilateral sweating to depend on lesion of the cervical sympathetic, or upon lesion in some part of the brain with which this ganglion is connected. The nature of the lesions was doubtless syphilitic gumma, as the symptoms were much relieved by antisyphilitic treatment.

Dr. Alexander Robertson disagreed with this view, and thought it was more likely that there was one lesion only and that in the pons: perhaps the sweating was due to this pons lesion, as the pons presides over vasomotor action; but as the patient suffered from pain in the neck, the sweating may have been due to disturbance of the cervical sympathetic.

Dr. Habran's case, a man, aged 34, suffered from unilateral sweating on the right side of the face. There was no neuralgia; violent emotion suppressed the secretion. It did not reach the shoulder, but

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ceased at the level of the clavicle and scapula: it occurred generally about three or four o'clock in the afternoon. It yielded to remedies.

Dr. Merewether Lewis records a case of ague with a prolonged sweating stage, in whom the sweat was limited to the right side of the body, and never passed the middle line. The temperature of the two sides was unequal, on one occasion being 104° in the right axilla, whilst it was only $100\frac{4}{5}$° in the left.

MM. Vulpian and Raymond's investigations on the innervation of the sudiporal glands were undertaken with a view of explaining the apparent discrepancy in the observations of other physiologists.

Several investigators concur in demonstrating that in the cat the division of the ganglionic chain of the abdominal sympathetic or of the sciatic nerve prevents any general excitant of perspiration from acting on the limb operated upon, while sweat may be excited by stimulation of the peripheral end of the divided nerve. It has been shown also that similar results may be obtained in the fore-limbs, by division of the upper part of the thoracic sympathetic, or of the brachial nerves.

But Dupuy (of Alfort) had observed that extirpation of the superior cervical ganglion in the horse caused redness of the conjunctiva, a considerable elevation of temperature at the base of the ear and the forehead, and sweating on the ears, the forehead, and the neck.

Claude Bernard observed similar effects from division of the cervical sympathetic on one side, and

found that if the peripheral end of the divided cord was stimulated, the perspiration ceased, the vessel became contracted and the temperature fell. These observations would seem to show that the paralysis of the sympathetic fibres for the sudiporal glands may lead to an over-activity of these glands of the face of the horse, whilst it arrests the action of those of the paw of the cat, and that the electrical excitation of these fibres arrests the secretion in one case and produces it in the other. To decide the question, MM. Vulpian and Raymond have repeated the experiment of Dupuy and Bernard, with a view of ascertaining whether there is not some point of the nervous system of the horse at which the same results can be obtained as in the cat. In these experiments they found that the section of the cervical sympathetic cord is followed, as Bernard stated, by an abundant production of sweat upon the different parts of the head and face, but they did not find that faradisation of the upper end of the cord caused constantly an arrest of the sweating. It only did so when it produced a considerable contraction of the vessels.

There are very few cases on record in which unilateral sweating has seemed to result from lesion of the cervical sympathetic.

In a case of Verneuil the superior cervical ganglia was compressed by a carcinomatous tumour in the neck, and there was not only contraction of pupil, but other symptoms showing that the vasomotor filaments were involved, elevation of tempera-

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ture, and increased secretion of perspiration on the whole of one side of the face. In other cases of unilateral sweating tenderness in the region of the ganglion suggested the idea that there might have been inflammation in or around it.

A man, aged 46, suffering from tabes dorsalis, had unilateral sweating of face and head, extending to the shoulder after each meal; no change in pupil, increased flow of saliva on affected side during the sweating. In two cases the sympathetic ganglia were found affected. Pierret thinks that the central origin of the sympathetic lies in the posterior vesicular columns and the tractus intermedio-lateralis of Clarke—a region very often sclerosed in tabes.

This poverty of pathological record in this region of the cervical sympathetic agrees well with experiment. In galvanisation of the peripheral end of a divided sciatic nerve, the stimulation produces no sweat when atropine has been injected, though vasomotor effects follow as usual. If a cat, in which the sciatic nerve has been divided on one side, be exposed to a high temperature in a heated chamber, the limb, the nerve of which has been divided, remains dry, while the feet of the other limbs sweat freely. This result shows that the sweating which is caused by exposure of the body to high temperature is brought about, not by a local action on the sweat glands, but by the agency of the central nervous system.

Further investigation is needed before the position of the sweat centre or centres can be determined.

Professor Foster thinks that it is probable that, like vasomotor centres, there exists a chain of sweat centres along the spinal cord, inhibited more or less by one such centre in the medulla oblongata, the pons, or even higher up in the cerebral cortex; that these centres are frequently stimulated by reflex excitation, as by indigestion, by sapid substances in the mouth, perhaps even by the impression of heat on the periphery; they are more usually excited in a direct manner by the circulation of hot blood, by abnormal proportions of carbonic acid in the blood, by certain emotions, and possibly by contact with the morbific elements of certain diseases; that vasomotor paresis in the sweat glands is a necessary accompaniment without which copious perspiration would be impossible. Professor Foster states that some drugs, as pilocarpine, which cause sweating, appear to produce their effect chiefly by a local action on the glands, since the action continues after the division of the nerves (though pilocarpine at least has, as well, some action on the nerve centres), and the antagonistic action of atropine is similarly local.

Perhaps the auxiliary importance of the vasomotors of the vessels of the sweat glands is seen in the conditions under which anidrosis, the absence of sweat, is met with. The small sweat glands may be congenitally absent, but, apart from this, anidrosis is particularly noticeable in certain chronic diseases of the skin, in ichthyosis, chronic eczema, and psoriasis, in elephantiasis Arabum, in scleroderma, and in

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myxœdema. In all these conditions the influence of the vasomotors on the vessels of the sweat glands is either absent or exceedingly small

Excessive sweating, hyperidrosis, is a morbid phenomenon—it may be general or local. The secretion may be normal in character, or decidedly acid, or very alkaline. It may be accompanied by fœtor. Dr. Pooley has written in a very interesting manner on this subject in the 'Ohio Med. Rev.' 1880–1, and says that the weight of evidence is such as to lead us to believe the hyperidrosis itself to be especially a neurosis of the sympathetic or vasomotor system. The statements given above go far to disprove this, except in so far as dilatation of vessels is a necessary concomitant. In certain forms of ague, in acute rheumatism, in phthisis, this excessive perspiration is seen ; but nothing met with in the present day comes at all near the sweating sickness which first visited England with the army of Henry of Richmond, afterwards Henry VII. The course was rapid, death often occurring in a few hours, and it was believed to be a contagious, inflammatory, rheumatic fever, with great disorder of the nervous system. Its fatal features are described by an old writer (Holinshed) thus: 'Suddenlie a deadlie burning sweat so assailed their bodies and distempered their blood with a most ardent heat, that scarce one amongst a hundred that sickened did escape with life; for all in maner, as soone as the sweat tooke them, or within a short time after, yeelded the ghost.' It was a violent inflammatory fever, which, after a short rigour, prostrated

the powers as with a blow; and amidst painful oppression at the stomach, headache, and lethargic stupor, suffused the whole body with a fœtid perspiration. All this took place in the course of a few hours, and the crisis was always over within the space of a day and night; this account suggests the idea of a morbid germ irritating the sweat centres in the nervous system. In 1506 a moderate epidemic of the same kind occurred in England. In July, 1517, there was the third visitation in London, which spread with great malignity all over England, and among the English at Calais, attaining its greatest violence in the sixth week. In 1528, the fourth outbreak of sweating sickness commenced in London, affecting the whole of England, and in the following year spreading universally all over Germany. In 1551 occurred the fifth visitation of this dire disease, in Shrewsbury; it gradually spread, 'with stinking mists,' all over England, and on July 9 reached London. The mortality was very considerable. Foreigners were not affected, but Englishmen in foreign countries sickened with this sweating sickness.

In an article on morbid sweating, M. Bouveret speaks of sweating of the leg, mentioned by Verneuil as a frequent sign of deep-seated varices, and even as a means of making a diagnosis. Parotidean ephidrosis, sweating over the region of the parotid gland, is a local sweating of reflex origin; it occurs only during mastication, and though met with where the skin of the face is perfectly healthy, is often associated with a wound over the parotid. Facial sweating is

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commonly reflex, and due to excitation of the nerves of taste. It is either unilateral or general, or it may be exactly limited to the region of the face supplied by the superior maxillary branch of the trigeminal nerve, and there frequently associated with neuralgia of that nerve. Von Graefe has seen four cases of palpebral ephidrosis, especially during effort or emotion. As has been already stated, this morbid sweating sometimes affects the whole of one side of the body. Habitual general sweating may be met with under various etiological conditions, and sometimes without much disturbance of general health. Thus, flushes of heat and sweating are not uncommon with women at the menopause. M. Liegois cites several cases of women who had been thus affected for several years.

Bromidrosis is simply sweating of an offensive odour, affecting the feet most often, but also, in a less degree, the axillæ and the neighbourhood of the anus. Much fœtor may, however, accompany profuse general perspiration. It depends sometimes on the chemical composition of the secretion, but more frequently on the admixture of unhealthy sebum.

Chromidrosis is rare; of the various colours, yellow is the most rarely met with, except when it occurs as a complication of jaundice. The next rarest is green; the commonest hues are black, brown and blue. The colouring matter in these three is probably identical, most likely indican, which, as it normally exists, is colourless, and occurs pathologically in human urine. The indican is believed to

be secreted by the sweat glands in a colourless state, and to be so acted upon by the air, as to become oxidised into blue, brown or black, as the case may be. But the blue may be due to the presence of the protosulphate of iron in the sweat. The red colouring matter of the urine (uro-erythine) has been found in the perspiration by Landerer; a blue colouring matter, allied to cyanine, has also been found in the sweat. As a rule, in chromidrosis, the flow is not constant, but appears suddenly, remains for a short time, and then disappears; it may come and go in this manner for weeks or months. It is frequently brought on by excitement, or emotion, or passion; fright is often mentioned as a cause, but it may appear without an exciting cause. As far as is known, it is never universal; various regions may be attacked, but it has been noticed more frequently upon the face, chest, abdomen, anus, hands and feet; the lower eyelids and cheeks seem to be its most favourite seat. Bouveret considers that chromidrosis most frequently appears among that set of symptoms that characterise hysteria. Blue sweat may appear on the feet, in the axillæ, the epigastrium, forehead, and cheeks, never on the ears. It may extend over large surfaces, or on little patches of the integument; the colour varies from blue to black, but is sometimes a deep violet; the colouring matter is produced under the influence of vasomotor disturbances, and its appearance is preceded by disorders of menstruation, or by local derangements of circulation or of sensibility. Various instances have

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been given in the chapter on Pigmentation. Dr. Hardy speaks of an epidemic at Brest of black discolouration of the surface of the eyelids; a black material, like soot, came away.

Ephidrosis cruenta, bloody sweat, has often been a matter of controversy; its very existence has been denied; it has been supposed to be the result of deception, and all the more that it is met with most usually in the neurasthenic. But Pooley, Bouveret, and M'Call Anderson all speak of it as a real abnormality; it occurs either in droplets or in the form of filiform jets. It is intermittent, proceeding by steps, and coincident with painful eruptions of the skin. Bouveret calls it a sort of hæmorrhagic hysteria; its rare occurrence may be judged of by M'Call Anderson's statement that it is seen once in eleven thousand cases. In his case, the parts implicated were the face, the arms, the front of the chest, and the legs. The hæmorrhage occurred from round erythematous patches of eruption, remarkable for their symmetry, one on the brow, one on the chin, one on each cheek, and four in a row on the front of each arm, two on each upper arm, and two on each forearm; a similar arrangement was observed in the sternum and on the legs; it was sudden in its invasion. Sometimes the exudation was like water at first, and changed into blood; at other times, especially on the face, the patches were at once covered with a complete dew of blood, which proceeded to actual bleeding. There was generally only one attack a day, but sometimes two separate portions of skin bled simultaneously.

This case seemed to be one of vicarious menstruation. The hæmorrhage came from the capillary system of the sweat-glands.

Dr. Hill, of Virginia, records the case of a boy, aged 4, the subject of malarial fever. Blood oozed from the face and neck during the sweating stage; the symptoms, like the ague, were relieved by febrifuges and quinine. Two months later fatal hæmorrhage occurred from the alimentary canal.

It may seem scarcely the place in a chapter on morbid sweating to say anything upon hemophilia or on purpura. But the same vascular condition may obtain in all three states. A paralysis of the vaso-constrictors is a factor common to all. Chemistry gives no assistance in explaining the causation of hemophilia: very little, if any, in a large class of purpuric cases, though, doubtless, at least one form of purpura may depend on chemical changes in the blood. It is a suggestive fact that hæmorrhagic purpura may depend on emotional causes. Thus a case has been recorded by Valdés Peres: A well-nourished boy, aged 16, of good family and personal history, received a severe fright, and on the next day had to leave work from an attack of epistaxis. The next morning he was covered with red spots, but felt somewhat better, and again went to work. Epistaxis again occurred and was followed later by copious hæmorrhages from the anus, urethra, conjunctiva, ears and nose. On the fifth day of attack he was admitted into Guy's Hospital in a state of profound exhaustion; he was covered with

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red patches of purpura, ranging in size from a lentil to sixpence or a shilling. Hæmorrhage from the mucous surfaces still continued, and the urine and stools were full of blood.

This view of the implication of the sympathetic in purpura was also brought before the profession in an interesting paper by Dr. Magee Finny. His view of the pathology of purpura is that the nervous system is primarily at fault, and that, through the influence of the vasomotor system, the blood and the capillaries are secondarily affected. He bases his views on the following reasons:

1. The majority of cases of purpura present a history pointing to severe nerve waste, such as from over-exercise, fatigue and mental emotion.

2. The exhaustion is a prominent symptom all through the attack.

3. The analogy which exists in the acknowledged influence of the sympathetic nerve over cutaneous eruptions, and the altered pigmentation in pregnancy, leucoderma and morbus Addisonii.

4. The symmetry of the eruption, and the rapid manner in which, at times, it appears and disappears.

5. The close connections between disease of the cerebro-spinal centres and purpura, such as are manifested in that malignant purpuric fever, or cerebro-spinal fever, which, as an epidemic, visited Dublin in 1868–9.

Dr. Stephen Mackenzie, Dr. Foster, and others expressed similar views at the meeting of the British Medical Association at Liverpool.

Purpura has been seen after extirpation of sympathetic ganglia and heightening of arterial tension by ligature of the aorta.

It is well to say, in reference to this theory, that there is apparently only one form of purpura that owns a neurotic origin, and also that, even in this form, the condition affecting the vasomotor ganglia is unexplained.

Although it must be conceded that the rôle of the sympathetic in idrosis and its morbid development is secondary in importance, though absolutely necessary, there is a temptation to magnify it when the secretion of sweat is unilateral. In this condition it must be conceded that, if the exciting irritation of the sweat-centres be direct, whether it be heat, malarial, or other general toxic agent, emotion, &c., it would be more probable that the secretion would be universal; but it is not difficult to understand that the cervical ganglia of one side only should be sufficiently affected to induce paresis of vasomotors. If this is so, the excitation of sweat-centres could only take effect on that side, because the vascular condition would not be sufficiently paretic on the other. If, as is so common, the existing influence is a reflex one, such as indigestion, it is quite in analogy with other reflex irritations that the extent of implicated region bears no proportion to the exciting cause, the very localised regions of pigmentation depending on semilunar irritation, the peculiar unilateral spasm or contraction, or the unilateral sensory affections that may be due sometimes to a morbid condition of one ovary,

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sometimes of both, sometimes of the uterus itself, the renal congestion affecting both kidneys, and evidenced by albuminuria, in cases of strangulated hernia, where the bowel is involved. The case recorded by the writer, in which pressure on the left humerus caused vomiting, presumably from irritation of both phrenics, and still more the influence of various emotions and other causes in inducing unilateral chorea—these, and other examples that could be easily multiplied, are proofs that unilateral ephidrosis might depend on general irritation of sweat-centres, or irritation of sweat-centre of one side, of central or bilateral reflex irritation, or of the same condition on one side only. But when, in addition to some one of these causes, there is found tenderness over the cervical ganglia on one side, or when, post mortem, the vessels of these organs are found extremely varicose, or the ganglia otherwise diseased or markedly compressed by a malignant tumour, a condition is met with that materially determines the unilateral position of the phenomenon.

Thus, in 1875, a case was recorded in Virchow's 'Arch.' by Ebstein. The subject was an anæmic man, sixty years of age, and the unilateral sweating affected the left side of the head and trunk and left arm; the affected parts were not reddened, nor was there any alteration in the pupil. At first the sweating only attended some severe anginal seizures, but afterwards it recurred on any considerable exertion, even on walking up and down a room. Examination of the sympathetic ganglia showed that those of the

left side, especially the lower cervical ganglia, presented brown dots and lines visible to the naked eye, and consisting of cavities filled with blood-corpuscles, and lined by a distinct endothelium. The walls were thick and contained many spindle-shaped nuclei. The vessels were dilated, sometimes varicose. The ganglion cells were pigmented, and on some the nucleus was quite concealed by dark-coloured granules.

Ebstein believes that transient interference with the function of the ganglion cells may have resulted from occasional temporary over-distension of the dilated vessels with blood, consequent on hindrances to the escape of the venous blood.

Looking, too, at the highway by which reflex irritation would reach sweat-centre, it must travel by way of spinal cord, and in its course upward may, more easily than not, involve one of the numerous vasomotor centres to be found in each segment of the cord. That it does not affect them all sometimes is matter of observation; that it affects all those of one side is borne out by cases already given; but of these vasomotor centres the most important are those in immediate connection with the superior cervical ganglia, ruling, as they do, the vascular condition of the head and face; and it is probable that these centres are very frequently affected by the same exciting cause as forms the eisodic influence in what may be called the arc of perspiration. Taking for the moment this secretion as influenced by reflex irritation, there are two reflex arcs that take part,

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necessarily, in the process, one composed of the nerve course of the original excitation, travelling up to sweat-centre, and reflected down through sweat-nerves; the other composed of the same nerve course of original excitation, affecting either all the vasomotor centres or those of one side only, or perhaps several, or even one only, of one side, the reflected influence of which leads to vasomotor paresis, and so to the vascular dilatation, which is a necessary element in the production of the secretion.

Here then, as in all other secretions, the cerebro-spinal system and the sympathetic manifest a mutual dependence, the vasomotors allowing a vascular dilatation without which the blood-supply for the formation of the secretion would be insufficient; the cerebro-spinal sweat-centre, stimulated by heat or other irritation, direct or reflex, affords the force for exciting the glands, and influencing the chemical change in them. The inhibitory action exercised over the secretion of sweat by the sympathetic is merely an expression of the fact that in the normal state the vascular tone of the vessels of the sweat-glands affords blood enough for their nutrition, and, perhaps, for an amount of secretion scarcely appreciable. It requires a paretic condition of the vasomotors before that fulness of vessel in the glands is reached, without which excessive secretion is impossible.

Consult—

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CHAPTER XII.

ANGINA PECTORIS.

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THE symptoms of angina pectoris are seen under very various pathological conditions. The most pathognomonic symptoms are substernal pain, the feeling of anxiety, the disturbance of the heart's action. The pain has its origin in the cardiac nerve plexus, and persists even if the vagus be divided, proving that the sympathetic cardiac nerves contain sensory fibres. The pain is looked upon as the most important phenomenon, and the group of symptoms is considered by some as a neuralgia of the heart. But it is more than this. Cases are met with with a slow feeble pulse, some faintness, a feeling of anxiety, and a sense of impending dissolution; without pain, or at least without pain for a long time. In such cases, if not cured, the attacks become more frequent and intense, and sooner or later pain will be added.

The communication between the cardiac plexus and the anterior division of the four upper cervical and first dorsal nerves explains the transmission of pain to the regions supplied by the cervical nerves; and as the first dorsal nerve forms part of the lower

end of the brachial plexus, and as there are freer anastomoses of nerves on the left side than on the right, the reason of the sense of pain down the left arm is apparent. This is not the only instance of the connection of the brachial plexus with other nerves. The nerves of this plexus may form, for instance, the eisodic portions of an arc of which the phrenics are the exodic nerves, and may thus be the starting-point of eructation or of vomiting. The conditions under which angina pectoris occurs may be said to follow an anatomical distribution. According to circumstances, the automatic ganglia of the heart may be irritated or paralysed; the inhibitory action of the vagus may be increased by irritation: the cardiac nerves may be paralysed; the vasomotor nervous system may be so influenced as to induce change in the tone of the vessels, and consequent change of blood-pressure, with perhaps some affection of the depressor nerve of Ludwig. The influence of this depressor nerve is felt in a large number of diseases.

On section of this nerve irritation of the peripheral end has no effect; but irritation of the central end causes pain, lowers the pressure of blood in the arteries, arrests the respiration and retards the heart. As to the mechanism of the depression, it is a reflex action exercised on the splanchnic vasomotor nerves, producing relaxation of the intestinal vessels; thereby a large way is opened to the passage of blood from the arteries into the veins, and pressure is lowered. It is not the activity of the splanchnic vasomotor nerves, but the reflex suspension of their

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activity that is obtained by the irritation of the depressor nerve.

The cardiac reflexes are constantly being met with. When one excites a sensory nerve, or one of the posterior spinal roots, there is generally observed a passing lowering of pressure. Brusque percussion of the abdomen may arrest the heart's action. Simply touching the peritoneum will do this, when it is inflamed by exposure to the air. In many cases of peritonitis the reflex action on the circulation is remarkable. All nervous action which lessens the movement of the heart is transmitted to it by the vagus.

In most cases of angina pectoris the sympathetic is primarily or secondarily affected. The pathological anatomy is very variable. Cardiac lesions may be present, sometimes of the cardiac ganglia themselves —hyperæmia, interstitial inflammation, hyperplasia of connective tissue, and fatty pigmental degeneration ; in some cases destruction of ganglion-cells and caseous infiltration of connective tissue. Such lesions are only exceptionally seen in angina. These ganglia are more usually affected by any lesion that deprives them of their proper blood-supply, as narrowing of the coronary arteries from disease. This coronary lesion, however, often exists without angina, and angina without coronary lesion.

Pressure by diseased glands on the cardiacus magnus, and on a branch of the vagus, has been seen. Rokitanski saw the right phrenic and the cardiacus magnus involved in a dark blue hard knot,

which also implicated the descending branch of the left vagus. Lancereaux has seen congestion and inflammation of the cardiac plexus, and Seeligmüller hyperplasia of the connective tissue elements in the same plexus.

As to the pain, 'Goltz has shown that the sympathetic nerves of the heart contain sensory fibres. Moreover, the analogy in the character of the pain of angina pectoris with that which is caused by irritation of other parts of the sympathetic, as in colic of the intestines, of the gall ducts or the ureters, would lead us to suppose it might be of sympathetic origin.' 'Eichwald has supposed that in cases in which, at first, there is a full slow hard pulse, there is an irritable state of the vagus, while in cases of rapid intermittent pulse there is rather a semi-paralytic state. A certain form of angina is met with occurring with diseases of the abdominal viscera, which may be considered as a reflex neurosis of the vagus, from its similarity to the results of the experiments of Goltz.

'By the excitation or paralysis above alluded to of the vasomotor filaments throughout the body a state of arterial spasm or of relaxation may be produced, which can, secondarily, powerfully affect the heart by altering suddenly the blood-pressure and amount of work the heart has to do. Cohen long ago advanced the theory that certain cases of angina pectoris are connected with the vasomotor nerves. Later, Landois and Nothnagel described an angina pectoris vasomotoria, which they referred to a general arterial

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spasm, often produced reflexively, especially by exposure to cold. It is accompanied by marked pallor, lividity and coldness of the extremities, and is successfully combatted by warm applications and friction.'

It is not possible to say that pain is the essence of angina pectoris, as it is of hemicrania. It is pain plus the sensation of impending death, a sensation never felt in pure neuralgia. If these phenomena occur in middle life, it is probable that they are associated with some organic change in the heart itself or in the blood-vessels.

The sensory impressions in man are carried up by way of cardiac plexus and sympathetic fibres to the first dorsal and all the cervical ganglia; and thence through the cord to the brain. It is possible also that the sensation of pain is partly transmitted by way of the vagus to the medulla oblongata and the brain, especially as in the frog sensory impressions seem to be conducted upwards by a special branch of the vagus. The pain most usually depends on irritation of the sensory nerve-terminations in the walls of the heart itself, probably from spasmodic contraction of the cardiac muscle. Gaskell's observations have shown that when the vitality of the cardiac muscle is impaired by exhaustion, by injury, by mal-nutrition, the cardiac muscle loses its force of rapid contraction, and contracts with a prolonged tonic contraction in the same way as unstriped muscle. And clinical experience has shown that, during the paroxysm of angina pectoris, the systemic arterial tension is in many cases very notably increased. In diseases, too, of the

coronary vessels, the degenerative process sometimes involves the coronary nerves, and under such circumstances, as Dr. Byron Bramwell remarks, the terminal nerve fibres in the cardiac walls would be in an unusually irritable condition.

This observer suggests a sequence of events in the paroxysm that is well worthy attention: 1. The blood-pressure in the systemic arterial circulation is suddenly increased, either as the result of changes arising in the central nervous system (vasomotor centre) or in consequence of some external condition (sudden effort, exposure to cold, mental agitation, &c.) or of reflex impulse arising within the body. The attack may arise independently of any sudden increase of the peripheral resistance. 2. In consequence of the sudden increase of the peripheral resistance, the left ventricle, or the portion of it that from any cause has become weakened, is thrown into a temporary condition of spasm or cramp, which is attended with severe pain. 3. This irritation of the terminal branches of the cardiac nerves is reflected, viâ the sympathetic branches of the cardiac plexus and the spinal cord, to other parts of the periphery. The left arm is specially involved in this radiation of pain, because the terminal nerves that are compressed are those of the left ventricle.

When the pain is radiated to the right arm, it either is explained by the peripheral irritation having been sufficiently severe to pass over to the opposite side of the spinal cord, or by supposing that some of the fibres of the right ventricle have also become

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affected, and the nerve-terminations in the walls of that cavity have become irritated, or that the primary seat of the irritation is outside the heart in the coronary plexus. It has been supposed by some that the paroxysm of angina pectoris can be produced by a sudden diminution of the blood-supply to the heart itself, the coronary arteries sharing in the general vascular spasm which is the cause of the increased arterial tension that is the exciting cause of the attack.

Or the primary lesion may be extracardiac, either from implication of the branches of the cardiac plexus from lesion of the base of the aorta, or from direct irritation of the same branches in pericarditis. Under such circumstances the degenerative changes would probably extend downwards to the peripheral terminations of the cardiac nerves in the heart, and the partially degenerated nerves might be more irritable than in health. If this occurred, cardiac spasms would be very likely to cause an attack of angina pectoris. Even when this is not seen, degenerative changes may occur in the coronary plexus, either dependent on lesion of the coronary arteries or not.

In other cases of angina pectoris, the primary lesion is possibly situated in the nerve-centres. This is probably the cause of the angina, like attacks that are sometimes met with in hysterical women. (The writer has met with this condition in women to whom the term 'hysterical' ought not to be applied. Emotional, fearful, easily startled, neurasthenic either from the surroundings of their life or from inherited tendency, but laborious, practical, true, they differ from the

class of women with hysterical physiognomy, selfish habits, self-concentrated minds, more or less useless in the world.) This form is rather a pseudo than a true angina pectoris. The various forms of pseudo-angina may be met with in both sexes. They are seldom associated with the sensation of impending dissolution, and Dr. Byron Bramwell is probably right in calling them neuralgiæ of the heart. The writer thinks that this pseudo-angina is the sole cardiac neuralgia met with, and that the agonising pain in so-called 'gout in the stomach' is associated with organic cardiac changes, and more nearly approximates to true angina.

Landouzi considers this pseudo-angina may often be diagnosed by the irregularity of the occasioning cause. In true angina the same occasioning cause invariably produces the same result; but this is not the case in pseudo-angina. Landouzi believes that true angina pectoris cannot be regarded as a curable condition.

The views held on the nerves affected have been very various. Most frequently the affection has been considered as neuralgia of the cardiac nerves; by Trousseau it was looked upon as an epileptiform neuralgia, by Romberg and by Friedreich as hyperæsthesia of the cardiac plexus; Binswanger, Nothnagel, Lustig, Halbert and others speak of attacks of angina pectoris that may become developed into true vaso-motor epilepsy. Other authors, again, have pointed to the other symptoms beside those of a neuralgic character, and more particularly to the changes in the

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heart's action during the attacks. Thus Stokes looked upon it as a transient increase of an already existing weakness of the heart, consequent on fatty degeneration of its tissue coexistent with hyperæsthesia. Bamberger, in opposition to Stokes, speaks of it as increased action of the heart, hyperkinesis combined with hyperæsthesia; and Dusch looks upon it as hyperæsthesia with spasm of the heart. Eulenberg agrees with those writers who do not consider angina pectoris as a pure hyperæsthesia of the cardiac plexus, but who assume also implication of some of the motor nerves of the heart; but he thinks it not yet possible to determine the individual nerves themselves diseased, the functional alteration of which produces the attacks. Eichwald is of opinion that the steno-cardiac attack is consequent on actual arrest of the action of the heart by a mechanical impediment; and that the pain results from an endeavour of the heart to overcome that impediment, in the same manner that over-exercise of the voluntary muscles produces a feeling of pain. But it is opposed to observation that over-action of the heart with a view of overcoming an impediment leads to pain of this nature—as is seen in some conditions of aortic disease, nor is it met with in diminished action from fatty degeneration.

The lesions, in connection with which the phenomena of angina pectoris have been found are very various. Ossification of the coronary arteries has been pretty frequently met with, and in cases of this affection it has been sometimes proved, and in other cases it may be fairly assumed, that the coronary

nerves, so closely connected with the arterial branches, have been involved. The difference of opinion and of nomenclature of this affection is due to a difference in nature. There is a form of true angina that is not associated with high tension in the vessels. It may result from the implication of a cardiac nerve in some lesion of artery, as has been described above. It is not improbable that, without so definite a lesion, these anginal phenomena may be the consequence of a morbid sensitiveness of nerve, depending on a depressed tone of the whole system; that this is so is rendered likely, partly by the ease to all symptoms from the exhibition of morphia, partly from the advantageous use of quinine or of arsenic in preventing the recurrence of the paroxysms. Usually in this form there is no sign of disease of the heart or great vessels. It is a neuralgia, and, like neuralgia in other parts of the body, it may be induced by lesion of nerve fibrile, by reflex irritation from a distant part, and from constitutional debility of system. In such cases the inhalation of nitrite of amyl affords no relief. There is no high arterial tension; there is no great spasm of the cardiac muscles.

When the close union, not only of the vasomotor nerves, but of the minute vasomotor ganglia, is remembered, in the external and in the internal coats of the arteries, it is a matter of certainty that ossification or great atheroma of artery must lead in various portions of the vessel to destruction of the minute ganglia, and consequent loss of their action, whatever

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that may be, on the smaller nerve filaments in connection with them. It may be that each section of vessels depends for its power of contraction on the influence of the small ganglia in its coats, this influence in health being inhibited and regulated by the ganglia higher up on the trunk of the vessel, and these in turn by the ganglia of the sympathetic, by the centres in the spinal cord, and all by the main centre in the medulla oblongata. But whether or no, the ossification of an arterial branch cuts off vasomotor influence from the parts of the vessel below the lesion—influence, that is, not only of the special small ganglia involved in the lesion, but ipso facto of all vasomotor centres above it. It is therefore not remarkable that so much is made of the association of angina pectoris with ossification of coronary arteries.

Perhaps next in importance is endarteritis, or aneurismal dilatation of the base of the aorta. This can hardly occur without implicating the cardiac plexus that lies on the root of the aorta. Examples of this have been recorded by Lancereaux. In one case, a patient, 45 years old, who during life had the usual symptoms of angina pectoris, died in one such attack. At the post-mortem examination, besides narrowing of the coronary arteries, and alterations in the aorta at that part on which the cardiac plexus lies, vascularity of the latter was also found; some of its fasciculi being enveloped in exudation, and their external sheath thickened. On microscopic examination, an extensive accumulation of round nuclei was found, which separated the nerve

fibres and compressed them; the contents of the nerve sheaths had a greyish hue and a granulated appearance. In two other cases of angina pectoris Lancereaux found alterations in the aorta at the same spot and of similar character, connected also with considerable narrowing of the coronary arteries, so that, according to his report, possibly an analogous affection of the cardiac plexus may have existed, as in the former case, but the plexus was not, however, examined.

A similar condition was met with in a patient at Bristol Royal Infirmary. Here, as in other cases, the lesions were somewhat multiform. The coronary arteries were healthy; but the left ventricle was hypertrophied, with disease both of the mitral and aortic orifices; but the important lesion was a pouch-like dilatation of the ascending aorta near the heart, involving the cardiac plexus.

Rarely are the symptoms of angina pectoris the result of implication of nerves and ganglia in pericarditis, but instances have occurred. The phenomena here would be complex; the paroxysms of angina pectoris occurring and passing away, and instead of the patient being left quiet and exhausted he will be restless, with high temperature, with precordial oppression, often with delirium. The connective tissue of the cardiac ganglia and around the nerves is hyperplasic; some nerve-tubes become strangled and their myeline sheath ruptured, and transformed into an amorphous fatty mass. The cardiac muscles are often fatty and degenerated.

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The valves, and especially the aortic valve, show every form of injury and disease. All forms of cardiac lesion may be accompanied with the symptoms of angina pectoris, but the conditions most usually met with are those which show some want of nutrition of the cardiac walls. The instances are few of true angina pectoris in which the heart is found quite healthy; nor does a description of the morbid change in the heart and vessels include the pathology or even the pathological anatomy of angina pectoris. Lesions sometimes found, at other times presumed to be present from a consideration of the symptoms, in the sympathetic cardiac nerves and in the sympathetic ganglia may be at the foundation of the phenomena.

Certain jars to the spinal cord, the consequence of accidents, may give rise to symptoms exceedingly similar. Such cases are not common, and when met with are not invariably free from previously existing lesions of the heart. In a middle-aged lady of weak health, whose heart was always considered weak, but more from imperfect innervation than from any structural change in the cardiac muscles or the valves, the writer found, for some months after she had been upset in a wheel-chair, a series of symptoms that could hardly be distinguished from those of angina—palpitation, precordial oppression, pain in the left arm, sense of impending dissolution. It was at least possible that the centres in the spinal cord, with which the first dorsal and third cervical ganglia are connected, had been jarred by the acci-

dent, and their nutrition thereby modified in a morbid sense. Nor is this alone met with. Lesions in the course of the vagus—tumours pressing upon it for instance, or involving it in their structure—may give rise to the phenomena, and probably also lesions affecting the nucleus of the vagus, such as modification of its nutrition from diseased vessels, &c. A similar lesion, too, may involve the important vaso-motor centre in the medulla oblongata; this also may depend on impaired nutrition of the centre from imperfect blood-supply, or from the pressure of tumour in its immediate neighbourhood. The pathological anatomy is yet to be discovered of the influence on nerve centres by emotion. Whatever it be, it must be the same as the influence of centripetal irritation. Both this and emotion probably act by similar modification in the nutrition of the centre, either by temporarily inhibiting the relations of interchange between cell and vessel by abnormally constricting the latter, or by the opposite paretic effect on vessel, which would likewise interfere with nutrition.

The sources, too, of this centripetal irritation will have their own pathological anatomy, various forms of indigestion for instance, or the morbid lesions of gout.

One more point suggests itself, and that in connection with the action of chloride of barium. This drug is found to act on the unstriped muscular tissue of the heart, and not by way of influence on the sympathetic system. If the local application of this salt, in diluted solution, to the heart in situ produces local spasm at the point of application, it may be

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that this spasm can be induced by bringing to the heart through the circulation various morbid elements that may act there specifically on the muscular tissue of the heart; that therefore the muscular tissue may itself be the seat of lesions without any implication of the various nerves that innervate the heart. It has been well remarked that the primary disturbance which gives rise to reflex angina pectoris is usually seated in the course of the pneumogastric nerves. The distinguished surgeon, Mr. Hilton, was attacked three years before his death with severe angina pectoris—so severe that the cramping thoracic pain would not allow him to walk many steps, and he was obliged to have a cab to go a distance of 100 yards. This pain, after causing great suffering for several months, passed away, and then very gradually during the next two years the symptoms of cancer of the stomach developed themselves, The heart and great vessels were found post mortem to be entirely free from all morbid changes. Dr. Moxon says 'that not only may the gastric branches of the pneumogastric thus influence the cardiac nerves, and give rise to the special pain of neuralgic heartache; but, on the other hand, I have met with some remarkable cases, in which aortic disease has been associated with abdominal pain in the hepatic region of a neuralgic character, and in its mode of onset and conditions of relief behaving just like angina pectoris. Nor is it only the sensory fibres that thus vicariously suffer. A lady, 50 years of age, has been for three years under my observation with no other signs of organic disease

than a weak heart and feeble pulse. This lady is reduced to a most sedentary mode of living by the fact that if she tries to walk but a few yards she is suddenly attacked with vomiting. Heberden, in his description of angina pectoris, mentions that some of his patients, who persevered in walking in spite of their pain, were attacked by vomiting. In this lady the vomiting appears without the preceding pain; the onset of gastric disturbance without previous nausea is as sudden and surprising to her as is the pain which seizes the chest in angina.

In observations on morbid conditions more or less under the influence of the vasomotor system, it is so much a matter of course that treatment must be directed to the question of arterial tension, that the realisation of this state of arteries must in all such diseases claim primary attention. To be led away by the pain and distress in such a group of symptoms as are witnessed in angina pectoris, without due regard to the state of vascular tension and the amount and character of cardiac lesions present, must inevitably lead to worthless or injurious therapeutics. It has been stated already that one form of what must be called angina pectoris is merely a cardiac neuralgia, and is not associated with any degree of arterial tension. It is in this form that the hypodermic use of morphia gives the best results, and, strange to say, it does so even in cases in which the cardiac neuralgia seems to be due to implication of some portion of the cardiac plexus in disease at the base of the aorta. It is, however, far more certain

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in its effects when no lesion of nerve can be discovered, and where the tendency to the paroxysm of pain can be overcome by quinine, arsenic, or the salts of zinc. In this variety no advantage will be gained by counter-irritation; but in those forms in which the changes in the action of the heart and in the circulation exhibit the characters of irritation of the excito-motor cardiac nerves, or of increased action of the vasomotor nerve system, counter-irritants have been found to be of practical use by their reflex influence on this system and on the cardiac nerves. Of counter-irritants the one that acts most rapidly is cutaneous faradisation, recommended originally by Duchenne, and spoken of in terms of praise by Dr. Eulenberg. The influence of this remedy differs according to its strength; if weak it causes increased contraction of heart, acceleration of circulation, and contraction of the vessels, by reflex irritation of the excito-motor and vasomotor nerves; if strong, on the other hand, it weakens the force of the heart's contraction, retards the circulation, and dilates the vessels by reflex irritation of the regulatory nerves of the heart and paralysis of the vasomotor centres. It is therefore in its stronger form that it influences for good those varieties of angina pectoris in which increased and violent action of the heart, cord-like contraction of the arteries, and small tense pulse exist; whilst in those cases in which from the commencement the phenomena of vagus irritation and paralysis of vessels are prominent, or in which during the course of an attack a tendency in this direction is early percep-

tible, counter-irritation is admissible either not at all or only in its mildest form.

It is probable that practitioners use this cutaneous faradaic instrument more frequently than the constant current; but Eulenberg states that similar considerations of the special symptomatology of the individual case must likewise guide us in the application of the constant current, the proper use of which forms probably our most important, and perhaps our only, remedy for angina pectoris. According to the nature of the symptoms, we shall have to choose the special method of application which will produce reflex irritation in part of the regulatory cardiac nerves at one time, and at another time immediate galvanisation of the cervical sympathetic.

From the very nature of the pathological lesions, the morbid conditions of the heart and arteries are beyond remedy. It is only possible to insist upon the avoidance, as far as may be, of the cardiac disturbances, often mechanical, that excite a paroxysm. In some instances the phenomena will recur quickly under every circumstance of care,—at other times, a breath of cold air, slight emotion, especially of anger or of fear, going uphill or upstairs, a little effort of stooping or lifting or in defæcation, may induce the attack. In the forms in which there is arterial tension, the paroxysm may be cut short by the inhalation of nitrite of amyl, or by the exhibition of a drop of a $\frac{1}{100}$ solution of nitroglycerine. Relief of spasm of heart and of vasomotor constriction of arteries is the result.

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Although hypodermic injection of morphia is most useful in the neuralgic form of the disease, it certainly also is often successful in relieving the agonies of an anginal paroxysm with diseased coronary arteries. In the form in which there is evidence of vasomotor paresis, some good results may follow the exhibition of some of those remedies that increase arterial tension, such as bromide of potassium, the acids, ergot, hazeline, belladonna, nitrite of potash, squill, Calabar bean, nux vomica and strychnia, turpentine, &c.

Besides the temporary expedient of treating the paroxysms, it is well to place the patient under the influence of a line of treatment directed towards lowering the arterial tension. Such a course would include one or more of such remedies as antimony, alcohol in large doses, chloroform, chloral, quinine in large doses, veratrum viride, gelseminum, and jaborandi.

In all forms it may be wise to combine therapeutic agents that, by being neurine tonics, render the nervous system in general less likely to be affected by exciting causes—pre-eminently perhaps, the salts of the metals, but including also caffeine, guarana, valerian.

In the case of a man, aged 60, under the writer's care, every paroxysm, including the fatal one, was induced by slowly walking up a steep street. In another case, when the lady, aged 45, had suffered from an early age from something the matter with the heart, this organ was found largely dilated, without murmur over any valves, but acting weakly and irregularly. It was not until the age of 45

that she began to suffer from angina, and she died a few months after coming under observation, not from the angina, but from gradual œdema of lungs and from hæmaturia. In a third case, also associated with cardiac lesion, here in the form of an incompetent mitral valve, the young lady, aged 17, suffered from attacks of pain so terrible that death was imminent. She had been weakened by a previous attack of diphtheria, and under treatment she was relieved for some time and afterwards lost sight of. It is probable here that the cardiac lesion was of old standing, and, considering the age of the patient, the angina here was a mere cardiac neuralgia induced by the nerve weakness consequent on her previous illness. It is curious, however, as occurring with decided disease of mitral valve. In an other case, aged 26, there was no valvular disease, although the patient had had rheumatism; but between the paroxysms the heart-sounds were very weak and distant. The case disappeared from notice after a time, but the evidence of arterial tension during the attacks, the pain, the sensation of approaching death, the extension of the pain down the left arm, rendered cardiac lesion at least probable. The age, 26, was an unusually early one. In another case, aged 48, the first attack had occured eight years previously, and she had had four attacks in the eight years. The first attack came on whilst she was reading in a church, the second and third and fourth whilst walking either up an ascent or against the wind. Cardiac impulse could hardly

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be felt anywhere. The fifth attack, occurring under observation, lasted two hours (this was before the introduction of nitrite of amyl). The haggard countenance, the precordial pain, radiating to the left arm, the sense of approaching death, the hands clasped over her head, afforded a typical example of the anginal paroxysm. Dyspnœa was present in this case, but this is a very variable symptom.

Instances of the purely neuralgic type, or of paroxysms occurring in middle-aged people with gouty tendencies, but without cardiac lesions, are common enough in the experience of all practitioners. The writer has met with one case, in whom most distressing phenomena of anginal type occurred every day for several years, but without any ill result to the general health. Remedies for gout did no good, but she eventually improved under arsenic. These attacks often mean that the nervous system is out of gear from overwork, especially anxious work, and that the cardiac nerves are as weak as the others. The fact that arsenic, guarana, quinine not in very large doses, general tonic treatment, diminished work, and increased amount of fatty food do so much good, is a proof of this.

In yet another case, one of a type, a lady of a rather gouty family suffered several times a week from symptoms of which pain in the heart was not the most prominent. There was, however, the sense of impending death, without dyspnœa, and some pain and numbness down the left arm, followed by so much exhaustion that it became impossible for her to

carry on the duties of life, in her case very onerous. Nothing did her good except an alkaline and diuretic treatment, and recovery was complete. Here there was no organic disease of any kind.

In brief, then, it seems certain that the forms of angina are various: (1) with spasm of heart and arterial constriction; (2) a pure neuralgia which may or may not be associated with disease of heart or of aorta; (3) a condition of vasomotor paresis from a central origin, or excited by reflex irritation, or under the influence of emotion; that precordial pain is the symptom common to all forms; that, although it may be convenient, as in the case of hemicrania, to class all forms under one heading, yet in causation and pathology they are separate diseases, and demand a treatment wholly different in each variety; and that the sphygmograph is perhaps the best auxiliary in forming an accurate diagnosis and determining upon the therapeutic agents to be used.

Consult—

Eulenberg and Guttman, op. cit.
Chapin, op. cit.
Marey, 'La Circulation du Sang'
Ziemmssen's 'Medicine.'
Seeligmüller, op. cit.
Byron Bramwell, op. cit.
Landouzi, 'Med. Record,' xi. 468.
Dr. Spender, 'Relief of Pain,' 193.
Dr. Sturge, 'Brain,' v. 494.
Dr. Laycock, 'Med. Times,' 73, i. 435.
Dr. Dowse, 'Lancet,' 1879, ii. 686.
Dr. Moxon, 'Lancet,' 1881, i. 685.
M. Huppert and others, 'Med. Record,' vi. 468.

CHAPTER XIII.

HEPATIC NEURALGIA. DIABETES MELLITUS.

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HEPATIC neuralgia is a reality, but probably much less often than is commonly supposed. There is no pyrexia, but the pain may be even more severe than that of acute hepatitis, and is usually accompanied by tenderness of epigastrium and hypochondrium. Dr. Spender speaks of the successful exhibition of hydrochlorate of ammonia in this affection. Innervated from nearly the same sources as the stomach, there is no reason, except that of experience, why the liver should not be as much subject to a neuralgia as the stomach. It may be set up by anything that induces anæmia of the liver, such as constriction of the smaller hepatic arteries, perhaps also from lesions of the right semilunar ganglion, or one of the higher nervous centres. Dr. Allbutt is inclined to look upon it as a pain aroused by the coincidence of an impressionable or neurotic habit with the presence of gallstones at rest in the gall-bladder, and believes that it is in many cases a subacute pain arising in the gall-bladder or in the ducts, due either to the irritation of gallstones or to some vicious quality of the bile. The last point is

probably the most important. The passage of inspissated bile along the ducts, a fact the reality of which is proved by the after presence of dark flakes of bile in the evacuations, is often associated with pain as intense as the worst neuralgia. In a first attack the diagnosis of this pain from that caused by actual gall-stone (of which this condition of the bile may be an early stage) is almost impossible. In a patient under the writer's care, the passage of such inspissated bile is attended with nausea, malaise, rigor, even definite tremors, and great faintness, and on several occasions by partial loss of consciousness; and invariably the evacuations on these occasions contain the dark flakes above mentioned. Hepatalgia from such a cause is very common among people of a sedentary habit, and these by no means always persons of a neurotic tendency. But hepatalgia without any such lesion may occur, though rarely.

The vasomotors of course take the same place in hepatitis that they do in other inflammations.

The vasomotors of the liver itself are seldom directly implicated in the causation of chronic congestion of that organ. This condition far more frequently depends on morbid states of the heart itself. In a case of acute atrophy of the liver Dr. Shingleton Smith found the liver very small, and an atrophied condition of the nerve-cells in the sympathetic ganglia. Certain injuries to the solar plexus cause increase of the circulation of blood in the liver, and of bile.

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Injury of the vasomotor centre for the liver in the medulla oblongata, or of the nerves that arise from it, in their course down the spinal cord, or from the cord to the hepatic plexus, leads to paralytic dilatation of the vessels of the liver, producing hyperæmia and diabetes mellitus. Claude Bernard found the exact spot in the medulla oblongata, close to the origin of the vagus, between the nucleus of this nerve and of the auditory. He proved that the vagus has nothing to do directly with the diabetic phenomena, but the sympathetic in its relation to the circulation. The mechanism is by means of paralytic dilatation of these vessels of the liver, causing an increased flow of blood, and thus by transformation of the glycogen, an augmentation in the quantity of the sugar formed ; this, entering the general circulation, shows itself in the urine.

The vasomotor nerves of the liver can also be paralysed by injury of the cervical and upper thoracic ganglia of the sympathetic. Division of the splanchnic nerves does not cause diabetes. Apart from experiment there are not many cases on record that definitely show a connection between diabetes and the sympathetic. Klebs found atrophy of the solar ganglia with integrity of nerve filaments to the hepatic artery ; Lubimoff, sclerosis of the cells of cœliac ganglia. It has been seen in lesions of the cerebellum, especially of the vermis, of the optic thalami, the crura cerebri, the pons varolii, the middle crura of the cerebellum, and in injuries of the medulla oblongata. It is met with sometimes as a sequence of small hæmorrhages in old people in the motor

area of the cerebral convolutions. But saccharine urine has been seen in cases of sciatica, and has disappeared when the sciatica was cured. Symmetrical sciatica has been observed in association with diabetes; and in a recent case symmetrical neuralgia of the fifth nerve was met with by the writer under the same circumstances. In fact all neuralgias in diabetes are usually symmetrical. In three cases of diabetes there was also hyperidrosis unilateralis, and Burdel found sugar in eighty cases out of eighty-one of intermittent fever. The glycosuria became slighter when the fever lost its intermittent and took on its remittent type. Verneuil has recorded a similar case. Dr. Powell writes to the author: 'I am reminded of a curious case of profuse and obstinate perspiration over the whole body, but more especially the hands, forearm, neck, and thorax, associated with congested liver and diabetic urine, which occurred in my practice lately, and after trying various remedies, together with attention to the digestive function, I found that $\frac{1}{25}$ gr. of sulphate of atropia every night was the only remedy that made the slightest impression, and not only stopped the perspirations but the sugar disappeared as well. I have thought that this instance of decided connection between diabetes and the sympathetic might be of some interest to you, as it decidedly was to me. I may add that the mother of my patient was the subject of diabetes mellitus, and ultimately died from it.'

Not only is the vasomotor action on the hepatic vessels a necessary element in the production of

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diabetes, but Schiff speaks of the same influence on the capillaries of the general circulation. The immediate mechanism for producing diabetes is the formation of a substance that can be easily transformed into glucose, and the presence of a ferment to induce this transformation. Schiff declares that the ferment is also produced in the capillaries of the general circulation, with the necessary element of stasis of the blood, and conditions which lead to stasis are all-important to this end.

Diabetes may be a term that includes diseases of various origin. It is perfectly certain that congestion of the liver, necessary as it may be to the formation of the products that induce diabetes, is not in itself the one and only cause. How many cases of congestion of the liver come under notice in which these diabetic phenomena do not obtain? But in a large number of cases, a number that increases the more readily the relation is realised of one part of the body to the rest by means of nervous influence, the floor of the fourth ventricle is the centre of a reflex arc for diabetes. Its centrifugal effects are not carried to the liver by the vagus: the cervical ganglia may or may not be involved. The bulbar influence is carried down the cord, doubtless by sympathetic fibres which pass along the cord simply as along a roadway, not partaking of the nature of the organ; and joining the arch between the last cervical and the first dorsal ganglion, is transmitted down the splanchnic nerves to the solar plexus, whilst some communication may also take place

along the thoracic ganglia and the first abdominal ganglion, and the solar plexus be thus reached. Thence fibres proceed to make up the hepatic plexus, and to rule the condition of the circulation in the liver. But this is not all. Over and above the dilatation of vessels and the increased hyperæmia of the organ, there are transmitted through the same channels other fibres that influence the phenomena of nutrition and secretion, fibres that play a part in the liver resembling that played by the fibres of the chorda tympani, which excite secretion in the submaxillary gland.

The influence of the sympathetic system is therefore very great in diabetes. It includes direct or reflex lesion of a bulbar centre, or lesions so near as to be almost direct; hyperæmia of the liver; sometimes stasis in the capillaries of the general circulation; an influence on the secretion of glycogen, and of the ferment necessary to its transformation.

With reference to the view that under the one term 'diabetes' several morbid conditions are included, it is only necessary to refer to the causes of diabetes. These have been formulated by Chapin thus: (1) There is increased formation of sugar, due to (*a*) rapid digestion of starch and sugar; (*b*) failure of the glycogenic function of the liver and muscles; (*c*) increased transformation of glycogen into sugar. (2) Lessened combustion of sugar, caused by (*a*) insufficiency of ferment which would convert it into lactic acid; (*b*) altered quality of sugar, enabling it

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to resist the action of the ferment; (*c*) diminished circulation through the muscles. The nervous system may in various ways be the means by which the pathological condition of glycosuria is produced. Thus the failure of the glycogenic function of the liver may be due to a dilatation of the hepatic vessels and an increased rapidity of flow through them, not giving time enough for the transformation of the sugary products of digestion into glycogen. This is one cause of those intermittent forms of the disease which are observed as occurring after meals, and which, if the meals are frequent enough, become continuous but remittent. The dilatation in these cases is probably confined to the portal vein.

Secondly, the increased transformation of the stored-up glycogen back into sugar may be due to a larger proportion of ferment, or to an increased circulation through the organ. This last may be brought about either by increase of the arterial pressure or by a dilatation of the vessels of the liver, especially of the hepatic artery. It is plainly by means of the vasomotor nerves that these conditions are produced.

Dilatation of the hepatic vessels which stand in immediate causative relation to the appearance of sugar in the urine may be due:

(*a*) To paralysis of the vaso-constrictor nerves going to the liver.

Numerous cases are on record in which diabetes was evidently due to lesions of the cerebro-spinal centres.

(*b*) The vessels of the liver may be made to dilate, and the production of sugar increased reflexively by irritation of the vagus and other sensory nerves. Not only can the vagus be irritated along its main trunk, but excitation of its terminations produces the same effects as was shown by Schiff's sticking a needle into the liver, Pavy stimulating it by electricity, and Harley by injecting alcohol into the portal vein.

Brunton assumes that irritation of the other branches of distribution has the same effect. He attributes to this cause the glycosuria observed in intestinal irritation, and after the inhalation of amyl nitrite and the various anæsthetics, the latter irritating the pulmonary branches of the vagus. The way in which mental acts operate to modify the functions of the nervous system is not much understood, but perhaps it comes under reflex better than elsewhere; at any rate, it is well known that mental emotions are not an infrequent cause of glycosuria.

(*c*) Glycosuria is produced also by rise of the arterial pressure, and this in turn is due sometimes to the action of the vasomotors. It is to the changes in the blood-pressure that we find sugar in the urine in convulsions of various kinds; with impeded respiration, after exposure to cold; with cholera and chills. In these cases, from the carbonic acid in the blood, from the toxic element of the disease, or from the action of cold, the vasomotor centres are stimulated, and there follows a rise in the arterial tensions.

Congestion of liver is not enough by itself to cause glycæmia. There must be over-activity of the

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function of glycogen and of ferment. This occurs probably by excitation of the vaso-dilators of the liver and of secretory fibres.

Lesion of the medulla oblongata does not act on glycogen by means of the vagi, nor of the cervical sympathetic. It is a reflex action transmitted by the spinal cord, probably by way of the anterior grey matter, and the sympathetic nerves springing from it. From the cord the passage is by way of the great sympathetic at the level of origin of the inferior cervical ganglion and upper thoracic ganglion and the splanchnic nerves. Most of the vasomotors of the liver come from the solar plexus; this may be connected with the cord by the splanchnic and upper thoracic ganglion, or by the lower thoracic and upper abdominal ganglia. This wealth of communication between the liver and the higher nervous centres may be owing to the importance to the system of the glycogenic function of the liver, and the necessary influence on it of the cerebro-spinal system.

In the 'Arch. für Psych.' 13, a case is recorded of diabetes associated with a solitary tuberculous tumour, the size of a bean, situated just below the left olivary body in the medulla oblongata, and reaching to the exit of the first cervical nerve. A similar case, although the tumour was placed a little lower, was recorded in the Bristol Royal Infirmary Reports, 1878–9, in connection with saccharine urine. In these kinds of instances the diabetes must depend on the implication of the grey matter in the cord, in

the cervical region, and saccharine urine is not found if the white matter only is involved.

There are no specific changes in the sympathetic ganglia in diabetes; no changes, that is, which are not met with in other diseases, and even almost in health. There may be excess of pigmented cells in the ganglia in diabetes, but this change is also found under other circumstances. Kaposi insists on the community of the origin of all diabetic dermatoses, and claims that their common cause is the impregnation of the tissues with sugar. The excitation caused by this impregnation causes alteration of the sensitive nerves, which gives rise to pruritus; then alteration of the secretory and vasomotor nerves, as a consequence of which anidrosis, urticaria, &c., are produced, and then, by direct irritation, inflammation of the vascular walls and other tissues. Some recent investigations by H. Niemeyer prove that in the cœliac ganglia the proliferation of nuclei and the pigmented degeneration that are received by Eulenberg under the name of 'lymphatic infiltration' are not abnormal. He found that, whatever may have been the matter with the patient and the cause of death, in both old and young it was quite usual to find the interstitial cellular proliferation and granulations of pigment within or outside the nerve-cells. Friedreich has found glycosuria follow psychic excitation (persistent emotion, intellectual strain), neuralgia, cerebral disturbance (various injuries of the head and spinal column). In the most recent collection of cases of diabetes, Dr. Windle found that, out of 184 cases, 91

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had no cerebral lesion; of the other moiety the cerebral lesions were exceedingly variable, though perhaps a larger number of lesions affected the floor of the fourth ventricle than any other spot; of 58 spinal cords examined, 37 were normal; 11 showed excavation round vessels, with sometimes dilatation of the central canal; 2, hyaloid thickening of the coats of vessels; 2, congested; 1, soft in cervical region; 1, abscess in grey matter of cervical region; 1, some change in posterior column; 1, cord diseased (nature of disease not specified); 1, congestion of vessels and clot beside cord at third, fourth and fifth dorsal nerves, softening of cord there and at fourth cervical nerve; 1, myxoma of dura mater, with changes in cord, including proliferation of epithelium in central canal and sponginess of grey matter; 1, old disintegration of grey matter at seventh cervical vertebra.

As to the ganglia of the sympathetic, out of 17 cases 8 were normal; 5 showed increase of connective tissue, thickening in capsules of cells, diminution of cells, and of medullated fibres in ganglion; 2, hyaloid thickening of coats of vessels; 1, pinkness of some when compared with others; 1, small.

Dr. Hall White has found, in all cases examined, some changes in the sympathetic—usually of a chronic inflammatory nature. Thus, in one specimen of the thoracic sympathetic, there was much increase of small cells, almost completely hiding the nerve-fibres. In this case the splanchnic showed the same change, whilst the semilunar ganglia presented great increase of interstitial fibrous tissue; abundance of

the new small cells in many places, developing into fibrous tissue. The semilunar ganglia from the second case were almost the exact counterpart of this, but in the third case the changes were less marked. In the fourth case there were numerous scattered brown degenerated patches. The vessels were so dilated as to resemble cavernous structure.

The liver was examined in 220 cases: 84 were normal; in 38 this organ was enlarged; in 19, enlarged and congested; in 12, congested; in 12, fatty (in all these was atrophy of cells); in 9, the liver was small; in 4, congested and hard; in 3, there was hypertrophic cirrhosis; in 3, it was homogeneous; in 2, congested and fatty; in 2, dark and homogeneous; in 2, there were dilated capillaries; 2 had liver enlarged and containing abscesses; in 2, there was tubercle; in 2, malignant disease; 1 was congested with fatty changes and extravasations; 1 was congested with fatty changes and coagulation in veins; 1, congested with coagulation in veins and peculiar capillary dilatation; 1 showed syphilomata; in 1, the liver was large and calculous; in 1, cirrhotic; in 1, it contained a large abscess; in 1, it was pale; in 1, there was hypertrophy of the right lobe; in 1, the liver was small and anæmic; in 1, it was dark with small well-marked lobules; in 1, hypertrophied with colloid patches; in 1, there was cloudy swelling of the epithelium.

The uncertainty and variability of the lesions seem to prove that the glycosuria may frequently be the result of reflex irritation. This may be the explanation of a peculiar case lately recorded by Dr.

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Joseph Landsberg, of transitory diabetes with transitory paralysis of right abducens nerve. The case got well after a sojourn at Carlsbad. Such a case points to irritation of the medulla oblongata, either by some intestinal abnormality or possibly by gouty blood.

Dr. Habershon speaks of diabetes occurring with exophthalmos, and states that in true diabetes the branches of the pneumogastric are especially affected, and not only is the glycogenic function of the liver found disturbed, but the other parts supplied by the pneumogastric are involved, the pulmonary nutrition is interfered with, and a chronic pneumonia is often induced; palpitation and disturbance of the heart are common symptoms, and the voice is often altered in character.

In lesions of the medulla oblongata the part affected might easily take in the nucleus of the vagus, the vasomotor centres, including the spot, on wounding which sugar is found in the urine. The minute differentiation is scarcely compatible with lesions as they are found. Thus a case of diabetes, which lasted over three or four years, was related by M. Luys to the Paris Anatomical Society. At the autopsy the liver was found somewhat hypertrophied, sensibly increased in weight, deeper in colour, and much congested. The kidneys were somewhat hypertrophied and very pale, but had not undergone structural change. The anterior wall of the fourth ventricle was found highly vascular and its consistence notably diminished, very slight scraping bringing off a gelatinous pulp of a brownish-yellow colour, the

ventricle exhibiting the colour very markedly at certain spots. The lesion consisted in the molecular destruction of the histological elements, and their débris, loaded with yellow granulations, gave to the wall of the ventricle this peculiar colour. This lesion may be regarded as exactly corresponding to the circumstances proved by experimental physiology. M. Martineau has also brought a similar case, bearing upon this subject, before the same society. The patient, having been exposed to great heat two years before, was seized with intense thirst, which never afterwards became appeased, and was indeed the first indication of diabetes, which soon exhibited its other habitual symptoms. From the commencement the man complained of tingling and marked weakness of the whole left side of the body, symptoms which disappeared at the end of three months. He went from hospital to hospital till he died, at the end of about two years from the commencement of the attack, advanced tuberculisation of the lungs and atrophy of the papillæ of the retina being among the numerous lesions discovered after death. The most interesting of these, in the present point of view, was that affecting the fourth ventricle. The floor of this, especially near the calamus, was of a marked grey colour, produced by its abundant injection, the vessels coursing along the surface of the ventricle being also larger and more apparent than in the normal state.

The rationale of the effect of these nerve-lesions in producing glycosuria was sought for in vain. But in 1874 the question was solved by Dr. Pavy. He for

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some time previously had been led to look to an altered condition of the blood flowing to the liver as likely to prove the most probable cause of the transformation of amyloid substance into sugar, which evidently constitutes the foundation of the artificial diabetes following operations on the nervous system. Schiff is of this view, and has referred the escape of sugar from the liver, and thence the production of glycosuria, to the development of a ferment in the blood, as a result of the hyperæmia (not necessarily of the liver) which follows the operations on the nervous system which occasion artificial diabetes; but although the experimenter has carefully examined this opinion, he cannot obtain evidence of the development of a ferment in the manner asserted. Dr. Pavy further tried the effect of introducing a secretion, viz. saliva, into the circulatory system, which is known to act as an energetic ferment upon the amyloid substance of the liver; and on one occasion he found that, from some cause or other, the urine became to a moderate extent saccharine; but in a large number of other experiments the operation was attended with a negative result. Having so far proceeded without success, it occurred to him to try the effect of introducing defibrinated arterial blood into the portal system. He was led to experiment in this way from having some time previously observed that, when arterial blood was allowed to flow through the liver—as, for instance, when the portal vein was tied and the hepatic artery left free—sugar escaped from the organ to such an extent as to render the contents of the

circulatory system strongly saccharine. He had not succeeded by this operation in producing glycosuria because, as it appeared to him, no urine was secreted, owing to the ligature of the portal vein leading to such a diversion of blood from the general circulation, by the accumulation occurring in the portal system, that the flow through the kidney was too slight to allow of it. He had endeavoured to overcome this obstacle by connecting, through the medium of a canula, the portal with the right renal vein after ligaturing the corresponding renal artery. If the experiment had succeeded, the liver would have been left with its arterial supply, but the portal stream would have been diverted and made to reach the inferior cava without traversing the hepatic vessels. As regards the operative part, this Dr. Pavy found he could accomplish, but each time he performed the experiment the object he had in view was frustrated by the canula becoming quickly filled with a plug of blood-clot. It was while under this difficulty that the thought of collecting blood from an artery, defibrinating it, and then introducing it into the portal system, occurred to him. He had considered it possible that some slight effect might be perceptible, but had not anticipated the strongly marked result which is producible. The amount of blood used was from ten to eighteen fluid ounces. After the production of anæsthesia by chloroform, the blood was collected from the carotid artery, stirred in order to defibrinate it, strained, and then very slowly injected into a branch of the mesenteric vein. In one

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experiment, where half an hour had been occupied in making the injection, the urine at the completion of the operation contained a notable amount of sugar, and half an hour later showed by analysis the presence of fifteen grains to the fluid ounce. In a second, the urine contained ten, and in a third, fourteen grains to the fluid ounce, when collected three-quarters of an hour after the operation. Having noticed the effect which has been described from the injection of oxygenated blood into the portal system, it became necessary to ascertain positively that it was attributable to the oxygenated condition of the blood, and not to any other cause. To decide this point an appeal to the counterpart experiment was made. Defibrinated venous instead of arterial blood was injected into a branch of the mesenteric vein, and upon each occasion where such an operation has been performed a negative result has been obtained. With the evidence thus furnished the conclusion may be warrantably drawn, that oxygenated blood in some manner influences the liver, so as to lead to the production of glycosuria. It may be inferred that, contrary to the effect of venous blood, it promotes the transformation of amyloid substance into sugar. The suggestion naturally occurs that, what has been stated above, affords an explanation of the glycosuria occurring after Bernard's puncture of the fourth ventricle and the various lesions of the sympathetic. Without any new agent being called in, sufficient is presented in the state of the blood to account for the production of sugar that occurs. By a vasomotor

paralysis affecting the vessels of the chylopoietic viscera, the blood will reach the portal system, without having become dearterialised in its natural way; and in this state it has been shown by the experiments narrated to possess the property of acting within the liver in such a manner as to determine the production of glycosuria.

It has long been known that Bernard's puncture induces a hyperæmic state of the chylopoietic viscera. Schiff asserted many years ago that lesion of the nervous centre in the region of Bernard's puncture was accompanied by a dilatation of the small vessels of the intestines and liver, producing a kind of paralytic hyperæmia of these organs. The same condition has been noticed to have been produced through the medium of injury to the sympathetic.

Dr. Pavy states that 'one of the main points I have brought forward is that the effect of blood unduly charged with oxygen reaching the liver by the portal vein is to occasion glycosuria.'

'It happens that this is just the state into which the portal blood is thrown by vasomotor paralysis affecting the vessels of the chylopoietic viscera, and such, I consider, constitutes the key to the explanation of the saccharine condition of the urine in diabetes. It may be observed by superficial examination in the case of division of sympathetic in the neck, that not only is there a hyperæmic condition of the ear, but that the veins contain much redder blood than natural. In fact the blood passes with such velocity and in such volume through the affected

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part that it does not become properly dearterialised. A similar state existing in connection with the vessels of the chylopoietic viscera will give what is sufficient to produce glycosuria. Without any new agent being brought into the question, the swift passage of blood through the vessels, in such a manner as to cause it to arrive at the portal vein in an imperfectly dearterialised condition, will supply all that is wanted to account for the unnatural passage of sugar. In the vasomotor paralysis, which observation shows is produced by lesions of the nervous system that give rise to glycosuria, we have a condition that leads to the presence of imperfectly dearterialised blood in the portal vein, and in this presence of imperfectly dearterialised blood in the portal vein we have a condition that suffices to determine the escape of sugar from the liver in a manner to produce a diabetic state of the urine.

'Physiologists have referred the production of glycosuria, under the circumstances alluded to, to hyperæmia of the liver. Doubtless hyperæmia of the liver accompanies the exalted flow of blood noticed through the other viscera of the abdomen, but it is not specially this which is required to account for the phenomenon. I have already stated that I have not found glycosuria follow division of all the nerves passing in the lesser omentum to the liver, an operation which might be rather expected to cause hyperæmia of the organ by paralysis of the coats of the artery.'

We know that by operating upon the medulla

oblongata and the sympathetic system diabetes may be artificially produced; but it is not necessary that attention should be confined to these structures, for Eulenberg has shown that the state of the arteries is affected by lesions of certain parts of the grey matter of the brain, probably because spots at the surface of the brain stand in the position of cerebral vasomotor centres, or at least of inhibitory centres of the activity of the vasomotor system. It may happen, therefore, that diabetes may arise either from a lesion affecting and involving a loss of power in vasomotor centres, or a lesion in some part or other of the cerebro-spinal system leading to an inhibitory influence being exerted upon them. Bouchard has also called attention to the frequent absence of knee-jerk in diabetic patients.

The outcome of various investigations, then, seems to be that,

1. The vagus is the sensory nerve of the liver.

2. That after section of the vagus, sugar appears in the urine only after stimulation of the upper end of the cut nerve.

3. That puncture of the fourth ventricle causes glycosuria, when the nucleus of the vagus is wounded. This irritation of the vagus centre acts by inhibiting the vasomotor centre of the liver, and thus produces diabetes. When this irritation is removed sugar disappears, and in this respect the effect of this irritation differs from that produced when the vasomotor nerves of the liver are divided, as in the latter case the glycosuria is found to be more persistent.

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4. That irritation of some of the branches of the vagus is followed by glycosuria. This is especially the case with the pulmonary branches, as their stimulation by air inhibits the vasomotor nerves of the liver.

5. That the irritation of these pulmonary branches by carbonic oxide, ether, chloroform, &c., before the stage of narcosis is reached, explains the presence of sugar under such circumstances.

6. That the good effects of opium and its salts, especially codeia, are probably due to the fact that they lessen the reflex inhibition in the liver.

7. That 'reflex inhibition may follow irritation of other parts of the encephalon, and possibly also of sympathetic ganglia, as well as of the roots, trunks, and branches of cerebro-spinal nerves.'

8. That 'the diabetes which has been observed after injuries of the cerebral lobes in man, of the cerebellum in animals by Eckhard, of the superior cervical ganglion by Pavy, of the optic thalami, cerebral peduncles, pons Varolii, middle peduncles of the cerebellum, and of the cervical sympathetic cord and sciatic nerve by Schiff, is probably due to this cause, as in all these cases it is only temporary and not permanent. Irritation of the sciatic nerve in man seems to have the same effect as in animals, for temporary diabetes has been observed during an attack of sciatica.'

9. That a brisk circulation of arterial blood through the liver, especially through the hepatic artery (because the blood from this vessel enters the

portal vein, and increased circulation in the hepatic artery leads to the same phenomenon in the portal vein), is a main factor in the production of sugar in the liver.

10. That this increased circulation through the hepatic artery may be induced by any cause that raises the pressure of blood in the arteries generally, or by any cause that induces dilatation of the hepatic artery, the general arterial pressure remaining the same.

11. That the blood globules are the chief agents in bringing about this increased production of sugar in the liver. Some observers believe that they are so by containing the ferment (pancreatic or other) on which the transformation of glycogen into sugar depends.

12. That the vasomotors of the liver have their origin in the vasomotor centre of the medulla oblongata, pass a short way down the cord, and either leave the cord by the fibres that accompany the vertebral artery, passing in them to the lower cervical ganglia, thence to the first dorsal ganglion, and so through the upper portion of the dorsal gangliated cord to the splanchnic, the cœliac ganglia, and the hepatic vessels; or else pass wholly down the cord, only to leave it by communicating branches that lead to some of the dorsal ganglia.

13. That the condition of general arterial pressure, and the dilatation of the hepatic artery, and in a less degree of the portal vein, are under the governance of the vasomotor centres; and that this is the

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manner by which this system of nerves is associated in diabetes.

14. That the connection is almost invariably reflex, and that the vasomotor system generally affords the centre of the reflex arc and the exodic nerve. In most cases the eisodic nerve is from some region of the cerebro-spinal system, and most frequently is some branch of the vagus.

It is on this view that electricity may be employed as a curative agent. It is recommended that electrostatic currents of sparks and shocks should be passed through the liver and other parts of the body. Favourable results were obtained by Dr. Clemens of Frankfort-on-Main by employing induced currents in the direction from the back of the neck to the liver. This can only have been by modification of the calibre of the hepatic arteries.

15. That that form of glycosuria that depends on imperfect combustion of sugar, due to a want of the ferment which should prepare it for oxidation rather than to the want of oxygen itself, has little to do with the sympathetic system.

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CHAPTER XIV.

VISCERAL NEUROSES.

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UNDER the head of neurasthenia something will be said about variation in the movement, sensation, and secretions of the stomach. A morbid condition of the vasomotor nerves is a necessary factor in inflammation anywhere. In considering diseases of the stomach, it would therefore be impossible to leave this nervous system without reference. In gastric lesions, too, are found the various methods by which the vasomotors of a part are affected, direct excitement of their peripheral termination, as by certain poisons taken into the stomach itself, by some morbid influence affecting the vasomotor centres higher up in the course of the gangliated cord, in the spinal cord, or in the medulla oblongata; or by reflex action set up by irritation from a distant organ. It has been seen also that as regards blood supply the vasomotors play a not unimportant part in secretion, though the strictly chemical elaboration is the work of cerebro-spinal nerves. In secretion of gastric juice, therefore, the rôle played by the vasomotor must be considered, nerve disturbance vitiating secretion, and as is so common in the sympathetic

plexuses, vitiated secretion or any organic abnormality setting up nerve disturbances.

Most of the experiments on the cœliac plexus prove that it is sensitive to pain. In a similar way hyperæsthesiæ are met with of the solar, mesenteric, hypogastric and spermatic plexuses. That lead colic is connected with toxic lesion of the sympathetic (as of other nervous tissues) seems proved by its association with the small hard pulse due to cramped narrowing of the whole peripheral arterial system, with whiteness and coldness of the face and extremities. The cardiac energy is diminished; the apex beat is scarcely felt; the pulse may vary from 30 to 60 in the minute. In a few cases lesion of the sympathetic has been found. In one there was an increase of volume, and a greyish yellow colour of the abdominal ganglia; in the other sclerosis of the connective tissue in the cœliac plexus. Asthma, vertigo, perspiration, tenesmus, suppression of urine and palpitation are all met with in connection with lead colic.

The two following cases show a divergence as regards sensation that is interesting. A gentleman, aged 60, became suddenly affected with acute myelitis. In the course of two or three hours he became absolutely paraplegic (motor and sensory); the sphincters were useless. There was girdle-pain just above the umbilicus, up to which spot anæsthesia was complete. In the eight days he lived, his appearance was remarkable from the very intense intestinal distension. Under ordinary circumstances, such distension would

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have caused much pain; but, although he resembled a barrel in appearance, he complained of no pain below the umbilicus, though the pain and distress above this point, consequent partly on the impossibility of diaphragmatic action, was considerable. The other case was that of a young lady, aged 17, not hysterical. It was thought she had tried her spine by over-riding. Constipation had been troublesome for six months before her illness, and it persisted as a marked symptom for many weeks, as a rule. Very large and drastic enemata were retained, but from time to time fæcal masses were got rid of, in one of which, on a single occasion, were found some peas which she had eaten raw or imperfectly cooked fully two months before; the peas had sprouted in the bowel. The agony this girl experienced was extreme, the symptoms being those of almost complete paralysis of the plexuses of the sympathetic that rule peristaltic movement. In this case there was tenderness along the spine from the mid-dorsal region down to the coccyx. The case must be classed under the heading of neurasthenia, possibly with hereditary predisposition, the mother having been a martyr to gastric neuralgia; as it would not be likely that increased inhibition of peristaltic movement, due to irritation of the splanchnics, would persist for so long a period.

The neurasthenia of the abdominal sympathetic in adult life may be only due to premature old age. The deficiency of peristaltic action, and often more or less of intestinal secretion that is often met with in old age, depends on exhaustion, on deficient blood-

supply, quantitative or qualitative, to the parenchymal ganglia, or on commencing atrophy of the spinal cord; and is simulated much earlier in life by any thing that depresses power, such as various exhaustive diseases, the nerve-fatigue consequent on coition, prolonged travel, &c. Very little is known in regard to the movements of the stomach, but the motor fibres to this organ are supposed by Flint and Von Braam to be in the vagus; but Flint thinks that they are derived by the anastomosis of this nerve with the sympathetic. The peristaltic movement of the intestines, though automatically excited by the parenchymal ganglia, yet seem capable of receiving accelerating influences from the sympathetic, but not through the splanchnics. The vagus has no influence on them (Von Braam and McKendrick). According to the latter writer, the accelerating nerves are from the abdominal sympathetic ganglia, while the inhibitory fibres are from the lumbar-spinal nerves. The descending colon and rectum receive motor fibres, according to Nasse, from the plexus around the mesenteric artery. The most generally received views, then, are that the stomach and intestines contain in their parietes ganglia which send out motor fibres to the visceral muscles (Budge, Kölliker, and McKendrick), and that these ganglia are stimulated reflexively by fibres running to them from the mucous membrane (Henle). Moreover, these ganglia receive other fibres from central ganglia in the stomach, through the vagus, and in the intestines through the lumbar cord and sympathetic. The idea of Pflüger's, that the splanchnics are inhibitory

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nerves of peristalsis for the intestines, is disposed of by the experiments of Basch, which show that these nerves only secondarily affect the intestinal movement by their action on the circulation in the gut; but Vulpian asserts that the arrest of movement in the small intestines, consequent on faradisation of the great splanchnic nerve, can be obtained without appreciable change in the intestinal vessels. Galvanisation of the vagus only causes pallor of the intestinal vessels indirectly, by influencing the whole circulation through abnormal inhibition of cardiac action.

To return for the moment to lead colic. Both theories as to the rationale of the symptoms point to the sympathetic system. One is that lead colic is due to a constriction of the intestines due to the special irritation of the lead on the ganglionic plexuses of the sympathetic, destined to the intestine; and that the constipation is the result of this contraction. The other theory suggests that the lead acts as an excitant on fibres contained in the splanchnic nerve, producing a relaxation of the intestinal muscular tone, the constipation being the result of the inertia of this tunic. In choosing between these two theories, both of which are somewhat hypothetical, it is well to look at the teachings, sparse as they are, of pathological anatomy. Constriction and apparent thickening of the muscular coats of the large intestine have been found, and an increase of the connective tissue, and atrophy of the nervous tissue in the abdominal ganglia of the sympathetic. Segoud found the same condition in

several cases of endemic colic at Cayenne. Tanquerel des Planches narrates a case in which there was marked enlargement of a number of the abdominal ganglia. Also Seeligmüller speaks of two autopsies, in one of which there was increase of colour and a greyish yellow colour of the abdominal ganglia; in the other sclerosis of the connective tissue in the cœliac plexus.

In the absence of knowledge as to lesion of nervous centres in lead colic, it is difficult to explain the pain except on the hypothesis of irritation and compression of the intestinal nerves, this compression being induced by this spasmodic constriction of the gut. Constipation would be a necessary adjunct to this spasmodic state, and although it may be true that it is due, in part at least, to the cessation of the intestinal secretion by contraction of the blood vessels, this condition is hardly necessary for its explanation.

The symptoms of lead colic in part may be compared to those of that form of angina pectoris associated with cardiac spasm and high arterial tension.

The colic of lead in no respect differs from other varieties of colic except in its causation. Colic in general is intestinal spasm, and it may be produced from direct or reflex irritation. Chill to the surface of the body is not a very unusual cause of the latter kind; but it is most frequently induced by the presence of deranged bile or of some other vitiated secretion in the bowel, or by the presence of an

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undigested and indigestible article of food. It is most probable that the ganglionic centres in the coats of the intestines receive the primary irritation; but Warburton Begbie calls attention to the part played by the vagus in the movement of the intestines. It has been already stated that the influence of this nerve on the intestinal circulation is probably through its inhibitory action on the heart. But Begbie states that it has been shown by carefully conducted experiments, that the pneumogastric nerves possess an influence on the movements of the intestinal canal. Such experiments have exhibited the contractions of the muscular coats of the intestines under the application of electrical irritation to the vagi of as rapid and violent a character as those of voluntary muscles when their motor nerves have been subjected to a similar irritation. Again, when on irritating the ganglionic plexuses surrounding the aorta, by means of the rotary apparatus, the small intestines and colon, which had been previously wholly inactive, when the current began to operate, were seized with universally active movements, which continued for a long time after the current was interrupted. It is of further interest to note that among central portions of the nervous system it is the medulla oblongata which, when irritated by the galvanic current, excites in a decided manner the movements of the stomach and the intestinal canal. Budge saw the same result produced in rabbits, but in a less degree, by irritation of the cerebellum.

The spinal cord and cerebrum possess no such

influence. M. Martineau has pointed out that while the pneumogastric nerve is more especially distributed, as is well known, to the stomach and liver, a portion of the right nerve passes to the semi-lunar ganglia to anastomose with the splanchnic nerves of the great sympathetic, and thus to form the solar plexus. Galvanisation of the solar plexus, and of the superior mesenteric ganglion, equally causes contraction of the small intestine and more rarely of the large. Valentin has made the very important observation that an irritation of the fifth nerve at the base of the skull invariably gives rise to peristaltic movements of the small intestines, especially of the duodenum and upper part of the jejunum. Such being proved experimentally, we can understand the occurrence of intestinal spasm or colic as the direct consequence of some forms of cerebral irritation. And although, as Romberg has remarked, little is known respecting the influence which is exerted by the affections of the spinal cord and brain upon spasm of the bowels, the very potent operation of the emotions, fears and fright especially but in some instances also joy, in increasing the movements of the intestines, is thoroughly appreciated. There seems little doubt therefore that in the lighter forms of colic the excitant, if located in the bowel itself, irritates fibrils that are connected with the small sympathetic ganglia in the coats of the intestine, that the effect of their irritation is spasm of the bowel, with pain as its consequence, the pain being of sensory nerves of the sympathetic itself. If however

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the irritation be excessive, its effect is carried farther and travels by way of spinal cord to medulla oblongata, being reflected down the vagus to the intestinal muscles, causing a greater spasm and a more intense form of pain. Eulenberg and Guttman, however, believe that pain may reach the sensorium through the splanchnic nerves, through the rami communicantes and filaments in the posterior roots and posterior columns. Possibly both modes of conduction may be used indifferently. It is an instance of which there are so many, of parenchymatous ganglia possessing a certain independence of action, but associated with the higher nervous centres both by accelerating and inhibitory nerves.

It is not always easy to diagnose colic from enteralgia. The latter is apparently a neuralgia of the sympathetic, although increased by the presence of indigestible substances in the intestine, or vitiated secretion, and augmented also by any peristaltic movement of the bowel. It is very frequently associated with gout, and in such cases resists ordinary treatment, unless this is combined with gouty therapeutics. Enteralgia, however, exists as one of the phenomena of neurasthenia, Dr. Allbutt thinks, more often in the melancholic than in the brisk neurotic, but this may well be from the frequent association of melancholia with a gouty history. The chief seats are the umbilicus and the right iliac fossa; but it is met with in the hepatic plexus of the colon. It differs from colic in that the pain is seldom so severe, that it seldom occurs in paroxysms with any

periodicity, and is seldom also relieved by pressure, though pressure does occasionally relieve and is often borne well. Like other sympathetic neuralgiæ its boundaries are more diffused than in neuralgia of a cerebro-spinal nerve ; this depends, of course, on the anatomical distribution, extension, and anastomoses of the sympathetic nerves. There is often much flatulent distension, relieved either by the eructation of flatus or its discharge per rectum. The position of the pain in neuralgia is scarcely sufficient for itself to differentiate this from colic, as in the latter the pain attacks the scrobiculus cordis or the umbilicus by preference. In lead colic, however, and in colic depending on morbid or on retained secretions, the symptoms are commonly associated with arterial tension, from reflex inhibition of heart by irritation of the medullary centre of the vagus. It is sometimes associated with a subjective sensation of faintness and a sense of impending death, from a neuralgic irritation of cardiac nerves, whilst in enteralgia there is no such tension and the pulse is depressed, feeble and irregular. The tongue in enteralgia may be normal, and the bowels are not, as a rule, either constipated or relaxed, whilst in bad colic constipation is the rule, and in ordinary colic from vitiated secretion it is common enough to meet with diarrhœa. Enteralgia depends upon constitutional causes, comprised under the head of neurasthenia, often affecting the nervous system as a whole, but occasionally manifesting their morbid effects in some of the plexuses and fibrils of

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the abdominal sympathetic, and having no recognisable pathological appearance.

Various painful affections of the rectum, at times almost amounting in intensity to colic, are met with in neurotic cases. Sometimes this seems to depend on a spasmodic condition of the detrusor muscle, sometimes it is an hyperæsthesia of the rectum, and as soon as any fæcal matter enters this portion of the gut it is ejected with pain. More often it is a pure neuralgia, often exceedingly painful. In a case under the writer's care, a neurotic case of very marked degree, the pain was unceasing, only more intense at the moment of defæcation. The most careful examination failed to find any lesion, and it only passed away on the substitution of a neurosis of another organ. A form of neuralgia by no means common has been mentioned by Dr. Myrtle. It is purely neurotic in character and very fitful in its attacks, coming on at long intervals and when the subject of it is apparently in the best form; he will go to bed perfectly well, and awake at any hour with a gnawing, grinding pain in the sphincter. This gradually increases in intensity, acquires its maximum in a few minutes, and then gradually goes off without treatment.

It is a form of neuralgia, and produced by exposure to cold, either from the bedclothes getting off the part or being too scanty or insufficient to protect it. During the day it may arise from sitting on a cold seat. However or whenever caused, it is at once relieved by the application of warmth. When

any of the rectal neuralgia are accompanied by spasm, the motor influence (over and above the parenchymatous ganglia) comes from the plexus round the inferior mesenteric artery, as the descending colon and rectum receive motor fibres from thence.

Pain in the stomach is met with under very variable conditions. It may be caused (1) by the pressure in its interior of foreign substances of an irritating character; (2) by organic diseases altering the abdominal structure of its coats; (3) by perversion of its secretions; (4) by perversions of innervation, either proper to the stomach, or reflected from other organs, or originating in the nervous centres. By the first, pain may be caused by a spasmodic contraction of the pylorus, or by absolutely wounding nerves in the coats of the stomach, or by exciting inflammation. Foreign bodies of all kinds, or corrosive poisons, as the mineral acids, arsenic, antimony, and the caustic alkalies, come under this category. By their action gastric pain is caused, but not gastric neuralgia, and this pain is often associated with much vasomotor disturbance in the mucous membrane. Of organic diseases which alter the anatomical structure of the coats of the stomach, the most common are inflammation, cancer, and chronic ulcer. In the case of perverted secretions, the pain may be either by direct influence of the morbid secretion on the nerves of the stomach, or more indirectly by exciting spasms of the pylorus. Pyrosis is a pure neurosis, connected only with the vasomotor system. It is a result of a

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paretic condition of the vasomotors, and it is a matter of doubt whether the fluid itself is a cause of gastralgia, or whether both phenomena are the consequences of one and the same defect of innervation; the fact that pain is somewhat relieved by the ejection of the fluid from the stomach points rather to the former theory, and yet there is no special chemical peculiarity in the fluid of pyrosis that would seem likely to render it a cause of pain. The fourth variety of gastric pain, however, depends on no coarse lesion. It is a neurosis. It is a question as to the nerves affected. Romberg divides these neuralgiæ into two classes, those of the solar plexus and of the vagus, and distinguishes the former by the attending sense of faintness, while the latter is often marked by perversion of appetite.

The points of greatest interest are the conditions under which gastric neuralgia is met with. It is frequently associated with other neuroses, with neuralgia of head and face, with hemicrania, with asthma, with uterine and ovarian neuralgia, with the complex states expressed under the terms 'hysteria' and 'hypochondriasis.' The frequency of gastric neurosis in hysteria may be estimated by the statement of Briquet, that of 358 cases of this affection, only 30 had no signs of gastralgia or epigastralgia, 130 had only pain at the epigastric regions, while 187 had both pain and derangements of the digestive functions; and this author states that the latter are among the first symptoms in females in whom hysteria is slowly developed. This statement of Briquet is

only misleading in that the so-called hysterical cases should many of them be termed rather neurotic.

But in some the defects in gastric innervation are the result of reflected irritation, especially when the cause is seated in the uterus. Such cases are hysteric as regards the source of irritation, often not hysterical in the ordinary misused meaning of this term. Henock says that in some cases of this affection the pain has been known to occur only at the menstrual periods, to cease with pregnancy and to return after delivery. This form is pretty certain to diminish in intensity, and even to disappear, with advancing life; whereas gastralgia in persons of a neurotic type may persist to old age—at any rate far beyond the climacteric period.

It is probable also that gastralgia as well as vomiting depends on the reflex irritation of gall-stones, of diseases of the duodenum, and abdominal aneurism, sometimes also of disease of lungs and heart, especially pericarditis.

It is not often that gastralgia owes its origin to lesion of the nervous centres. Dr. Wilson Fox, however, records a case where epigrastic pain, as well as vomiting, without disease of the stomach, occurred in connexion with hæmorrhage of the brain.

The most frequent form of this neurosis depends upon local and constitutional causes. Anæmia, general, and anæmia of the peripheral nerves and nerve centres of the stomach itself, gout pre-eminently, exhaustion, the state of system induced by chronic renal disease, especially granular kidney, the conse-

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quences of arterial regurgitation, the influence of malaria, a form of sluggish digestion which leads to very imperfect assimilation of food, depressing influences of a moral character and operating through the nervous system, grief, fear, anxiety or severe intellectual effort, probably also prolonged alcoholic poisoning (though this is usually complicated by catarrh of the stomach), certain kinds of shock and the abuse of tea and coffee, are some of the etiological points in the causation of this affection. It may be hereditary.

Of the symptoms, pain is the chief. It may be the only phenomenon; it may be associated with flatus and with perverted secretion. The most intense case that ever fell under the notice of the writer was in the person of a single lady, not neurotic, not hysterical, bright, active, practical in an eminent degree. She was approaching middle life. She had been long treated by starvation diet and aperients, with the effect of greatly increasing her distress. The pain was most agonising, and was markedly associated with a sense of approaching death. Arsenic and better food quickly restored her to health. The pain varies greatly in duration and intensity. It generally becomes intense and of a tearing or gnawing character.

From the close connection of the solar plexus with cardiac plexuses, the action of the heart is often interfered with, and irregularity of heart ensues, followed by syncope. It is interesting also to note, in connection with the pathology of convulsion, that

this phenomenon has been known to be a result of the pain. The pain is occasionally somewhat relieved by pressure, but this is very uncertain. Frequently the slightest pressure cannot be borne. There is no general rule as to the time of the attacks. They may be irregular or periodical. They may be most intense before the ingestion of food, and be relieved by a hearty meal, and often enough by one that would be likely to be indigestible; or, on the other hand, they may immediately follow a meal.

In gastralgia that depends on reflex irritation the pain is usually excessive after food, and is frequently only removed by the rejection of the meal by vomiting. It may last for several hours or only for a few minutes, and may pass away with eructation of gas or with vomiting of an acid or an alkaline secretion.

In a class of case somewhat different the gastralgia is accompanied, not caused, by some depraved state of the gastric secretions. Here the same influence, that induces the pain leads also to the imperfect or perverted elaboration of the secretion. The symptoms frequently include a coated tongue, such as is seen in very subacute gastric catarrh, with flatulence either constipation or a rather worrying diarrhœa, the intestinal dejections being small but frequent. Vomiting is a very variable phenomenon in gastric neuralgia. It is frequently present, it is as frequently absent, and its presence or absence cannot be predicted in any given case, except that it is more likely to occur in cases in which the neuralgia depends on a gouty state of system, or excess of alcohol or

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of tea or coffee, even where there is no superadded catarrh, and perhaps in instances in which the ailment has been caused by prolonged and excessive intellectual exertion. It is especially present in the form that owns a reflex irritation as its cause, and more particularly when the starting point of the reflex irritation lies in the bile ducts, in the kidneys, in the uterus or ovaries. Reflex vomiting without gastralgia is so common under these circumstances, that its presence accompanying reflex gastric neuralgia is not to be wondered at. The recognition of this gastric neurosis depends, therefore, upon some of the following considerations:

1. The presence of some of the causes above detailed that lead to nerve exhaustion, or the fact that the patient is one of a neurotic family, or has some neurotic history from early age, or in himself or among his nearest kin has manifested gout in any form, or phthisis, the latter disease being frequently associated with a neurotic history.

2. The co-existence of other neuralgiæ or neuroses, or the fact that such morbid conditions have preceded the affection of the stomach, or alternate with more or less periodicity with the attacks of pain.

3. Except when anæmia is present, the tolerably healthy appearance of the patient, notwithstanding the frequency of the paroxysms of pain and their degree; in a word, the disproportion shown between the severity of the gastric symptoms and the general state of the patient.

4 The fact that pain is most liable to recur when

the stomach is empty and to be relieved by a full meal, leaving out the theory that the pain is caused by nervous exhaustion.

5. The complete remission of the pain—a diagnostic mark that is not wholly trustworthy, as these remissions are sometimes met with in gastric ulcerations and in cancer.

6. The absence of pyrexia has its value in diagnosis, but there is often no pyrexia in ulcer and in cancer of the stomach.

7. Exactly the same may be said of the fact that the tongue is unaffected. The clean tongue differentiates the gastric neurosis from catarrhal and inflammatory changes, but by no means from all other affections of the stomach.

8. The presence of vomiting is of little use. It may be present or not in gastric neuralgia. When present the patient usually derives relief from the ejection of fluid.

As to the pathology of this ailment, it is in accordance with its nature that pathological anatomy should leave no traces of morbid action. This is only what is so constantly met with in morbid conditions affecting the sympathetic. Among the authorities collected by Chapin under neuralgia of the solar plexus, Autenreith and Romberg have included certain painful affections, presumably of the stomach, occurring in the epigastric regions, and which they consider to be distinct from ordinary gastralgia, which is an affection of the vagus. With regard to the diagnostic symptoms of this neuralgia, Romberg

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says, 'the feeling of weakness accompanying the pain, the sensation of coldness of which the patient often complains, the disturbed circulation, anxious look, and feeling of impending dissolution are pathognomonic.' He believes the cardialgia sometimes accompanying intermittent fever, and mentioned by Borsieri, to be due to implication of the solar plexus. Wittmaach supports Romberg's ideas, and adds, as farther diagnostic points, that cardialgia does not occur so frequently in young persons, is less often associated with menstrual disorders, and does not last so long as gastralgia. Valz, in one case of cœliac neuralgia, found the nerve pressed upon by cancer of the pancreas, but in eighteen other cases no lesion was found. Bamberger and Henock do not believe in the occurrence of this affection, but think that every form of gastralgia is due to implication of the vagus. Eulenberg and Guttman state that anatomy and physiological experiment certainly show that twigs from the solar plexus take part in the innervation of the stomach, but in no way prove that sensory nerves, having a reflex action on the stomach, come from the same source. The truth seems to be that in almost all cases both the vagus and the sympathetic are affected. There seems to be no doubt that the vagus is not the only nerve connected with the stomach containing sensory filaments. It is equally without doubt that most of the sensory filaments of the stomach are included in that nerve. The sensory fibres of the sympathetic portion of the solar plexus must be wholly ignored. But if most of

the pain may travel through the vagus, it is certain that the attacks of syncope, the aguish paroxysm, the epigastric sinking, the frequent pyrosis, the alteration or perversion of the gastric secretions, own a sympathetic or a vasomotor origin, and are connected either with changes in the vascular supply of the stomach itself or with the connection of the gastric sympathetic with the plexuses of other organs. Nay, more, in one form the gastric neuralgia is evidently the result of anæmia affecting the vagus. This anæmia may be general, and it may then be a question as to whether the centre of the vagus or its periphery is mainly affected.

But in many cases it is proved by the action of remedies to be the periphery, and this local anæmia is completely under the influence of vasomotor action. It is this form that is so quickly remedied by the ingestion of food, the irritation of the food leading to a better blood-supply in the stomach, and thereby nourishing and stimulating the peripheral branches of the vagus in a normal manner; and once again, to go over old ground, the share taken by the sympathetic in this disease is rendered more probable by the association of other neuroses, confessedly sympathetic, with the gastric pain, or their alternations with it. Here, again, is the mutual dependence met with of the sympathetic and the cerebro-spinal nerves. As in exophthalmic goître, as in angina pectoris, as in hyperidrosis, as in the severer forms of colic, both systems of nerves are involved, and the morbid phenomena can only be

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explained by the fact of the implication of both, so in gastric neuralgia the causation in some cases, the accompaniment and complication in all, point to the sympathetic; whilst the greater portion of the pain, which is to the patient far the most prominent symptom, is due to the implication, either at its centre or at its periphery, of the pneumogastric nerve.

In connection with this subject of the influence of the vasomotor on the stomach, the vasomotors of the mucous membrane of the stomach are in spinal and peculiar activity in those not uncommon cases of periodical hæmatemesis that seem to be vicarious to suspended menstruation. But similar results of vasomotor parexis are seen under other circumstances. Thus Dr. Handfield Jones records a case in which a weakly female, whose heart and lungs appeared sound, had various symptoms of aguish disorder, with frequently recurring hæmatemesis and extreme debility. With quinine, iron, country air and rest she improved much, and recovered at last after a voyage to Ireland. A severe relapse was induced on one occasion by her resuming work while too weak for the exertion. The hæmatemesis in this case was more a kind of frequent oozing of blood than an arterial gush—once or twice, however, pretty free bleeding occurred. The system was under the influence of a depressing poison, operating primarily on the nervous organs. She had many symptoms showing the effect of this influence on the cerebro-spinal system, neuralgia, bewildered feelings, forget-

fulness, loss of sight; whilst the hæmatemesis was one of the proofs of the influence on the sympathetic and vasomotor nerves. Cases such as this are not very uncommon.

The writer has lately seen a case of a young woman who died of peritonitis associated with cancer of the peritoneum. Three days before death there was violent hæmatemesis, of such a character as to induce the belief that she had the additional lesion of gastric ulceration, probably of a cancerous nature. At the autopsy the gastric vessels were found greatly enlarged, but there was no ulceration. In this case it is probable that the irritation of the cancer in the peritoneum or of the inflammation itself had exercised a paretic effect on the vasomotors of the stomach.

It is not so common to meet with vasomotor hæmatemesis as one of the effects of alcohol, but this is sometimes the case. Alcohol modifies the composition of the blood directly, and by its action on the various tissues. It may induce transient or persistent alterations in the cells of the pepsine-forming glands in the muscular coat of the stomach, in the walls of its vessels. It may cause vascular disturbances in the gastric mucous membrane, either by modifying through the circulation the peripheral extremities of the vasomotor nerves, or the ganglionic plexus in the gastric walls, or the ganglia of the thoracic-abdominal sympathetic, or the parts of the cerebro-spinal system in relation with the central extremities of these nerves; or by means of reflex action it may give rise to constrictions or dilatations of the vessels; or, again,

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by its morbid action on the liver, it may facilitate sanguineous stases in the mucous membrane of the stomach. It may act, too, directly or reflectively on the secretor nerves, and so alter or pervert the secretions of the organs. And thus in a variety of ways it may cause hæmatemesis or give rise to certain aggravated forms of indigestion.

Constipation may depend on deficient peristalsis, or on deficient or perverted secretion, or on both. Von Braam, who opened the abdominal cavity under a solution of common salt at the temperature of the blood, arrived at the following results. The vagus is the motor nerve of the stomach, but not of the small intestines, and movements of the latter after the irritation of the vagus are due to the escape of the masses from the stomach into the intestines. Neither has the vagus any influence on the movements of the colon or the uterus. The splanchnics are at once the inhibitory nerves of the intestines and stomach and the vasomotor nerves of the intestine, and it is for this reason that the gastric movements are less readily induced by irritation of the vagus if the splanchnics remain uncut. Von Braam does not decide whether inhibitory action of the splanchnics exists independently of their vasomotor action or is merely a consequence of it. That the latter is more probably the case is shown by the circumstance that the movements of the intestines are in great measure dependent on the condition and amount of blood in the intestinal vessels. When Professor Nothnagel

laid before the profession the results of experiments on the action of opium and morphia on the intestine, he stated that the constipating power of these drugs appear due to their being irritants of the splanchnics, the inhibitory nerve of the intestines. That nerve is specially influenced by morphia, just as the vagus, the inhibitory nerve of the heart, is acted upon by digitalis; in fact, in both cases small doses excite, large doses paralyse. It was shown in a discussion on this question that the peristaltic action of the intestines is not necessarily the same in man as in animals.

Antiperistalsis does not appear to occur in the latter; in our species it is known to exist, though when obstruction exists peristalsis in the ordinary direction is quite sufficient to account for fæcal vomiting.

Dr. Rosenstein, however, had seen chronic fæcal vomiting in a patient of his, where no mechanical obstruction could be found.

Professor Preyer stated that he had seen anti-peristaltic movements of the small intestine in animals, and pointed out that the filling and emptying of the cæcum, especially of the very long cæcum of some animals, could only be effected by alternate peristalsis and anti-peristalsis. It is commonly assumed that during diarrhœa the peristaltic movements are increased in energy and are more constant than in normal circumstances. Nothnagel found it no easy matter to produce even a moderate degree of inflammation. The most suitable agent was a concentrated

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solution of chloride of sodium. Five centimetres of this were injected into the rectum several times in the course of twenty-four hours, the return from the rectum being prevented. The movements of the bowel were observed next day in the usual salt bath. Nothnagel, however, never succeeded in exciting inflammation in a larger tract than from 30 to 60 centimetres, and the enteritis was always slight in the upper, and considerable and often hæmorrhagic in the lower part. The production of inflammation in the small intestine was much more difficult. Attempts to produce it by injecting irritant substances into the stomach by means of an œsophageal sound, and thus to imitate an occasional course in man, were unsuccessful. Even sulphate of copper failed. Strong solutions caused rapid death and weak solutions had no effect. Only local inflammation could be produced by the injection of concentrated solutions of chloride of sodium through the wall of a loop of the small intestine, withdrawn from the abdomen for the purpose through a small incision.

In both the large and small intestine the peristaltic movements were rapidly increased by the inflammation, and the affected portion became filled with fluid, manifestly inflammatory exudation. The peristaltic contractions took the form partly of oscillatory movements and partly of circular constrictions. Sometimes, however, the affected portion became the seat of a prolonged tonic contraction which involved both the transverse and the longitudinal muscular fibres. Very different, however, was the condition, when twenty-

four or forty-eight hours were allowed to elapse after the injection. The inflamed and empty section of the bowel was motionless, no contraction could be seen in it as long as the experiment lasted, and this even when the inflammation which had been excited was slight in degree. The conclusion, therefore, is that the early stage of inflammation is attended with very strong contractions in the inflamed portions, but that when the initial period is over, and the inflamed part of the bowel is empty, the movement is not greater than in the normal bowel.

Moreover, it appears that the contents of the bowel are, in the early stage, moved through the inflamed portion with increased rapidity. These experiments of Nothnagel do not forbid the view that the increased peristaltic movements may be connected with the increased amount of vascular supply. A paretic condition of vasomotor is an early stage of all inflammation. The temporary increase of the blood-supply consequent on the vasomotor paresis may be the factor that stimulates the peristaltic movement, or rather the small ganglia that possess an independent action of their own, though controlled in their different ways by the vagus and splanchnic, the latter being the largest vasomotor nerve in the body. The view that the splanchnic arrests intestinal movements by causing contraction of the intestinal vessels, and thus expelling from the walls of the intestine the blood which would stimulate them to movement, has received additional confirmation from the experiments of Von Basch, which show that when the peristaltic

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movements occur after the injection of nicotine, the blood-pressure continues to sink, indicating that the intestinal vessels have become dilated. The increased movement is attributed to the dilated vessels allowing more blood to circulate in the intestine, and thus increasing the stimulus to their action. When the splanchnics are irritated the intestinal vessels contract, the blood-pressure rises, and the peristaltic movements cease. The arrest of peristalsis occurs at the same time that the blood-pressure reaches its maximum. This view is further supported by the fact that Meyer and Von Basch found that the intestinal movements can be sometimes arrested by stopping the circulation, either by ligaturing the thoracic aorta or by irritating the vagus until the heart ceases to beat. But although the inhibition of peristalsis seems to depend mainly on anæmia of the intestinal vessels, the anatomy of the ganglia in the coats of the intestine and their connection with the vagus and spinal nerves from the lumbar cord prove that this is not the only factor. Probably, in ordinary peristalsis of health, the secretions that enter or are found in the intestine, and pre-eminently the bile, are sufficient to reflexively stimulate the ganglia in the intestinal walls without, so to speak, troubling either the splanchnics or the vagus or the spinal nerves. The absence or perversion of these secretions may, therefore, induce constipation negatively. In the most common form of jaundice depending on catarrh of the bile-ducts, and still more frequently on slight catarrhal puffiness of the disordered mucous membrane, preventing the discharge of the bile from

the common duct into the intestine, constipation results from this very cause—that the intestine is deprived of its wonted stimulus to the minute ganglia in the intestinal walls, the exodic response to which is shown in peristalsis. The opposite condition, diarrhœa, might be caused by taking away the ganglia of the solar plexus. The immediate result will be dilatation of the intestinal vessels, thence stasis, thence œdema, which, instead of infiltrating the intestinal cellular tissue, shows itself as an exudation in the intestinal canal. After the extirpation of these ganglia, there is no longer any inhibition of the ganglionic plexus of the intestinal walls; these, therefore, take on an exaggerated action, causing increased secretion.

In ordinary circumstances diarrhœa is reflex; it may be from cold, the first impression being on the skin, carried thence to cord, and thence to the abdominal ganglionic plexus. Enteritis may cause reflex diarrhœa; so also the diarrhœa from dentition, and possibly also that influenced by emotion, especially by fear; but emotion often consists in a complication of reflex acts, having its primary influence on arterial tension, thence through the heart, involving the depressor nerve of Ludwig, thence through the medulla oblongata and the spinal cord, the splanchnic, thence allowing dilatation of intestinal vessels, and so inducing not only increased peristalsis but a large amount of exudation fluid. That articles of undigested food, foreign bodies bearing morbid secretions, can reflexedly excite increased peristalsis and diarrhœa is a matter of daily experience. The

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exudation thus induced is not the ordinary secretion of the intestines. It is more fluid, and contains not only numerous leucocytes but often also some red blood-globules. Increased peristalsis also is not a necessary accompaniment to increased exudation. This is seen in the action of some purgatives. The irritation of certain substances or secretions in the intestines may influence, not the ganglia that rule the intestinal movement, but the ganglia that rule the calibre of the vessels. Thiry and Radziejewski have fathered a view that purgatives act, not by inducing a greater flow of liquid into the intestine, but by exciting movements more or less intense of that part of the digestive canal. The intestinal liquids in health are being constantly reabsorbed. But if the intestinal movements become, from any cause whatever, more energetic, these liquids are thereby pushed onwards from small intestine to large, and thence to the anus, without remaining long enough in any one spot to be reabsorbed. This is their theory of the action of purgatives. The objection to this view is, 1st, that the peristaltic movements are often not more energetic after purgatives than in health; 2nd, that the secretion of the intestinal liquids is remittent, and not, therefore, constant enough to explain the diarrhœa. Quite irrespective of increased peristalsis, salts, for instance, cause a considerable flow of morbid fluid into the intestine simply by osmosis, by contact of the purgative saline with the intestinal mucous membrane. It is possible that this large amount of exuded fluid may in its turn stimulate the ganglia,

and so bring on increased peristalsis. This may or may not be in any given case; but the effect of the saline has *per se* no influence on peristalsis. Jalap gives rise to the same phenomena. Radziejewski's opinion, that evacuations produced by purgatives are not constituted by the products of transudation but by the ordinary contents of the bowel, is refuted by experiments on isolated portions of gut. By these experiments it is seen that salts cause no increase of peristalsis, but that drastic purgatives do, probably at the moment when the secreted liquids commence to accumulate in the gut. The liquid poured out under the influence of a purgative seems to be the result of a catarrh of the intestinal mucous membrane, and this slight catarrh is the primary effect of a purgative. Such a catarrh can only occur with dilatation of vessels, due, if the stimulus of the purgative be slight, probably to reflex excitation of the vasodilator nerves of the intestine; if intense, to paresis of the vaso-constrictor. This is certainly the case with sulphate of magnesia, the exhibition of which causes evident irritation of the intestine, at all points that come into contact with the purgative substance, and this without any remarkable movement of the small intestine.

In some of the experiments on purgation it was incidentally remarked that intestinal contraction is sufficient of itself to expel fæcal matter through the anus, as the abdominal wall having been opened for purposes of observation and experiment, all intervention by the action of the abdominal walls

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was rendered impossible. In ordinary cases, however, intestinal peristalsis is materially assisted by the contraction of the abdominal muscles. It is with drastic purgatives that, besides the exudation thrown out on the intestinal mucous membrane, there is usually an inward peristalsis, the difference in this respect between their action and that of the milder drugs being one of rapidity of action and energy of movement.

Purgative substances injected into the veins do not always set up purgation.

They certainly do not induce the catarrh of the mucous membrane just spoken of, unless they are of a nature to be carried to the intestine through the general circulation. Although Ch. Bernard found aperient effects from the sulphate of magnesia when thrown into the veins (probably because it was carried to the intestinal mucous membrane), yet the rule is the other way. Aubert and Rabuteau at different periods have performed experiments on this point. The latter observer even thought he had induced constipation by the injection into the crural veins of some sulphate of soda, because instead of exciting exosmosis, as it would if inserted into the intestine, an osmotic current, the inverse of this was produced, that is to say from the interior of the digestive canal towards the blood circulating in the mucous membrane of the bowel. A similar negative result is met with by injecting purgative substances hypodermically; if the amount used is large a local inflammation is set up at the seat of injection, which prevents

the substance injected being absorbed. If, however, very weak solutions are used purgation will result. If the whole process of the action of purgatives is followed anatomically, the important part played by the sympathetic system is seen. Thus purgatives, introduced by the mouth, irritate the mucous membrane of the gut. This irritation modifies the intestinal epithelium, and excites the peripheral extremities of the centripetal intestinal nerves. This excitation is carried to the lower thoracic nervous ganglia and the intra-abdominal (the ganglia of the solar plexus, of the mesenteric plexus, of the plexus of Meissner and of Auerbach); thence it is reflected by the vasomotor nerves on to the vessels of the intestinal walls, and by the secretory nerves on to the anatomical elements of the mucous membrane, to the glands of Lieberkühn amongst others. There results a more or less intense congestion of the intestinal mucous membrane (a reflex vasodilator action), a desquamation of epithelium, with a rapid and abundant production of mucus, with or without emigration of leucocytes; and an active secretion of intestinal fluid, with which in certain cases are mingled the products of a profuse transudation, formed chiefly of water and of certain salts of the blood, and due to the exaggerated and vitiated work of which the elements of the membrane are the seat.

Whatever the theory of the causation of cholera be—whether the symptoms are due to the comma

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bacillus or to some other microbe—in some points of view the sympathetic is not unaffected, and its implication accounts for some of the phenomena. Thus Marey believes 'that the original poison excites the sympathetic; as a result of this, vascular spasm is seen, preventing the passage of the blood, enfeebling and even suppressing the pulse in the contracted vessels. This retraction of the vessels diminishes the volume of the tissues, and thus thinning of the nose is met with, and the falling in of the eyes within the orbit. There is coldness of the superficial organs, whilst the reflux of blood towards the viscera raises their temperature. The muscles of the bronchi are affected by spasm, whence results defects of oxidation, and so cyanosis. The blood thus charged with carbonic acid induces cramps. The second period is one of vascular reaction.'

Most physicians believe that the sympathetic plays no part. The rationale of the morbid phenomena is that the poison acts directly on the intestine, causing desquamation of the epithelium. The mucous membrane, deprived of its epithelium, allows a great part of its serosity to transude. The blood, thus deprived of some of its serum, thickens and cannot circulate properly, nor is it fit for purposes of nutrition. The loss of serosity also lowers arterial tension, and as a consequence of this there is suppression of secretion, especially of the urine. Nutrition is interfered with. The heart, badly nourished, contracts feebly. The circulation is hindered, and the blood, therefore, is badly oxidised in the lungs. The blood-globules

become altered from the presence of carbonic acid. Others think that the nervous system is affected as well as the intestinal, and even before it. Certain it is that the nervous affection may kill before the intestinal phenomena occur.

Eulenberg believes that the disturbances of circulation are similar to what are observed in contusions of the intestine; that the circulation is hindered by two causes: (1) by the loss of serous fluid; (2) by the propagation of the irritation of the intestinal nerves along the cord to the bulb, so exaggerating its inhibitory action on the heart.

Nelter attributes the highest importance to intestinal transudation. The vascular constriction is only the consequence of this initial phenomenon. The toxic agent limits its action to the intestine. It provokes within the gut a transudation, which deprives the histological elements of the mucous membrane of the water necessary to them. By reason of the power in the cell-life, these elements experience a true need of the water. They call upon the artery of the local circulation, and the artery in answer contracts, and thus pushes towards these elements the liquid demanded. Thus dilating anew it draws fresh supplies of blood from the principal arterial trunk. This suction is incessantly repeated whilst the transudation continues, and the calls for fluid by these tissues weaken more and more the mass of the blood and the fluid of the elements of other tissues, which in their turn experience the same need, and the cellular thirst, which was at first local, becomes general.

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The true theory is that the poison, when by any channel it enters the body, produces a toxic effect on the whole system. The nervous system is affected, like everything else, not exclusively. The channel for stimulation is the intestinal canal. The work of elimination presupposes contractions of the intestines, perhaps, too, the excitation of the secretor nerves, and thus the part played by the nervous system is seen. The contraction, too, of the vessels of the periphery is a direct effect of the poison in the nervous system, as it occurs quite independent of the alvine discharge. It is a true tonic spasm. This vascular spasm is less in degree but the same in kind as in arsenical poisoning. It must be supposed that the malaise experienced by the heart from the reflux of the blood from the periphery is immediately transmitted by the nerve of Cyon to the vasomotor centres, dilating the abdominal vessels notwithstanding the general vascular contraction elsewhere; and thus the action of the sympathetic leads to the loss of serous fluid, the object of which is doubtless to free the system from the toxic agent that induced the initial phenomenon. From every point of view, therefore—the constriction of vessels of the periphery, the state of the heart, the implication of the nerve of Cyon, the vascular dilatation of the intestinal vessels, and the involvement of the secreting nerves of the intestine—some portion of the sympathetic plays an important part—not in the causation of the disease, but in the production of some of its morbid symptoms.

It may be noticed also that in cases of cholera

the semilunar ganglia have been found diseased by Delpeth, of Montpelier, Loder, of Moscow, and others.

Consult—

Dr. Clifford Allbutt, 'Gulstonian Lectures.'
Chapin, op. cit.
Vulpian, op. cit.
Quain's 'Dict. of Med.'
Seeligmüller, op. cit.
Dr. Warburton Begbie, Reynolds's 'System of Med.'
Eulenberg and Guttmann, op. cit.
Habershon, 'Diseases of the Alimentary Canal.'
Wilson Fox, 'Diseases of the Stomach.'
Briquet, 'Traité de l'Hystérie.'
Handfield Jones, op. cit. p. 43.
Von Braam, 'Med. Times,' 1873, ii. 260.
Nothnagel, 'Brit. Med. Jour.,' 1882, i. 790.
Nothnagel, 'Lancet,' 1882, ii. 671.
Von Basch, 'Stricker's Medicinische Jahrbuch,' 1874, 45.
Poincaré, op. cit.

CHAPTER XV.

NEURASTHENIA.

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A VERY large class of cases, accompanied by various physical and mental phenomena, often resisting all treatment and constantly resulting in spontaneous recovery, occurring, too, almost exclusively in the female sex, has always ranked among the opprobria of medicine. From the connection that in some cases exists between the abnormal symptoms and the uterine functions, this multiform disorder has been termed hysteria; and the same word has been used where no connection can be traced between the symptoms and the generative organs; and considering how many phenomena are reflex, and how frequently the starting point of reflex action lies in the intestinal and pelvic viscera, and how many morbid symptoms, bodily and psychical, disappear after proper treatment directed to the pelvic viscera, and how pigmentation and irritable vomiting are connected with pregnancy, it is not surprising that too great an importance has been attributed to abnormalities of the uterus and its appendages, and that hysteria has been accepted more or less roughly as a fair explanatory term. As the protean groups of sym-

ptoms hitherto classed under this term are really neuroses, depending on a diseased nervous system, generally hereditary or with a causation derived from one or both parents, it is more in accordance with experience to use a word like 'neurasthenia,' in preference to one that seems to fix the causation too entirely on the pelvic viscera.

Unlike hysteria, the name cannot be restricted to such patients whose complaints are considered to be more or less exaggerated, or who are not bonâ fide anxious to get well and to cease from being the objects of nursing and sympathy. In many cases, women, apparently of healthy religious tone and of good common sense, are the victims of this malady, when the practical nature of their daily duties and the desire they manifest to return to active life are evidences that, as far as their will is concerned, there is a bonâ fide anxiety for recovery. Is it not the custom to generalise too much on this subject, and to class together a number of nervous ailments, which, resembling each other only in external manifestations, are really dissimilar both in physical and psychical causes. Then, too, must not a distinction be drawn between a girl who, whilst she complains of pain in some special part, is yet so universally sensitive that she shrieks out on pressure on any part of the body, and another who, without this abnormal sensitiveness, yet suffers from true hyperæsthesia of certain nerves? In each case the sensation of pain may be highly exaggerated, but the sensation of pain is very subjective, and the physician has no

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data wherewith to measure it. The pain is probably a great reality to each patient, and if one professes to suffer agony from an external impression which in another would cause no remark, it is no more right to call the former hysterical in the ordinary offensive sense of the word than it would be in the case of a person whose sense of sight or of hearing may be unusually acute. A vast number of patients are highly emotional, and this condition may be accompanied by very various physical symptoms, and by the manifestation of a morbid temper or of a weakened or perverted will. The list may include the girl who is the subject of globus, on the one hand, and patients suffering from mania on the other. But as diagnosis becomes more accurate, it is possible to assign many of these cases to causes other than the so-called hysterical. In a well-organised brain the intellectual and the emotional faculties exercise a mutual influence and check upon each other. In childhood, before the intellect is ripened, the emotions are supreme. In after life, in seasons of weakness or exhaustion, they reassert their sway. In cerebral softening, or in paralysis following cerebral hæmorrhage, they again obtain the mastery. And thus in many cases of neurasthenia, where the intellectual faculties are weak originally, or have been interfered with by debilitating causes—sometimes, it may be, from an epidemic moral influence—it is only natural to expect that the emotions should become more or less beyond control, and that emotional manifestations should give rise to very prominent symptoms.

Chlorotic patients are not particularly subject to the emotional symptoms of neurasthenia. They are often somewhat melancholic and depressed, but by no means excitable. In advanced conditions of the disease they are even apathetic. But they suffer from some of the physical evils to which the purely neurotic patient is subject—borborygmi, constipation, amenorrhœa, palpitation, dyspnœa. In some cases also chlororis is accompanied by morbid fancies and exaggerated ideas of their own illness, and perhaps occasionally there may be no desire for recovery. There is, however, this marked point of distinction between the simply chlorotic and the neurotic patient, that the idea of her own condition in the former, exaggerated as it may appear, may be justified by the event, chlororis being a condition leading to a fatal termination sometimes. In speaking of the possible causes of neurasthenia, it will be found that the same circumstances which tended to produce one condition have a similar tendency to the production of the other, and that, therefore, the two diseases do not stand towards each other in the relation of cause and effect, but are connected only as owning a common parentage. Neurotic symptoms, however, occur commonly in people who are either of a weak constitution or who have become more or less debilitated by some exhausting process. This statement is denied by a high authority, but it surely is in accordance with experience. It is not an unusual thing to see women, who when in health are naturally strong-minded, practical persons, and very far removed

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from neurotic phenomena, yet during the convalescence from an exhausting illness become emotional in the highest degree. The same thing is constantly observed in men also. It is a state that differs only in degree from the maniacal delirium sometimes seen at the close of acute diseases. A phthisical patient, for instance, who during a long illness had shown herself to be an amiable and religious person, became suddenly maniacal twenty-four hours before her death, screaming with the utmost violence and using blasphemous language, at the same time endeavouring to strike everyone that came near her. In another case, a woman with old-standing disease of the heart and dropsy, but without albuminuria, was pregnant and very anæmic. She became maniacal after only a few hours of restlessness, in which she seemed suspicious of all around her. This patient was possessed with the delusion that parturition had taken place, and held up her nightdress as evidence of the truth of her statement, saying that it was covered with blood, whilst in reality it was unstained. Here was delirium with hallucination. Neither of these cases depended on toxæmia, but simply on a want of due blood-supply to the brain. Such a condition is, however, only the ultimate phase of a state which usually commences in a more gradual manner with confusion of ideas, and proceeds to morbid judgment with illusions and delusions.

When these symptoms depend on prolonged lactation, the disease is generally more tedious, and in some cases more serious. Under these circum-

stances there are not only many symptoms of physical debility, but great melancholia with depressing emotional phenomena. This melancholia is often accompanied with morbid thoughts and imaginations, with great fear of insanity, with dark and sorrowful views both of this life and of the future, with temptations to vice and even to crime, and with extreme irritability. Except under circumstances unusually favourable, the physical prostration, the cares of family and household, the hold which such views take upon the mind before the patient seeks advice, are causes sufficient to render the recovery tedious and difficult, although ultimately certain. The morbid fancies, the perverted will and temper, as well as in many cases certain physical phenomena, palpitation, hyperæsthesia, tympanites, globus, &c., are the connecting links between this condition and what is seen in hereditary neurotic patients.

In some of these cases it is worthy of remark that suspicion is a very prominent symptom. The patient thinks that everybody is speaking or thinking of her, or plotting always to her disadvantage. There is a morbid self-consciousness about her, allied to that which induces an insane person to believe that persons are constantly stamping at his door, or that officers of justice are pursuing him; only in the latter case the patient is generally suffering from pure delusion, in the former it is merely illusion. She does not fancy the existence of people who never existed; but from morbid imagination, she suspects and

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attributes wrong motives for the simplest actions of people present.

A good many neurotic cases are full-bodied healthy-looking young women, with rather more than an average amount of strength. In many of these cases there is more or less impairment of the catamenial functions, more often in the way of diminution than of excess. Where, however, the latter condition exists, and the catamenia are either excessive or too frequent, the case must be tabulated with those that depend on anæmia or other debilitating causes. In women with this general appearance of good health, the more usual uterine disturbance is amenorrhœa. Excluding phthisis and other morbid diatheses, amenorrhœa may be due to one of five causes.

1. Arrest of development. The uterus and ovaries may not be developed in the same ratio with the rest of the body, and especially with the brain.

2. Sudden fright and excitement of any kind may stop the catamenial flow, and both the mental disturbance and the amenorrhœa to which it gives rise will assist in the production of neurotic phenomena.

3. In women, sexual excess not unfrequently has this effect, without the supervention of pregnancy; but in these cases the amenorrhœa is generally of very temporary duration.

4. The most common cause in women of this description is exposure to cold and damp during the menstrual period.

5. Excessive brain work, perhaps especially in

the early years of puberty. It is well also to speak of pregnancy in the unmarried female as a frequent element in the causation of neurotic symptoms, but this not so much from the resulting amenorrhœa as from the mental and moral disturbances consequent on the expectation of future discovery. The dependence of neurotic symptoms on some one or other of these causes of amenorrhœa, or their disappearance when the amenorrhœa has been successfully combated, afford evidence of the exciting cause being certainly sometimes in the pelvic viscera, though even here the phenomena are generally met with in persons with weak nervous centres.

There are, however, other uterine conditions besides those of excess and diminution of the catamenial flow, which share with them as the starting-point of neurotic phenomena. Chief among these are the various forms of dysmenorrhœa. Dr. West has described these symptoms under the three forms of neuralgic, congestive, and mechanical; and these divisions are practically of great importance, although cases occur in which these forms may run into each other. Thus, in a case constantly under the writer's care, in which hysterical convulsions, associated with very intense headache, mark almost always the catamenial access, the lumbar and uterine pain was believed to be due to mechanical malposition of the uterus. Anteversion of this organ was found and relieved, and for three times the menstruation passed over without difficulty; but dysmenorrhœa recurred without any malposition of the uterus, and was

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associated with facial neuralgia. In the same case there had been, at times, reason to believe that dysmenorrhœa depended on a mixture of the two causes. Of these forms, perhaps the congestive is the most easily remedied, whilst the two other forms are more frequently accompanied by hysterical symptoms, associated sometimes with cerebral phenomena of a serious nature, such as rambling delirium, from which it is occasionally impossible to rouse the patient to a consciousness of surrounding circumstances. These points are of importance with reference to the part played by the vasomotors in this neurosis. There are other women who not only have the appearance of perfect health, with normal catamenial functions, but yet constantly manifest some neurotic symptoms. In these there is often a prurient disposition, hereditary or acquired. In so-called hysterical mania, the worst form of disease, it is generally possible to trace the mental infirmity gradually growing up, step by step, from phenomena purely hysterical to attacks manifesting the most aggravated symptoms of insanity. More especially is this the case in the form that bears the name of religio-eroto-mania. In many of these cases uterine disturbance is sought for in vain, nor can there be traced, either in disappointed affections or in fanatical religious influences, a cause for the insane symptoms. But masturbation has often much to do in the production of this condition. It would be of course unjust to consider masturbation an invariable cause of the neurotic symptoms in such patients, but it

would be against experience not to look upon the probability of the existence of this habit with grave suspicion. In many cases it is not the cause but the effect of grave disturbance of nervous centres. With many others of the same class it is open to question whether some of the abnormal nervous phenomena are not evidence of the struggles of a refined and modest person—struggles only partially successful—against the temptation to words or acts of an indecent nature, such as in a more advanced stage of the disease, when conscience is silent and the faculties are dulled, are shown in all their naked enormity in the wards of an asylum. The writer has seen somewhat similar symptoms in the case of a person from whom both ovaries had been removed. About the time of the monthly periods the ideas were distinctly sexual, as though the absence of the catamenia rather encouraged the mental feelings on this head.

Other circumstances, however, besides those connected with the sexual passion, may affect a mind naturally weak, and give rise to hysterical conditions. Certain religious impressions, especially such as involve whole districts at once—as, for instance, the dancing mania of the middle ages—would act as an exciting cause. But putting aside these great epidemic influences, sporadic cases are constantly met with in which some particular conversation, some religious shock, the influence of some special person, may act as excitants on a weak brain.

Closely allied to this condition is that produced by the dull ascetic solitude sometimes enjoined in

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convent life, and which, added to a defective education and superstitious instruction, gave rise to the dreams, the visions, the celestial appearances so common in the lives of some of the Romish saints. Even scientific men have found it difficult to avoid giving credence to certain hallucinations, when the body and brain have been weakened by long study or excessive abstinence. It is not easy for the strongest persons under such circumstances to resolve into the bare facts of abnormal cerebral circulation and overwrought nerve tissue the objects of thought which seem to take visible form before the eyes. How much more difficult must it have been for the old ascetics, to whom supernatural appearances were matters of faith, and whose healthy feminine instincts towards charity and usefulness were fed with abstractions instead of realities?

Another phase of neurotic patient is the touchy woman, one who is not associated with by her friends or the world at large, one who cannot live with her relations because they consider her illnesses nonsense, and will not give her the quiet and repose so necessary she thinks to the state of her health. She generally is past her first youth, and has had her disappointments. There is usually in her a vast amount of fine feeling, too fine for the rough usage of the world. She has the largest views of universal benevolence, only hindered by her health, or her means, or the circumstances of her life. She lives a restricted life of self-concentration, taking no interest in any other thing or person, living in herself, to herself, and for

herself. To such a person a state of chronic ill health is a necessity. She must have attention from some body, and tries therefore to get it from her doctor, or more usually from a succession of doctors. She is often anxious to extract a certificate of disease to show her unbelieving relatives. She is generally suffering, in her own idea, from a mortal malady. One person under the writer's care insisted upon having, at the same time, cancer, consumption, and stricture of rectum. Under the latter impression, she had for many years performed defæcation by scooping out the rectum with her fingers, and could never be dissuaded from this habit, although the necessity for it existed only in her own imagination. She was at the same time perfectly sane on any subject which did not touch on her own health.

Other cases, closely connected with these, are the subjects of 'temper disease.' Marshall Hall recognised this as a distinct morbid condition. The ebullitions of temper sometimes occur at intervals, the patient at other times being calm and sensible. Often, however, the usual condition is one of chronic discontent, interrupted with bursts of passion. Occasionally these outbursts alternate with symptoms of a purely neurotic character. It is extremely difficult to determine at what age, or after what length of time, the controlling power of the will ceases to influence such patients. Perhaps a strict moral control in early life would do much towards preventing the further development of the evil. But after a certain period the habit of uncontrolled

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passion, and still more of looking at everything from an ill-tempered discontented aspect, grows so strong that the will is absolutely powerless in resisting the morbid condition. With some, too, even in early life, the outbursts of violent passion, under circumstances and causes of a most trivial nature, seem to be insane impulses, and to be entirely beyond voluntary control.

In others, the nagging ill-conditioned mind, which looks upon every plan as directed against herself, every word as having some covert meaning to her disadvantage, and which carps or sulks at all the circumstances of daily life, is at once one of the most trying and most pitiable objects of humanity. The selfishness, the wholly subjective life of such persons, the absence of interest in external things, are the connecting links between these patients and many other phases of the neurotic condition.

In speaking of the general appearance of such patients, there remains a class distinguished from others by a more advanced age. At a period when the catamenial functions are gradually ceasing, nervous conditions, very closely resembling in symptoms some varieties of hysteria, are commonly met with. This climacteric period is frequently accompanied by great irregularity of the menstrual periods. and by some feeling of malaise which is evidence of disturbed organisation. The irregularity is either shown in a gradual decrease in the amount of uterine discharge, the flow occurring at regular monthly intervals and becoming less and less until it finally

ceases altogether; or in a somewhat sudden cessation for several months, and even for a year or more, followed by an increase of discharge amounting to menorrhagia, with a recurrence of this alternation of symptoms perhaps for several years; or in a great increase of the menstrual flow both in amount and in frequency of recurrence, the menorrhagia being either persistent or stopping for a few days, to be renewed with increased vigour. It is evident that in the latter condition there is irritability of the generative organs, as well as debility from the constant hæmorrhage, either of which would be sufficient to produce hysterical phenomena; and it is in patients who suffer in this way that these symptoms are most common.

Dr. Laycock's remarks will be easily endorsed by all practitioners. 'The vigour of the reproductive system begins to decline about the age of forty or forty-two, and from this period to the age of forty-nine there is a state of system exceedingly analogous to that of the period during which it was first developed. The morbid phenomena are however favourably modified by circumstances. Age has blunted the sensibilities in some degree. There is less of passion, less disappointment, less mortified vanity and fewer causes for indulgence in evil tempers and foolish caprices, while those mental ills which unadvoidably happen are soothed by more or less of religious feeling. The hysteria is of the cachectic type; the uterus and mammæ, or, if the patient have not had children, the thoracic viscera, are principally

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affected; asthma, angina pectoris, menorrhagia, neuralgia of the mammæ and their concomitants are observed; and when the catamenia cease, if the health be not re-established, gout, hypochondriasis, or general cachexy supervene, and cancer attacks the reproductive organs or their glandular appendages.' Dr. Laycock probably means by angina pectoris a reflex phenomenon acting through the sympathetic, as true angina pectoris will hardly occur as an hysterical symptom. A spasmodic respiration, simulating asthma, and a cardiac neuralgia of great severity, simulating angina pectoris, are met with in such patients, in whom the bronchial tubes and the heart are perfectly healthy. Occurring under such circumstances, it is usual to see all such symptoms pass entirely away, when the general health has been re-established after the complete cessation of the catamenial functions.

And that the pelvic organs should be the starting-point of neurotic symptoms is no more remarkable than that peculiar emotional phenomena may be caused by the irritation of concretions in the pancreas.

Apart from these varieties of hysteria, cases are met with owning no uterine cause for the nervous symptoms, and yet during the whole of their lives forming the subject of certain nervous peculiarities. They are women of intense superlative mode of speech, who cannot look at any question calmly, are flurried on the least excitement, terrified by the least misfortune. They suffer from acute sensibility of

the nerves of touch, from abnormal sensations in the stomach and bowels, from neuralgia, palpitation, faintness, &c. In a word, they suffer from the neurotic temperament, a very real disorder, though one that may afford no post-mortem record. Such a state is largely due to hereditary influence, and is often seen in the children of drunkards. It is a condition, too, that frequently leads to a habit of alcoholic excess.

In speaking thus of the varieties of the general appearance of hysterical patients, some of the causes of this condition have been touched upon. It is necessary to speak of them more fully. The nervous temperament may be transmitted from either parent, but perhaps most from the mother. If the mother has suffered from so-called hysterical symptoms, if she has been nervous, excitable, emotional, still more if she has suffered from any form of insanity, the daughter may suffer. As has been said above, alcoholic excess in one or in both parents is a fertile cause for this morbid condition. This is only in accordance with what we have found in more serious states of mental deterioration. Morel was only giving the results of general experience when he wrote, 'Les habitudes d'ivrognerie chez les parents ont été signalées aussi, et je ne puis passer sous silence une cause que je regarde comme des plus actives dans la production de l'imbécillité congénitale. Dans son excellente statistique des causes d'aliénation mentale dans la province de Westphalie, le docteur Ruer a déjà fait observer que le nombre des enfants idiots et imbéciles

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augmentait d'une façon notable dans toutes les contrées où l'ivrognerie était un vice commun au père et à la mère.' What is here said of idiocy and imbecility may be stated with truth of other forms of mental alienation, and later investigations teach that the hereditary influence of drunken parents may not only induce a neurotic temperament in the succeeding generation, but many nerve phenomena, some at least of which are accompanied by coarse lesion of the cerebro-spinal centres. The effect, too, of the training given by such parents will not tend to subdue passion, selfishness, or excitability.

Another cause may be found in the mother living in a whirl of social excitement. The character of the life and the fatigue engendered by it will often have a morbid influence on the offspring.

The whole question of parental influence on the child during intra-uterine life is involved in almost as much obscurity as hereditary tendency to phthisis, cancer, or gout. It is a matter of daily experience to have marks on the child attributed to ungratified wishes of the mother, during pregnancy, for certain articles of food. The writer has met with a case in which the fingers of the child being united together like the feet of a web-footed bird was attributed by the mother to her desire for roast goose during her pregnancy. Many, too, of the malformations in the infant are referred to the sight of strange objects during pregnancy, objects which at the time made no impression on the mother at all. Such statements are worthless. But it is a very different question how far a real mental

impression, especially one of terror or of grief, may not cause arrest of development in the embryo, just as in some cases it may induce abortion. When the part played by terror in exciting certain neuroses, chorea, hysteria, epilepsy, is considered, emotion must be looked upon as a powerful influence for evil on the body, acting mainly by way of the vasomotor nerves, and thus affecting the circulation. Any cause which interferes with the due course of the maternal circulation will be liable to produce one of two effects on the fœtus—malnutrition evidenced by arrest of development, or abnormal nutrition inducing malformations either in excess or defect. This malformation has of course no reference to the particular object of terror on the part of the mother. It may be said that this effect is much less direct, and depends on the condition of ill-health into which the mother may be thrown by any sudden shock during pregnancy.

But in those cases in which the effects of maternal emotion are not seen in external abnormalities of development, the induced cerebral conditions of the infant are often very severe. In many instances they reveal themselves to naked eye observation; but they vary extremely in degree, and where no anatomical lesion is discovered, whilst the patient shows deficiency of control and a want of co-ordination between various portions of the brain, it is only reasonable to believe that the circulation has been in fault and the development of part of the brain, or of one of its layers of cells in some situation, has been arrested.

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Maternal shock during pregnancy may induce defective cerebral organisation in the child, and after birth this defective organisation may manifest itself in psychical peculiarities, which later on may take the form of neurotic phenomena.

Although this neurasthenia is mainly a disorder of the female sex, it is met with in boys and men, either from hereditary taint, from exhaustion, from the effects of dissipation, sometimes from over study. From the age of fourteen to eighteen, and again from forty-two to forty-nine, the periods of the development and decline of the uterine functions, women are most liable to conditions that may be justly termed hysterical. But not exclusively so. Just as the practice of self-abuse is found to exist long before it is possible that any seminal fluid can be excreted, so in the female, hysterical symptoms may precede for some years the catamenial functions. Such cases are met with at the age of eleven and twelve, and even earlier, not in children of precocious uterine development, but generally when there is a strong neurotic tendency, or where they have constantly before their eyes the example of persons so affected. So also, long after the menstrual functions have ceased, the nervous system in some persons seems unable to shake off the morbid habit, and the woman remains hysterical even to advanced age. This may be only an example of that tendency to periodicity of nervous phenomena so constantly seen in neuralgia, in asthma, and in epilepsy.

Connected with this question of age is the mental and moral condition of young women about the age of

puberty. There is no period of life at which both the moral and physical nature are so liable to deteriorate as between the ages of fifteen and twenty. The girl's mode of thought and feeling are altered; and it is at this period that the gentleness, purity, and refinement of the mother exerts such influence for good or evil upon the child, an influence essential to the due and proper balance of the human mind. It is at this age, too, that diseases of the nervous system, through direct alteration of the blood elements circulating through the nervous masses and ganglionic centres, are so frequently noticed; and, as results, vitiated sensations, perverted emotions, or even disturbed intelligence may occur. This period of puberty, too, is remarkable for a great change in many habits and tastes. Many girls would gladly live without any animal food at this time; and this dislike for meat will persist for two or three years. The affection of girls for girls at this period falls far below that of boys for some special school-companion, and contrasts markedly with the love of little children, and still more with the depths of affectionate self-devotion so frequently manifested by these very girls in after life. Much of this change of moral habit is due to the weakness induced by over growth, whilst at the same time the brain is making great calls on the circulation for carrying on the studies of school life. Much, however, depends on the special condition of the generative organs. The dragging pains in the back, the commencement of a whole series of new sensations, the irritability which is partly the consequence of these sensations and partly of weak-

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ness, the marked disturbance of circulation, all these tend to fix the thoughts of the individual on herself, and to prevent that diffusiveness of affection which is most opposed to selfishness.

It is not certain that any special employment can be charged with causing hysteria, but many occupations are harmful from one or more of four evil influences. The length of time, the vitiated atmosphere, the constrained posture, and the monotony of the occupation may each and all lead to great neurotic tendencies among many other morbid phenomena, and the writer believes these tendencies can be set up *de novo* in the members of non-neurotic families. It would lead too far to enlarge here upon each of these four points. Generally, they are quite unnecessary; and it is very remarkable how frequently the master of a shop will take trouble to ventilate his premises, whilst he will not listen to the suggestion of allowing his assistants to sit down, even at a time when they are not attending to customers.

Among the affluent and luxurious, neurotic complaints are peculiarly common. Wealth and common sense are often very far from going hand in hand. A girl brought up in luxurious habits will lie to a very late hour in the morning in a soft bed, its very softness being an evil will not dress herself, will have everything brought to her throughout the day, will take no personal trouble, and make no personal exertion; will possibly go out lying back in an easy carriage, and will often become gradually so sated with the tedious pursuit of pleasure, that she will be

incapable of being interested in the facts and incidents of daily life. It is not easy to say whether, in such cases, the neurotic symptoms are induced by the physical conditions consequent on luxurious habits, or by the mental discontent from the ennui of such a mode of life. Quite apart from the many habits that may induce functional uterine malady, the unsatisfactory sensation of the want of something to do, impossible to define, the sense of injury, and of want of attention, and the failing to obtain the precise position with reference to other people to which she thinks herself entitled, will make a girl magnify slight ailments, with the view of insuring attention from her relatives and her doctor; and, if this plan is successful, will cause a continuance of the simulated ill-health, which will result in protracted and persistent hysterical paralysis or hyperæsthesia. This morbid mental condition, this longing for notoriety, this desire to be first in some way, in however small a circle, is at the bottom of many of the strangest phenomena in such cases. It leads not only to the most remarkable physical symptoms, but even to self-accusations of vices or of crimes of which, in act at least, the patient is wholly guiltless.

Not only are special employments and special habits conducive to neurotic evil, but certain diatheses and temperaments are more subject to it than others. Thus, with some families the temptation to lustful pleasures is so strong that it amounts to hereditary disease. In well-principled members of such families the tendency to early marriage is great.

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Among men, if such marriages are impossible, life becomes a daily struggle against temptation. But for women, owing to social customs and habits, the case is far worse; the choice between sexual feeling and married poverty is not in their hands. The temptation is probably in many cases the same, and is combated heroically both on principle and from natural modesty: whilst the very acknowledgment to themselves of the necessity for such a struggle seems to them desecration. For many a long day there is no external manifestation of it even in look. The patient may throw herself into all kinds of useful occupations, but still the trial continues, and the result may be self-abuse, or thoroughly bad habits of life, or a form of nerve disease which is not unlikely to terminate in eroto-mania. In this latter condition the mental malady is not, as is sometimes the case, the primary cause of the sad physical manifestations, but, on the contrary, the physical condition with ungratified desires leads gradually to the mental aberration, with apparently loss of all sense of shame and modesty. This seems often to be the explanation of those strange abnormalities of the moral sense which are met with occasionally amongst women of high principle and religious feeling, and are worthy of all possible pity.

One more point connected with the generative functions is often overlooked in determining the causes of this malady, viz., marriage, in which the natural desires for conjugal intercourse are interfered with, or again in which such intercourse is indulged

in to excess. In one of the most marked instances ever met with by the writer, this intercourse constantly took place many times in one night, and the husband eventually died in an asylum, utterly broken down by this excessive indulgence, whilst the wife was the subject of aggravated neurotic symptoms. This cause, however, is not so common as its opposite. From various circumstances, disparity of age, want of affection on one side or the other, incompetence from ill-health, or habits of illicit intercourse on the part of the husband, there are some married women in the position described. To quote M. Morel once more, 'La proportion de l'hystérie est moindre sans doute dans le mariage, et Ambrose Paré l'avait déjà remarqué en disant que ces accidents adviennent peu souvent aux femmes mariées ayant la compagnie de leurs maris. Mais, ajoute avec beaucoup de justice M. le docteur Landouzy, il ne faut pas voir dans ces mots " ayant la compagnie de leurs maris " une naïveté ou un pléonasme, mais le résultat d'une observation que font chaque jour les médecins, et que devaient faire peut-être les philosophes et les législateurs. Combien de femmes, en effet, dit cet auteur, n'ont pas la compagnie de leurs maris, parce qu'ils sont trop âgés? Combien parce qu'ils sont trop jeunes? Combien par absence de sympathie? Combien par maladie, retraite anticipée, relations illégitimes? Les observations constatent qu'il y a mariage, contrat légitime, sans examiner s'il y a union intime, si l'une des fonctions les plus

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importantes s'exerce moralement : en un mot, si les besoins du corps et du cœur sont satisfaits.'

It is not likely, however, that this condition would excite a neurotic paroxysm, except in persons who from natural temperament or other causes have been already predisposed to such symptoms; and under most circumstances all uterine ailments, and even the irritation consequent on a partially-consummated marriage, would act as predisposing to the malady. The exciting cause is in a vast majority of cases a mental one. Anything sudden, either fright or grief or joy, a harsh word or look, a worry to the temper, an evil dream, may all produce neurotic symptoms under certain conditions, and still more surely may strong appeals to the imagination do so, especially from the pulpit, where the picture enforced upon the imagination, either of holy awe or of mortal terror, is intensified by the authoritative sacredness of the surrounding circumstances. But of all exciting causes imitation ranks highest. To persons even slightly predisposed to this affection, the presence of one suffering from any of the physical phenomena will act as a sure means of prostrating them into the same or similar conditions.

So many neuroses that some would call hysterical seem to depend directly or indirectly on irritation of the generative organs, or of the nervous centres of these organs, that misunderstanding may easily arise on this point. It is well to bear in mind the possibility, even the frequent probability of such a connection. It is better still to avoid, if possible, all treatment of

the organs in question; and taking all forms together, the causation will be found to lie, in much the larger number of cases, in parts of the body wholly independent of sexual feeling, in conditions of hematosis, in irritability of cells of the cerebral convolutions, or of the nuclei of particular nerves, still more in states of the circulation of the nervous centres under vaso-motor influence, and in irritability of the sympathetic symptoms. The symptoms are multiform, and seldom, if ever, co-exist in the same person. They are partly physical, partly psychical; and of these latter many phenomena depend upon one of the chief characteristics of neurotic persons, an exalted reflex function.

Among psychical symptoms is found general nervousness, the patient being easily startled and taking alarm on very slight occasion. She is easily excited to laughter or tears, and in the latter case the weeping will be excessive and uncontrollable. Slight causes will provoke discontent, pettishness, and these feelings will find vent in bursts of convulsive sobbing. The temper is exceedingly infirm. Self-importance is a marked feature. The mental condition may result in some form of eroto-mania, in catalepsy or in melancholia, and this is often accompanied by dysphagia, or rather by a mental determination not to eat. Such patients sometimes offer the greatest resistance to the stomach-pump, but generally they seem to like it, as being a process which gives a great deal of trouble to other people, and exalts the patient for the time into the important

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position of being attended to. This determination not to eat does not seem to be connected with a desire to starve themselves. It is prompted by a mental condition similar to that which suggests the impossibility of micturition and the consequent use of the catheter.

In the melancholia of the neurotic patient she will do nothing for herself, will take no exercise, make no movement except on compulsion, will be dressed and placed on a sofa, and there remain until moved again, apparently taking no notice of anyone. Such a state is very little removed from catalepsy, and indeed may pass into it, although the cataleptic fit more usually occurs without prodromata. In other cases the mental phenomena will be mainly those of hypochondriasis, and the patient will magnify various bodily sensations into symptoms of the most terrible diseases. Such ideas generally amount to real delusions, and only differ from the delusions of mania in that they are ever changing. Perhaps one of the most prominent mental traits in patients of this kind is their weakness of self-control, combined with a marked obstinacy in opposing themselves to the views or suggestions of others. The physical symptoms are mainly connected with the nervous system, not wholly so. Convulsion, contraction, paralysis, various abnormalities of sensation are some of them. Besides these, disturbance of digestion, hæmorrhages, dilated pupils, morbid condition of the uterine functions, find a place.

Rapid twitchings and oscillations of the eyelids

are common, and helpful in diagnosis, when the patient is first seen in a paroxysm of convulsion. This symptom never occurs in true epilepsy, and when it exists the patient is probably always conscious. This clonic convulsion of the eyelids is so rapid that it may occur nearly 200 times in a minute. It is a symptom less under the control of the patient than almost any other, and depends on an alternating spasm of the levator palpebræ and the orbicularis. The breathing of such patients is generally abnormal. They seldom seem able to take long deep breaths, and never adequately fill the lungs in ordinary respiration. But apart from this a peculiar breathing is met with, quick, spasmodic, irregular, accompanied by sobbing or choking. This mode of breathing is exactly like the panting of a person who has been taking hurried exercise, and is a form of dyspnœa. Occasionally this breathing is accompanied by excessive sighing or even sometimes by yawning. After a paroxysm in which many convulsive phenomena have appeared, this panting-sobbing respiration is often the last symptom to disappear. It depends upon spasm of all the respiratory muscles in turn. At the commencement of such a paroxysm the respiration may be temporarily suspended from a tonic fixed contraction of the respiratory muscles, as in epilepsy and tetanus.

Palpitation is very common in hysteria. The pulse may exceed 120 for days and even weeks together, and without any marked disturbance of the general health. More usually palpitation is

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temporarily excited by some external cause acting on the nervous system. Such a cause will generally be of a nature which would produce no result except in neurotic persons; a slight noise, a sudden word, some speech which offends the touchy temper, the reading an account of an accident, the approach of the medical attendant, the thought of anything likely to excite emotion, either of joy, anger, or grief, all these will induce palpitation, either by exciting the accelerator cardiac nerves, or more usually by causing paresis of the vagus. This palpitation is often reflex, of which the eisodic portion of the arc is generally some nerve of special sense, especially the optic and the auditory, whilst the impression is conveyed to the heart by the vagus. The etiology of palpitation has been discussed in earlier pages. It may suffice to say here that as palpitation may be caused in other disorders either by stimulating the sympathetic cardiac fibres, or by paralysing the vagus, so in this various reflex excitations, and sometimes the fact of abnormality of the circulating fluid itself may induce these cardiac phenomena, acting sometimes on the vagus, at other times on the sympathetic. Thus the breathing cold air, the act of eating food, the presence of food in the stomach or of flatus in the bowels, may each and all cause palpitation as a reflex action, and seem to connect this symptom with other phenomena belonging to the vascular system as an example of intensified reflex function.

More important than palpitation are the symptoms resembling angina pectoris that occur in neurotic

persons. True angina pectoris forms the subject of a special chapter. In neurotic patients many of the nerves are in a state of hyperæsthesia. The anginal symptoms may be mere cardialgia, and not unusually are so. But such patients are not free from the ailments common to others; and if the cardiac pain is accompanied with a peculiarly feeble action of the heart, with a low thready pulse, with blueness of the lips and nose and coldness of the extremities, with all the signs in fact of a depressed circulation, it is not to be passed over lightly. Under such circumstances this symptom may be serious, even with a previous knowledge of the patient, with the co-existence of tetanoid rigidity, of globus, of spasmodic breathing, or of other nervous phenomena.

Proceeding in the consideration of the muscular system, general convulsions are met with, varying both in intensity and in kind, resembling chorea, tetanus, or epilepsy. Daily experience gives sufficient evidence of the neurotic origin of chorea at the period of puberty. The history of many of the frenzies of the dancing mania of the middle ages shows this connection in a more marked degree. In most of these epidemics the chronic movements were more or less rhythmical. In chorea the will is weak, and may even be perverted, though seldom to such an extent as in older neurotic patients. The manner in which the chorea may merge into other neuroses, and the evidence there is that the tumultuous cardiac jactitation in chorea depends on a nervous influence (the heart often becoming tranquil

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under the stethoscope after the lapse of a short time) are additional reasons for believing in the nervous causation of much of the palpitation in neurotic persons. In so-called choreic hysteria, violent jactitation of the limbs, at first usually unilateral, is associated with much contortion of the eyelids and facial muscles. This form of malady is often accompanied by various hallucinations, by great depression of spirits, amounting in some cases to melancholia, by dislike of food, sleeplessness and constipated bowels. Some such patients will dance, turn round on their own axis, roll up and down the bed in strong convulsions. In one case, under the writer's care, a married woman, in whom the catamenial functions were healthy, was frightened by fire. In addition to cries and globus, with various affections of the muscular system, she had hallucinations of sight, had been favoured with three visions in which Christ had appeared to her, was suicidal and suspicious, believing she had enemies about her. If she heard of the illnesses of other people, she immediately felt all their complaints in her own person.

Differing from this form of hysteria, more in the character of the special convulsions than in any other respect, is that form which may be called the tetanic. In many varieties of hysteria tetanic spasm plays a subordinate part; but in this form the affection of the muscular system is almost wholly tetanic, although it may proceed to epileptiform convulsion. There is great difficulty in swallowing,

the spasm of the jaws amounting to absolute trismus. It is surprising how protracted this trismus may be, and how easily it is re-excited by any mental stimulus. As in true tetanus, the locked jaw may relax and allow food to be taken with tolerable ease into the mouth, and yet from spasm of the pharyngeal muscles deglutition will be impossible. In many instances of the dancing mania both emprosthotonos and opisthotonos were frequently met with, but more usually the tetanic condition consists in rigidity of the whole body, and rigidity shared even by the muscles of respiration. In some cases the respiratory muscles act so imperfectly that it is difficult to detect the breathing. Sometimes the rigidity is confined to one limb. It may pass away, or may persist and form a chronic contraction. But whilst in true tetanus it is easy during sleep to excite spasm, especially of the dorsal and abdominal muscles, in this tetanus of neurotics there is complete relaxation during sleep.

The most intensified degree of this tetanus is found in catalepsy. It is at least questionable whether consciousness is ever quite abolished in this disease. Such patients can seldom be roused by any external stimulus, and apparently take no notice of surrounding objects or of events: but this occurs in many cases of epilepsy between the attacks, and of melancholia, and yet these patients are able afterwards to give a very fair account of things that have happened around them.

A further stage of this condition is trance, in

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which there is an entire abstraction of ideas from all surrounding objects. But in catalepsy the consciousness seems very deep down, a long way off and yet not altogether lost. The limbs are not in a state of spastic rigidity, but rather in automatic contraction, a contraction by no means difficult to overcome. Where they are placed, in that position they remain, even though the position may be one which is difficult to retain without fatigue. It is in that tetanic form of hysteria that the mental abstraction which is the characteristic symptom of trance is most common. Laycock speaks with some disbelief of this condition. He says, 'If a young woman remains a long time (days or weeks) in coma, and, on being roused, relates the infinity of dreams she has had, everybody says she has been in a trance. There is no end to the marvellous stories about these trances, and the visions, revelations, and wonders the patient has seen and heard. If a crowd follow after her, she will prophesy, speak in unknown tongues, live without food, and perform all sorts of marvels.' But Laycock was too sceptical on the subject. A sufficient number of cases of trance have been observed by careful physicians to make it certain that this morbid condition is a reality, that motor and sensory phenomena may be in abeyance whilst the power of thought remains. There are several varieties of this condition.

1. A state of sleep or apparent coma in which there is no movement except of breathing, and no consciousness of external events, but in which there is

generally some mental activity, shown by dreams, and there may be an after recollection of these dreams.

2. The same apparently motionless coma, in which, however, the patient has the most acute consciousness of what is going on around her, but has no power of expressing that consciousness by speech or motion. It is under such circumstances that a patient may be buried alive; and an instance is on record of the mistake being discovered by the emotion of a patient inducing a copious perspiration, which revealed the fact that she was alive. The late Archbishop of Paris, who was murdered by the Communists, related his own experience of this kind; how in early life, after the exhaustion of a long illness, he fell into such a trance-like state, and though unable to give any sign of life was perfectly conscious of all the preparations for his interment, and was saved from being so buried alive by a brother monk noticing a faint muscular tremor.

3. Those varied phenomena, wholly morbid, that manifest themselves in hypnotism, sleep-walking, &c.

4. A fourth kind is closely allied to some forms of mania. With total temporary abstraction from all surrounding objects and circumstances, and without any appearance of coma, there occur hallucinations and delusions, the memory of which is so strong, the belief in them so real after the attack is over, that it is not only impossible to reason the patient out of her belief, but her after life is to a great extent influenced by them. Such, doubtless, was the condition in which Joan of Arc received her supposed

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celestial mission; such, possibly, the neurotic disorder which made Charlotte Corday an assassin.

5. One further series of phenomena remains, frequently met with among the insane, a state of torpor somewhat allied to catalepsy, but in which the mind seems an utter blank, though sometimes it is abstracted from everything else to feed upon one idea. Here is no sleep nor coma, but a complete absence of the perception of impressions and an almost complete loss of sensation. Such patients will endure extremes of heat and cold without complaint, and will not flinch under the most painful impressions. There is complete analgesia. They show no sign of hunger or thirst, and will make no voluntary effort for supplying any of the necessities of nature. There is no reason to suppose that such patients retain any memory of what has passed around them, or of the objects, if any, of their own thoughts. Two peculiar instances occurred to the writer almost at the same time, that illustrate forms of trance. The one was a lady approaching middle age, of highly nervous temperament, and liable to attacks accompanied by much screaming and tetanic rigidity. After one of these attacks she fell into a state of physical prostration, and was to all appearance so lifeless that the stethoscope alone revealed that the heart's action was still going on. Her mind, however, was perfectly clear and active, but she was utterly unable to express her feelings either by word or movement. When she recovered she recollected all that had occurred, and was able to repeat everything that had been said around her. In the second case, a woman,

also of middle age, with a phthisical family history, had herself suffered from hæmoptysis. She was a large stout person, though habitually afflicted with anorexia. She was a sick nurse among the poor, and whilst nursing seemed always to enjoy perfect health. When her day's duties were over, and especially during any religious service, she would fall like one insensible, the pulse becoming very weak and the extremities cold. Extreme trismus and rigidity of the trunk ensued, and later the patient began to talk with the utmost volubility on all kinds of subjects, calling the persons nearest her by the names of her relations, throwing her arms about them, apparently under the delusion that she was surrounded by those most nearly connected with her. These phenomena might persist from one to two hours, and then the patient would recover. She denied that she was ever insensible or unconscious of her surroundings; she remembered all that she had said or thought, and seemed to have possessed during the attacks a kind of double mental faculty, a true and a false consciousness, recognising by the one the personal identity of those around her, and by the other clothing them with an identity not their own. Something analogous to this is seen in those dreams in which the dreamer may witness his own death and burial, whilst at the same time conscious of his living identity. Does this condition depend on an unequal distribution of blood in the two hemispheres of the brain?

Closely connected with the tetanus of neurotics, often existing, as in true epilepsy, as the sequence of tetanic rigidity, at other times associated with uni-

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lateral jactitation, more frequently standing alone or only showing its affinity with the other forms of the several non-convulsive phenomena each one has in common, is that variety of the malady characterised by convulsions of an epileptic nature.

These convulsions sometimes resemble true epilepsy very closely, the muscles of the limbs being thrown into clonic spasms, the rapidity of which is far beyond what any voluntary effort could accomplish. A person in this condition will shake not only the bed, but the whole room; and the convulsions will continue without interruption for several hours at a time, and will leave the affected limbs more or less paralysed from exhaustion. More frequently the arms only are affected, and sometimes one limb or part of a limb. The intensity also varies from slight tremulous twitching to violent clonic convulsion, in which the limbs may be badly bruised. The facial muscles are generally tolerably free from this kind of convulsion, except that there is much twitching of the eyelids and often violent sobbing. There is no clonic spasm of the elevators of the nostril, of the upper lip, and of the angle of the mouth, of the risorius, the zygomatic muscles, or of the depressor anguli oris, such as is very common in true epilepsy; whilst although the temporal muscle, the masseters and the pterygoids are constantly affected with tonic spasm, thus producing a form of trismus, it is seldom that in this disease they are thrown into clonic convulsions, such as in true epilepsy produces that grinding of the lower jaw upon the upper, which in some cases

is so violent as to shatter the teeth of the patient A very distinct line of demarcation is drawn between epilepsy and what is called by Charcot and others hysterical epilepsy. As in many other cases, this line is a practical convenience for description, rather than an expression of the actual truth. The distinction drawn is that in epilepsy consciousness is lost, in hysterical epilepsy never completely: neither of these points of distinction is universally correct. Cases have been given in a former chapter of epilepsy without loss of consciousness. Every neurologist is familiar with instances of this. In some epileptics the ordinary fit with complete loss of consciousness alternates with another in which there is no such loss. Opposed to these cases are those numerous instances of hysterical epilepsy in which the tetanic condition, already spoken of, is followed by clonic convulsions, and in which pretty frequently the patient can give no account of anything that has taken place during the attack. The convulsions, too, as in epilepsy, may be followed by headache and sleepiness and gradual loss of memory. The extreme instances that have been observed by Charcot in the wards of the Salpêtrière Hospital, and that have been figured by Bourneville and Paul Richer, go through phases of tetanus, of catalepsy, of trance, of clonic convulsions, of anæsthesia, partial or complete; and certainly in them the loss of consciousness is frequently absolute.

There are infinite grades between the various symptoms of neurotic patients, and they are all

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represented in some instances of hysterical epilepsy, and in all the vasomotor sensitiveness plays an important part in the causation of the phenomena. Some other convulsive symptoms remain to be spoken of—globus, cough, and vomiting. There are several varieties of globus. It may exist with or without the sensation of choking. In the former case, gaseous collections may add to the seeming discomfort, but the essential cause of the symptom is a simultaneous spasm of the larynx and of the œsophagus. Or globus may exist quite distinct from the sense of choking, and then will be described by the patient as a feeling of a lump of flesh in the throat which cannot be swallowed. In this case there exists spasm of the œsophagus without any gas below the point of spasm. A peculiar example of the morbid reflex function is sometimes met with in cough. This is generally the exodic manifestation of an irritation that has been carried to the nervous centre by a nerve from a remote part. It may be set up by ovarian or uterine irritation, by undigested food in the stomach, by acrid substances or flatus in the intestines, and it seldom seems to be induced by any irritation within the lungs or air passages themselves. This cough is always more or less spasmodic in character, and it varies in tone and intensity according to the special portion of the organ of respiration to which the irritation has been reflected.

Another convulsive symptom in such cases is vomiting. This may be voluntary in those cases in which the patient is anxious to excite the sympathy

of others. It is remarkable with what ease vomiting may be excited in such persons. It may also be a natural effort of the stomach to reject some of the extraordinary substances with which the morbid taste of the patient is apt to try it. But besides these, there is a constant habit of vomiting, quite involuntary, and not connected with the contents of the stomach or the condition of its mucous membrane, but depending rather on reflex irritation from a distant organ, as in the vomiting of pregnancy, or in the sickness occasioned by the irritation of tubercle in the lungs. Several instances are on record in which the vomited fluid had all the qualities of urine. In these there seemed to be complete paralysis of the renal function, as no urine was passed in the usual manner, and none found in the bladder.

Many other local convulsions are met with in neurotics. Of these perhaps convulsive sneezing is one of the most peculiar. It will depend on uterine or ovarian irritation, and be as much a purely reflex phenomenon as the cough above mentioned, and more frequently it is due to hyperæsthesia of the nasal mucous membrane, that structure being as sensitive to slight impressions of cold as it is under other circumstances to the pollen of grass or to ipecacuanha. In a lady often under observation, sneezing occurs in long paroxysms, going on for an hour without interruption; and apparently as a consequence of the sneezing, the mucous membrane becomes swollen, congested, and painful. The symptom is best treated by quinine.

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Hiccough, sobbing, laughing, yawning, are convulsive affections which are one and all met with in such patients. Very various contractions of muscles or groups of muscles occur in such patients. They are often temporary, but when chronic may lead to deterioration of the contracted muscles. The contraction may take the form of wry-neck, of lateral curvature, of talipes, of flexions of the leg upon the thigh, the thigh upon the trunk, the body on the pelvis (the person never sitting in the erect position), the forearm on the arm; or it may occur as chronic trismus, in which the contraction is not quite complete, but allows a partial opening of the mouth. In speaking of these conditions, Sir Benjamin Brodie wrote, 'One remarkable feature of these diseases is that they seem to be suspended during sleep. When a patient is affected with spasmodic wry-neck, the muscle which is the seat of the spasm, probably the sterno-cleido-mastoid, becomes relaxed during sleep and remains so while sleep continues, perhaps during the whole night. I do not expect that there are absolutely no exceptions to this rule, but I am much mistaken if the exceptions are not comparatively rare.' In the case of a girl, aged 17, in whom contraction of the left sterno-mastoid, causing wry-neck, had existed for nine months, the neck being quite immovable by any manual force, there was no difficulty under chloroform in overcoming the contraction, and the limb was placed in a similar position on the opposite side. When the effects of chloroform had passed off, the wry-neck was as intense and as

immovable on the right side as it had previously been on the left. It seems impossible that this contraction could have been a mere voluntary action, as it resisted the continued efforts of two people to overcome it. Laycock says of this affection, 'This may be either acute or chronic. The chronic form is analogous to lateral curvature, being dependent upon palsy, usually of the left sterno-mastoid muscle. In acute torticollis the spasm is tetanic, the trapezoid is affected as well as the sterno-mastoid, and even other muscles of the face and neck. The head is drawn to the affected side in the acute form, to the unparalysed in the chronic.' It is more curable when it is spasmodic. The paralytic form, which may be paralytic from the first, is also frequently the consequence of the spasmodic form. Much the same may be said of lateral curvature. In this variety the bones are seldom implicated. After the affection has lasted a long time, the spine may be brought back into its proper condition. It may be voluntarily self-imposed, and at least to some extent under the control of the will. More frequently it depends upon temporary partial paralysis of the muscles supplied by the accessory nerve. It is possible that in some few cases this lateral distortion may be the effect of a tetanic condition, one side only being in a state of partial spasm. The less common position of the body being bent upon the thighs, the head of the patient looking towards the ground, is far more serious, inasmuch as originally it was assumed under the influence of the will, and it is only by overcoming this abnormality

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of the will that the patient can be brought back into the upright position. It is not unusual to see this distortion in asylums, in advanced and difficult cases of melancholia. After a certain term it may be impossible for the spine to resume its proper shape, but a patient may remain very long in this position without permanently altering the relations of the bones. If the mental condition be ameliorated, the spine will probably recover its normal shape and position.

A few cases are on record of extreme flexion of the thigh upon the trunk, on the leg, or the thigh. If such positions have been prolonged for a considerable period, it takes some time after the cure to get the ligaments of the joints into their normal condition. Talipes in the neurotic patient is more frequently paralytic than spasmodic. Trismus is not uncommon, and is frequently associated with difficulty of deglutition. It is usually a temporary condition, and passes off with the other morbid phenomena, the time of its continuance varying from a few minutes to several hours. But chronic trismus is sometimes met with, and must be looked upon as one of the contractions. It is seen not only in women, but in men of a highly nervous temperament. In a case under observation the trismus has persisted for fourteen months, the lower jaw being occasionally opened to the extent of nearly half an inch, and sometimes being altogether closed. The partial character of the symptoms enables the patient to be fed with liquids, but solids are impossible, and he has eaten nothing in a solid form for the whole fourteen months. In

this case there is reason to believe, from the evidence of the wife, that the trismus is not always suspended during sleep. If he is disturbed by dreams it is always present, but it also exists sometimes when he is in a deep sleep. It is probable that these contractions are far more under the control of the will than the convulsions in similar patients. Although in some cases of torticollis, of spinal curvature, of contracted arm and leg, no force that can be brought to bear upon the patient will overcome the contracted muscles, yet in the majority of instances the muscles can be forced into their normal position, and can be prevented from returning to their distorted state, by interesting the thoughts and attracting the attention of the patient.

Closely connected both with convulsion and with contraction is the subject of paralysis of neurotics. It has been already said that some of the most obstinate cases of contraction depend rather on a paralysis of the antagonising muscles than on spasm of the contracted ones. But without contraction, a condition is met with closely resembling paralysis. The patient cannot lift her arm or move her hand. She drags one leg in walking, or she cannot micturate. Aphonia is common, alternating with sudden return of the voice. To quote Brodie again, 'Hysterical paralysis has this peculiarity: it is not that the muscles are incapable of obeying the act of volition, but that the function of volition is not exercised is shown to be true by the sudden recovery so often witnessed in cases of this nature, a recovery so different from the slow gradual improvement that

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occurs in favourable cases of cerebral or spinal paralysis. In some cases there seems to be a perfect inability to exercise the will, whilst at the same time there is an extreme desire to do so: in others the function of volition is distorted, the will retaining its normal, or more than its normal force, but being exercised in abnormal directions. It is unusual that any violent mental impression, any sudden fright, has the power to overcome the paralysis, even whilst the mental agitation induced by the desire to move is very intense.'

Romberg considers this paralysis as reflex.

In seeking for the etiology of this condition, and excluding those cases in which a depraved volition is the cause, and all cases in which from uterine irregularities the paralysis might be supposed to have a reflex origin, these paralytic phenomena must be due to a want of nutrition in some groups of nerve-cells in the cerebro-spinal tract, this want of nutrition being determined either by spasm of vessels due to vaso-motor irritation, by local anæmia from other causes, or by loss of assimilative power in the cells themselves.

Aphonia, depending on paralysis of the laryngeal muscles, is perhaps the most common example of this loss of motor power. It generally occurs suddenly, and often is intimately connected with the emotional condition of the patient, being much more intense if the patient is excited. One lady, frequently under observation, will pay a morning call, and ask the servant in an audible voice if her mistress is at home; but on entering the room she will be seized

with such complete aphonia as to be obliged to write all her words on a slate. In the majority of cases this symptom is transient and often intermittent, but it may persist for several months, or even for more than a year.

In most cases of so-called paralysis of the bladder the will of the patient is largely engaged; and the urine is voluntarily retained in the hope and expectation of catheterism. If the prurient desires of the patient are yielded to, and the catheter passed, it is probable that the vesical symptoms will recur. Still, true renal ischuria is sometimes met with in these cases, apparently from paralysis of the secreting function of the kidneys. The diagnosis of such a condition is easy.

Paralysis of the muscular coat of the intestine, especially of the colon, giving rise to enormous distension of the intestinal tract by gas, to tympanites, and borborygmi, is an almost constant symptom. In two cases under care this condition causes acute mental suffering, the abdominal appearances so closely resembling pregnancy. It is probable that in such cases the mucous membrane secretes gas in tolerable abundance, but most of this distension is due to the collection of fæces, from the inability of the paralysed muscle of the intestine to contract and expel them, these fæcal collections giving out a large amount of carbonic acid with some sulphuretted hydrogen. The gases excreted by the mucous membrane are mainly nitrogen and carbonic acid, with small amounts of hydrogen and carburetted hydrogen.

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This abdominal distension is sometimes mistaken for pregnancy. Such patients may simulate pregnancy, even when such a condition involves a loss of character; and they will endeavour to implicate other persons in the trouble. More frequently however the wish is parent to the thought, especially in young unmarried women in whom the desire for children is strong. They are confirmed in their idea by internal sensations produced by flatulence, and misinterpreted by them as fœtal movements. There are no physical signs of pregnancy. Much more rarely, however, there occurs in such patients an affection of the uterus or ovaries, which has been termed 'simulated pregnancy,' in which the abdomen enlarges gradually, and sickness, sensation of movements in the abdomen, and other signs of pregnancy supervene, the catamenia being suppressed, the mammæ enlarged. This abdominal enlargement may go on gradually for ten months. Accurate diagnosis between this condition and pregnancy in the early months is exceedingly difficult.

Perhaps of all morbid symptoms in neurotics, the most common is some variation of sensation. Hyperæsthesia is more frequently met with than anæsthesia. A certain amount of diminished sensibility to touch occurs however in the extremities of those patients who suffer from great want of circulation and consequent coldness. Moreover, in many of the varieties of trance anæsthesia is a morbid symptom and not this only, but a very peculiar amount of analgesia. In some instances pain may be felt but not complained of, the condition being not

one of analgesia, but a moral determination not to manifest suffering. But in others the pain is not felt, whilst at the same time the patient is perfectly conscious; she knows what is going on around her, although she may give no evidence that she does so, and although no ordinary stimulus is sufficient to rouse her. In such cases the attention is so fixed upon one object, so abstracted from all others, that it is unable to recognise any impression that is not connected with the special object of its attention. Pain is so subjective, and from the same stimulus varies in degree so infinitely in different individuals, that it is always difficult to theorise about it, or to test its amount. But examples are constantly occurring to show how an impression, which in ordinary circumstances would cause pain, will have no effect if the mind is occupied in any engrossing pursuit, or in a state of excitement. In the rush of battle the soldier sometimes scarcely feels he is wounded; in the ardour of debate the orator rises above the infirmities of a diseased and agonised frame; in sudden terror the gouty or rheumatic will ofttimes find themselves enabled to use their swollen and tender joints. An interesting conversation, a fixed attention to study, may subdue temporarily at least the sensations of the worst neuralgia. All these are common instances of the ordinary sensations of pain being masked by mental abstraction. Thus also in neurotics. In those rare instances in which anæsthesia occurs whilst consciousness persists, the mind is so abstracted from surrounding objects, and is so fixed upon one

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point, and that point frequently one of self-concentration, that a stimulus calculated to cause pain has no effect whatever. Something more than this is seen in cases of hysteric epilepsy, such as Charcot has recorded. In his wards such patients often show localised regions of anæsthesia. By the application of metallic substances he claims that this anæsthesia can be removed, but that simultaneously with its removal a similar region on the opposite side of the body becomes anæsthetic. Vulpian has found that galvanisation of these anæsthetic regions restores sensation; and many observers believe that the principle of 'Metallothérapie' is the action of a weak galvanic current. Mr. Jessop records a case which agrees with Charcot's description of hystero-epilepsy, complicated with anæsthesia, analgesia, ischæmia, paralysis, contractures, amaurosis, and lastly chorea. Notwithstanding this, treatment by metalloscopy and ovarian pressure had not the slightest effect on the anæsthesia, fits, or chorea. The anæsthesia and rigidities disappeared somewhat suddenly during a fit. Dr. Franz Müller believes in the anæsthesia, and cures it more readily by means of a magnet than by metal plates. Some observers would fain throw doubts on the facts of the anæsthesia; but putting aside for a moment the accurate observation and the absolute scientific truthfulness of Charcot, the transfer both of anæsthetic and of convulsive phenomena, witnessed in epileptics by Buzzard, under the application of encircling blisters, affords important support to Charcot's facts, and Buzzard's experiments were conducted

long before Charcot's observations in the Salpêtrière. It seems certain that under some peculiarities of circulation in the brain, some of the cells of those regions whose functions is the perception of sensations are not only unstable but are inert, are in fact cut off from all activity; whilst, in Dr. Buzzard's words, 'We have the power, by the application of a local irritant to the skin, of occasioning molecular changes in the cells constituting that nervous centre in the cortex, to which impressions are conveyed by centripetal nerves proceeding from the portion of skin inflamed.'

In ordinary neurotic cases, however, an exactly opposite condition, 'hyperæsthesia,' is met with. This may be shown in various ways. It may be general over the whole body, or localised in one particular region. It may be constant or intermittent. It evidently varies in degree, although the patient speaks of it as amounting to agony. Like many other kinds of pain, but in a more marked degree, the patient ceases to feel it if her mind can be engaged for the time upon some other subject. It may attack one spot on the head, the whole spine, or any one vertebra, the nerves of one side of the face, the heart, the epigastrium, the sciatic nerve, any one of the joints. No nerve, no system of nerves in the body is free from the tendency to this condition of pain. In many patients it is impossible to touch any spot in the whole body, whilst the patient's attention is directed to what you are doing, without exciting a scream or a complaint that the touch causes her extreme pain.

It is difficult to determine which region of the body

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is most subject to this hyperæsthesia. In one case, when the spine was uncovered for examination, the patient screamed at the mere approach of the physician, and evidently no more pain was caused by the absolute touch than by the apprehension of it. This is not very common. More usually a patient will bear a mere touch, but evinces great pain if any of the spinous processes are knocked pretty smartly. Frequently a girl complains more during a special examination of the spine than if she is rudely jostled in a crowd, or struck purposely by the medical attendant whilst her attention is drawn away from the back itself. More common than this general spinal tenderness is increased sensibility to touch over certain vertebræ. Perhaps pre-eminently sensitive are the four or five upper dorsal vertebræ; but the lower dorsal and upper lumbar may be specially affected, and the hyperæsthesia may be associated with some motor paresis of the lower limbs, and occasionally with some distortions of hip from spasm of some muscles and disease of others, rendering diagnosis somewhat difficult unless set at rest by the history, the juvantia, and by other neurotic symptoms, and especially by the progress of the case. It is interesting to see a person who has been thus crippled for many years regain her power of movement and normal sensation on marriage. In one girl under constant observation for five years, always recumbent, and complaining of tenderness on pressure over the lumbar vertebræ and of many other neuroses, the improvement on marriage was very marked, as she not only bore long

railway journeys without injury or fatigue, but ascended Snowdon in the course of her wedding tour. Such instances occur to all practitioners. The region about the fourth or fifth dorsal vertebra is frequently the seat of pain, and not only in hysterical people, but in debilitated women generally. The pain here is present in many forms of dyspepsia, in gastric ulcer, in flatulent collections in the colon, in bronchitis, in cardiac debility from thinning of the walls of the heart, and it is probably a reflex neuralgia. The spinal hyperæsthesiæ, of which mention has been made, take on but rarely the character of acute neuralgia, and will seldom be benefited by treatment applied to one particular organ. By refusing permission to the patient to lie constantly on her back, by cold douches to the spine, by regulated horse exercise and gymnastics, and by moral treatment, a satisfactory result may be looked for. It should always be remembered that so-called irritation of spine is not to be recognised by external pressure. The existence of such a disorder is not denied. There may be some abnormality of spinal cells and vessels, in which the ordinary operations of interchange may be interfered with, and this without any change of structure appreciable to our senses; but it is impossible to believe that any such condition can be shown to exist by pressure on the outside of the vertebræ, inasmuch as this pressure can have no influence on the cord itself as long as the cord and its ligaments are healthy.

In such patients, neuralgia of the face is more obdurate than other forms of neuralgia. The pain

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does not generally persist in the same branch of the fifth nerve, but its position is varied, and in the same attack the seat of pain may be located in the temple, the teeth, the angle of the lower jaw, the superior maxillary bone, and the eye. The pain is always described by the patient as excruciating agony; but two points militate against the idea that the suffering is so intense. 1. That in nerves that have been the seat of very acute pain, reflex action seems more easily set up—*i.e.* the affected nerve fibres seem to transmit much slighter impressions to the nervous centres than a healthy nerve does, and the consequence may be seen in twitches or fibrillary tremors of the muscles, as is so often seen in the orbicularis palpebrarum and the elevator of the ala of the nose and upper lip when the fifth nerve has been much troubled with neuralgia, the reflex effect being transmitted to the seventh. This is not seen in this form of facial neuralgia. The second point is, that after severe facial neuralgia the patient unconsciously wears a look of habitual suffering, the plumpness of the face vanishes, the countenance has a drawn look and appears prematurely old. This also is not seen in neurotic patients. Even after many years of such neuralgia the normal appearance of the face is retained. In one lady who claimed to have suffered from this malady for more than twenty years, the *embonpoint* of the face was extremely good, none of the fat between the muscles having been absorbed, and there was wanting entirely that drawn look of the face which indicates prolonged suffering.

In another case of this facial neuralgia, the patient went to the length of having all her teeth extracted, with the expressed view of cutting short the pain. When she had partly recovered from her neurotic condition, she confessed that she had submitted to this painful process in order to convince those around her of the reality of her sufferings, but that the extraction of all her teeth had done but little towards the removal of the pain, for 'there was hardly any pain to remove.'

Neuralgia of the heart or in its immediate neighbourhood may be either infra-mammary or of the nipples. Dr. Todd used to speak of the infra-mammary variety as a constant concomitant of leucorrhœa, and probably connected with it. Most practitioners will agree in his observation. It is an example of reflex sensation, to be compared to pain in the knee in hip-joint disease, pain in the right shoulder in disease of the liver, pain in the glans penis from vesical calculus. The whole knowledge of reflex pain is yet in its infancy. Some people suffer sharp pains along the whole length of the penis if the umbilicus is irritated; the irritations of a small boil on various parts of the back will often cause pain in some intercostal nerve on the opposite side of the body; renal calculus may induce not only retraction of but acute pain in the testicle of the same side. The symptoms of internal tumours, especially aneurisms, teach the same thing. But numerous as are the isolated facts of this nature, the specific laws of reflex pain have yet to be laid down. Doubtless the

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irritation of each part of the body externally, and still more of each internal viscus, will, under certain conditions, induce pain in some corresponding nerve, not necessarily on the same side of the body, nor by any means directly connected with the seat of irritation. It is, therefore, not difficult to understand that an irritable condition of the glands at the upper portion of the vagina may give rise in a reflex manner to infra-mammary pain. But this explanation does not hold good universally. This infra-mammary pain is especially a symptom in weak anæmic women in whom the muscles are ill-developed. In these there is no spot more likely to be subject to strain and less able to resist it than the region just beneath the heart, over the attachment of the most important of the abdominal muscles, and where the skin has so little between it and the bone, and is therefore less able than elsewhere to bear being pulled upon.

Tenderness round the nipples is sometimes superadded to general tenderness over the whole body; but it may exist alone, and become a point of difficulty in the diagnosis of simulated pregnancy. The glands surrounding the nipples are occasionally enlarged, but there is no areola, and the tenderness differs somewhat in character from the soreness of this portion of the breasts in pregnancy.

Besides these mammary phenomena, there sometimes occurs a painful condition of the breasts, or more often of one breast, attended with partial swelling of the organ, which induces a belief in the patient that

she is suffering from tumour. The condition seems in some cases to depend upon the previous mental condition of the patient. She has seen or heard much of such cases, has some reason to fear impending cancer. There is no doubt that the fixing the mind upon any one organ of the body, and especially on one so largely supplied with vessels as the breast, may lead to abnormal sensations in it, and to differences in its size and condition, referable to some influence produced on the circulation of the part. The disorder, when so induced, is the effect of altered nutrition, the immediate sequence of a perverted influence on the vasomotors.

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Clavus is rather an affection of the scalp than of the brain. The scalp is often exquisitely tender. Clavus is sometimes accompanied by headache, or by hemicrania. In most cases the suffering is partly exaggerated, and will cease altogether if the attention of the patient can be diverted from it. In other cases it is more obstinate and is a real neuralgia of the fifth nerve. Pain in the epigastrium often accompanies chronic vomiting in neurotics. It is sometimes reflex, but frequently depends on indigestion, the consequence of the depraved appetite that may be present, or on flatulent distension depending on gastric atony. Here, too, the pain is probably often exaggerated, but for all that there is frequently some underlying mischief that partially accounts for it.

Besides these varieties of hyperæsthesia and hyperalgesia there are often modifications of sight and

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hearing in neurotic patients. The optic and auditory nerves are occasionally so acute, that such patients see and hear at distances and under circumstances that are quite unusual. On the other hand, they complain of the light hurting their eyes, or of slight sounds giving them torment in their ears; whilst if the attention be distracted the eyes will bear strong light, and the most acute sounds will have no effect upon the brain. Even in the most marked cases of this acute sensibility of the special nerves, there is nothing that can be compared with the suffering of this nature experienced in meningitis.

Dr. Russell Reynolds has written thus on sensation. 'The intensity of a sensation depends upon several conditions.

'1. The intensity or force of the impressing cause.

'2. The amount of attention bestowed upon its recognition.

'3. The degree of change induced in the organism, dependent upon the novelty of the impression or its habitual production.

'4. Constitutional peculiarities.

'5. The perfection or imperfection of the material organs for its reception and conveyance. We must therefore take all these conditions into account when interpreting the sensations of a patient.

'Increased sensibility—hyperæsthesia, the word generally employed to express this condition—has included the very various modifications of sensibility, one being true hyperæsthesia, *i.e.* the increase of

impression-effect, undue acuteness of sensation; the other hyperalgesia (as it has been somewhat inaptly termed), marked by the occurrence of pain upon the production of any sensorial impression. The difference between these two elements of disease is more than one of words; each modification has its own clinical relations, and there is little tendency for them to co-exist. A simple contrast of the patients with meningitis or tumour, who shun the light and bury their heads in the bed-clothes to avoid all sounds, from the increase of pain which such impressions cause, but who neither of them present any real morbid acuteness of sight or hearing, with the man of unsound mind, or the anæmiated and hysteric woman, either of whom can hear and see things hidden from the ordinary sense, but who may at the same time experience no pain from such sensation—this contrast will at once make evident the kind of distinction which is intended.

'By hyperæsthesia, therefore, is intended the simple augmentation of sensorial power, apart from any relation to pain, and except in minute degrees it is by no means a common symptom. It exists more frequently as a general condition, and is therefore probably referable to the central portion of the sensorial apparatus. For example, the muscæ and tinnitus of anæmia (which indicate a hyper-acuteness of sensation) exist in common with general exalted sensibility, and are probably due to the manner in which the organic condition of the body affects the brain. Genuine hyperæsthesia is most commonly

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attendant upon some mental change, or upon general systemic disease.'

Another symptom, almost peculiar to the hysterical among neurotics, is depraved appetite. This may take the form of dislike to all meat. It is common among young women of the upper classes, especially among the weak and chlorotic, and seems to be totally unconnected with the conditions of the menstrual function. Less common, and less susceptible of treatment, is the positive form of this morbid condition. When a girl swallows slate-pencil, cinders, sealing-wax, gravel, she may do so from one of three causes: 1, from a desire for notoriety; 2, from a distinct delusion that represents these hard substances as being of a totally different nature—this is seldom met with except where hysteria has advanced to the very borders of insanity, and resembles the idea under which maniacal patients do precisely the same thing; or 3, from a strange gastric craving, similar in kind but greater in degree than that sinking and irritability of the stomach seen in spirit-drinkers, and only relieved, and that very temporarily, by a repetition of the poisonous dose. One girl under observation suffered from a constant longing to crunch small birds with her teeth, not from any cruel feeling, but to relieve a similar kind of gastric craving. Another variety is met with in the desire for drinking blood, and this in the old days of bleeding occurred pretty often in the wards of hospitals. In some it was a real longing for blood; in others it was only from a desire to vomit it up again, and thus deceive the

medical attendant into believing that the patient was suffering from gastric ulcer.

Bulimia, or an inordinate desire for wholesome food, is an occasional symptom in neurotics, but it is very rare at any time, and is equally met with in old women and in young, and in men as in women.

One other physical symptom is worthy of attention here, *i.e.* hæmorrhage from one of the mucous surfaces. There is scarcely any mucous surface that may not be the seat of this species of hæmorrhage. Differing as to the constitution of the blood, both from purpura and from scurvy, the hæmorrhage of hysteria is mainly owing to a defect in vasomotor influence on the vessels. Hysterical patients are subject to the same hæmorrhages as others, and hæmatemesis and epistaxis may be with them not unfrequently vicarious. But the most common symptom in such cases is that they constantly find a small amount of blood in the mouth. There is no cough, no nausea, no vomiting. Usually no abrasion of the throat or mouth can be found, nor any sponginess of gums. The hæmorrhage is real. The utmost watchfulness fails to discover any source from which blood could have been taken for purposes of deception. There is no evidence in the lungs that the hæmorrhage is pulmonary, and yet the bright colour of the blood militates against the idea that it is gastric. It probably comes from the œsophagus. The vasomotor theory was started years ago by Dr. Handfield Jones. Hæmorrhage to a greater or less amount may result from causes of prostration acting

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through the vasomotor nerves on the small arteries and capillaries. The nerve paresis, causing dilatations of the arteries, flushes the parts that they supply with blood, while the same condition impairing the quality of the capillary wall allows actual blood to escape into the tissue or on a free surface.

Neurotic symptoms may be developed without any connection with the generative organs. Many lesions of the uterus and ovaries occur without any trace of such phenomena. The fact, however, that these symptoms are most apt to occur at the development or at the decline of the menstrual functions, that they are intensified by any morbid condition of the uterus or ovaries, that the reflex phenomena of hysteria may, in their most aggravated form, be controlled by pressure over the ovaries, must inspire the belief that in some cases the generative organs are in fault, and that it is upon a morbid excitability of the nerves of these organs that many of the symptoms, especially the convulsive symptoms, depend. But, even granting so much, the condition of these organs explains nothing as to the real nature of the disease. The facts of hystero-epilepsy and of hysterical mania demand a different kind of lesion. Rosenthal locates it chiefly in the cord. "There is a state of exalted activity of the centres, motor, sensory, and especially those for reflex action. It is to this altered reflex that most of the motor disturbances are to be referred. The varying locality and rapid changes in all the phenomena are to be explained on the ground that

varying areas of the cord are affected at different times, and that in most of these cases the disturbance, whatever it may be, is a purely functional one. The hyperæsthesias and anæsthesias are due, very largely, to changes in the circulation of that portion of the cutaneous surface involved, such changes being under the control of the vasomotor nerves. When these nerves contract the vessels, anæmia and anæsthesia result; when they dilate, we have hyperæmia and hyperæsthesia. Rosenthal has noticed this in his cases, and has found the temperature 3° C. below normal in the anæsthetic region."

But even if Rosenthal is right as to the causation of the sensory phenomena in some cases, his explanation does not hold good in all: nor if the cord were chiefly affected does this view account for the phenomena of epilepsy, trance, catalepsy, mania, hæmorrhage, &c., so often met with in these neurotics. The emotion, the exalted imagination, the perverted will have nothing to do with the spinal cord. It is no mere materialism to agree with Dr. Maudsley's remarks, that we have, as physiologists, to deal with volition as a function of the supreme centres following reflection, varying in quantity and quality as its cause varies; strengthened by education and exercise, enfeebled by disease, decaying with decay of structure, and always needing for its outward expression the educated agency of the subordinate motor centres. We have to deal with will, not as a simple undecomposable faculty, unaffected by bodily relations, but as a result of organic

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changes in the supreme centres, affected as seriously and as certainly by disorder of them as our motor faculties are by disorder of their centres.

A large number of neurotic phenomena, not directly connected with the sympathetic system, depend on an instability of cells in large portions of the brain, that is the immediate consequence of altered blood supply and nutrition. Nor does Rosenthal himself limit the seat of hysteria to the cord. In a later paper he uses an expression which seems to sum up the whole nature of the disorder. 'L'hystérie n'est qu'une faiblesse de résistance congénitale ou acquise des centres vasomoteurs.'

Whether it be granted that the great nervous centres are more specially the seat of this malady than the generative organs, or no, it is certain that no lesions or abnormalities of the latter could produce the physical and psychical phenomena unless these nervous centres were themselves in an abnormal condition. The question whether the uterus and ovaries are the starting-point of neurotic symptoms, or whether it is situated in the brain and cord, is simply the question whether the excitants of the phenomena are centric or eccentric; and more than this, whether in cases of eccentric excitation the excitant acts only through these pelvic nerves. The condition of the nerve centres is the same in all cases, whether that condition is brought about by hereditary tendencies, by the circulation in them of blood unrelieved by the normal menstrual depuration, or anæmic by loss of red globules from hæmorrhage, from depressing

conditions, from deficient hæmatosis, or fatigued or ill-nourished by an abnormal mental training. The relations of hysteria with the scrofulous and tuberculous diatheses is important from this point of view. Whatever the cause, the first effect of this depressed system is an irritability of the vasomotors that leads to extreme variation in the blood supply. This very irritability of the vasomotors may itself induce the instability of the cerebro-spinal cells. The symptoms, whether of contraction, convulsion, or paralysis, point to a decrease rather than an excess of power, and of the psychical phenomena, the abnormal development of the imaginative faculty seems due rather to an interference with the relations between imagination and the more highly intellectual powers, the manifestations of the former being in apparent excess from want of the controlling influences of the latter; whilst in many cases the torpor, the loss of independent will, and the occasional semi-comatose condition of such patients, are proofs that the brain is not exercising its power in a normal manner.

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CHAPTER XVI.

PIGMENTATION.

THE most usual variations in colour in morbid pigmentation are yellow, brown, and black.

The conditions under which pigment occurs in the blood, and eventually in the tissues, are various. It will be matter for consideration whether they own some lesion, transient or persistent, common to all, on which this morbid phenomenon depends.

The unhealthy conditions associated with pigmentation are—(1) diseased states of blood, such as ague, syphilis, malignant disease, chronic rheumatism, various cachectic conditions, as Hodgkin's disease, &c.; (2) certain violent emotions; (3) in a large group of cases reflex irritation from the abdominal and pelvic viscera. It is probable that the inflammatory lesions of the abdominal organs act really reflexly, the stimulus being carried to the solar plexus. Under this group of reflex pigmentations may be classed Addison's disease, not only from the special character of the lesion in the suprarenal capsules, and the peculiar anatomical structure of these organs, but also from the fact that similar pigmentations have been met with when the capsules themselves were healthy.

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Laycock considered that the morbid pigments, as distinguished from masses of altered blood-corpuscles, are carbonaceous excretions, and are often vicarious with the suspension or imperfect elimination of other carbonaceous excretions, as the carbonic and lactic acids, and the pigmentary constituents of the urine and bile, and are consequently associated with morbid states of assimilation as well of elimination (through the skin, lungs, liver, and kidneys).

That the hair is a normal channel of such elimination seems certain; and in grey horses masses of melanotic deposit are frequently found, apparently from want of this mode of normal elimination.

It seems questionable whether violent emotion can materially affect pigmentation when the blood is perfectly healthy.

The melanæmic pigment exists in the blood in granular form, sometimes free, or in cells, or in small hyaline coagula. It is equally distributed in the blood of the heart and of the great vessels. The spleen is especially affected. In the blood the white corpuscles are occasionally increased, but a considerable augmentation of pigment means that there has been a great destruction of red blood globules.

After intermittent fever the spleen may be amyloid, and contain little pigment; and in this case the liver and bony marrow will contain much. In the spleen and the bony marrow the capillaries pass into very wide veins, a circumstance which must act in diminishing the rapidity of the blood current. This point bears materially upon the condition of circula-

tion necessary for the deposit of pigment. The partial nature of some pigmentations may depend on some peculiar relations of the capillaries to the veins, as in the above instance; but for general pigmentation the whole vascular tone must be modified more or less, and for this the influence of nervous centres, and especially of the sympathetic, is all powerful. Melanæmia is a transitory condition, and is quickly replaced by melanosis of spleen, liver, and bony marrow. Mosler thinks the pigment is first taken up by the white corpuscles. The dark colour of the skin is produced by the abundance of pigment in the vessels of the cutis, at least at first. The small vessels are sometimes obstructed by pigment.

The spleen is the organ where many of the constituents of the blood are broken up. In it are formed albuminous collections containing iron, derived probably from the destruction of many corpuscles that held hæmato-globulin. It is the organ in which, under certain circumstances, there is the greatest development of pigment; and here, too, partly owing to the delicacy of the splenic tissue, though mainly to the great tendency here to stasis of blood from the peculiar arrangement of the vessels, capillary extravasation is frequently met with. Whether this breaking up of blood cells is wholly destructive, or whether, as is more probable, it is only a step towards a new evolution of blood, is not quite certain. The latter view is supported by the presence in the spleen not only of collections of pigment, but of a large amount of coloured blood-globules

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the mixture of coloured globules with colourless corpuscles containing nuclei, and the accumulation of the products of change of material. The veins of the spleen are richer in red globules than its arteries. This is especially the case after section of the splenic nerves, whilst irritation of these nerves has no effect on the increase of the coloured corpuscles. Irritation of the splenic nerves, or a certain irritation of the medulla oblongata, contracts the spleen. Tarchoneff noticed on section of all the nerves of the spleen an enlargement of that organ, followed by leukæmia. Jaschowitz divided the sympathetic nerve of the spleen in cats and dogs, with the effect of producing an increased flow of blood to the organ, and a copious deposit of hæmatin pigment in its cells. This experiment is the starting-point of pigmentation.

Other factors are necessary in the deposit of pigment in the skin, in this organ or in that. For all practical purposes the spleen is the chief centre for the formation of pigment; and its formation to a morbid extent—*i.e.* to a degree beyond the requirement of new blood formation—depends on a paretic condition of the splenic nerves that closely imitates the loss of all nervous energy by experimental section. It is not the only organ with this function. Possibly destruction of red globules obtains more or less over the whole body. The liver takes a notable part in it, to obtain the colouring matter of its own secretion; and Dr. Coupland thinks it is the chief seat of this destruction of red blood globules: for not only does the blood passing out of the liver con-

tain comparatively fewer corpuscles than that entering it, but its cells are laden with pigment derived from the blood, which they excrete with but little modification in the bile, of which one pigment, the bilirubin, is identical with hæmatoidin.

The capillaries of the splenic artery pour their blood into a system of large sinuses, whence it passes into the efferent veins. This arrangement of the vessels in the spleen, inducing, as it must, a diminished rapidity of blood current, and so favouring more or less stasis of blood, is a factor which, in the spleen, tends to the facile deposit of pigment. All over the body, wherever pigment is deposited, we meet with a similar stasis.

This being the condition of the spleen after section of its nerves, it remains to see whether disease ever imitates wholly or in part this nerve paresis.

Thirty years ago, Dr. Planer stated his belief that the development of pigment was closely connected with the occurrence of intermittent fever. In many cases of ague-cachexia the blood contains many granules and flakes of pigment. Pigment is found in the blood of the heart; it exists largely in the spleen, and was found by Virchow and Meckel in the cells of that organ; whilst Planer found agglomerations of pigment-granules in a hyaline substance. Granules of pigment were numerous in the liver, sometimes in the vessels, but also outside the vessels between the cells of the liver. In the cerebral substance the vessels were often affected by this pigment change, and the cerebral capillaries might be blocked

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by it. It was found also in the kidney, in the vessels of the cortical substance, and in those of the Malpighian corpuscles; it existed in the kidney also outside the vessels. In variable quantity it could be seen in the pulmonary vessels.

In acute ague these pigmental changes are seldom met with; but in an interesting paper, some seven years ago, Dr. Mackenzie quotes various authorities who have seen depositions of pigment in the chronic form of the disease. Thus Kelsch states that he found pigment in every one of 24 patients suffering from pernicious fever, whose blood he examined for this purpose, and of 47 cases in which he examined the blood he met with negative results in 21, whilst in 26 he found melanæmia present. Of these 26 cases he found pigment in the peripheral vessels in several during life. He says: 'I am led to believe that the pigment is a constant and characteristic product of severe or long-continued endemic malarial affections.' He states that the pigment-granules are taken up by the colourless corpuscles in the same way that cinnabar, artificially injected, is; and that they gradually disappear from the circulation by being deposited in the tissues.

Magnus Huss states that he has almost constantly found melanæmia in cases of malarial cachexia observed at Stockholm.

Frerichs was led, from the investigations he made, to the conclusion that melanæmia was most frequently met with in quotidian or tertian agues, rarely in quartan. Dr. Hammond appears to have found

pigment in the spleen in all the cases he examined. Cohn regards the condition as very common. Hertz says it occurs less frequently in simple acute malarial fever than in the tedious, long-continued or pernicious remittent form, and in chronic infection and malarial cachexia.

Pigment-granules are sometimes found in the urine in chronic ague.

Though the spleen is the chief seat of the formation of pigment, the liver, the lymphatic glands, and the medulla of the bones all share this function; and the pigment may occur in free granules, in pigment-holding cells, or in flakes which Fuhrmann attributes to the coalescence of the nuclei and granules resulting from the destruction of colourless corpuscles.

It goes without saying that these views ignore the theory of Lanzi and others, to the effect that the pigment of ague is composed of granules identical with the pigmental sphero-bacteriæ of Cohn, and take their origin from the pigment-granules found in decaying algæ.

Another condition of blood in which pigmentation is not uncommon is syphilis. Besides the general muddiness of colour that is sometimes present, there are often seen brownish or whitish stains, notably on the neck, in size varying from a threepenny piece to a shilling.

Dr. Drysdale claims to have seen them in the female fourteen times in 41 cases. When white, the stains are whiter than the normal skin, showing an absence

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of the ordinary pigment. The neck may be encircled with them, or the stains may be only on one side. They are like ordinary freckles, except that in ephelis the stains are larger, more markedly coloured, and less often situated in the neck. Other observers deny this syphilitic origin, and attribute them to nerve paresis or to anæmic malnutrition; but Dr. Fox, of New York, thinks the mode of development of the so-called pigmentary syphilide to be this, that the maculo-papules of early secondary syphilis, whether upon the neck or elsewhere, induce primarily a hyperpigmentation; at the periphery of these dark stains an atrophy or degeneration of the pigment-cells ensues. The dark stains dwindle to dark points, which speedily disappear, and circular or oval whitish maculæ, of a larger diameter than that of the original lesions, occupy their site. The portions of skin adjacent to these whitish maculæ become, as in ordinary vitiligo, the seat of complementary pigmentation, and thus present a discoloured appearance.

Besides the affection of the liver that is so common in the subjects of hereditary syphilis, the spleen in the syphilitic newborn child is often more firm and voluminous than in the normal condition. According to Dr. Samuel Gee, the spleen is augmented in size in about one-fourth of the cases of hereditary syphilis, and sometimes it is accompanied by an analogous enlargement of the liver and lymphatic glands. If the child do not die, its spleen diminishes in size in proportion to the progress of its recovery. Thus the size of the organ may furnish an

element of the prognosis of infantile syphilis. In those cases of hereditary syphilis, amongst other lesions of the skin, a form of papular syphilitic erythema is met with, the patches of which, originally of a dull red colour, assume afterwards the coppery tint. These patches have their favourite seat on the abdomen, the lower part of the chest, the neck, and the inner surface of the limbs. The crusts, too, of syphilitic impetigo in young children are surrounded by a copper-coloured areola, and a somewhat similar areola is seen to surround the patches of syphilitic erythema. In acquired syphilis, the changes which have been met with in the spleen are sometimes partial or general splenitis, sometimes gummata, and lastly an hypertrophy from augmentation of the cellular contents or of the pulp. This latter lesion is the most frequent. With reference to it Lancereaux states, 'That since no mechanical impediment to the hepatic circulation existed, and since there was no other cause to explain the existence of this change, we are almost compelled to attribute this modification to the syphilitic diathesis.'

That this hypertrophy of spleen can be induced by a syphilitic condition of blood is more than probable; but, considering how often the glands in the abdomen are affected by syphilitic lesion, it is open to question whether the pressure of such glands on branches of the sympathetic connected with the solar plexus may not cause that paresis of the splenic nerves of which hypertrophy of spleen has been experimentally proved to be the result.

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In New Granada and the north-east part of South America a peculiar pigmentation is found that is supposed to be syphilitic. There are three varieties of 'carate'—blue, white and rose-coloured. The blue is the mildest form. Round or oval spots of blue colour appear on the face, coalesce, and extend to the neck and chest, and the ribs are marked out so that the patient appears like a zebra. It attacks the hand specially, and the end of the tibia, and sometimes the glans penis. The white variety is rare in the male, and in women is generally accompanied by uterine disorder. The rose-coloured variety is the worst, and often follows the white. The very minute red spots gradually enlarge, until the whole region assumes a pale red colour.

To a less degree than in ague and syphilis, the phenomenon of pigmentation obtains in some cases of chronic rheumatism and chronic gout. Here, too, as in the above-named diseases, the sequence is gradual. In none of these morbid states does pigmentation seem to be the consequence of acute toxæmia. In all the growth of the symptom is slow; the nerve-paresis leading first to increase of spleen, then to breaking up of red blood-globules to an abnormal extent, or if not so, to the destruction of blood-globules as in health, without the corresponding formation of new ones. This accounts in some measure for the anæmia and its sequences met with in these diseases and in Morbus Addisonii. Later on the increase of pigment in the spleen, the result of this blood destruction, is manifested both

in the internal organs and in the regions of the skin.

Another cachectic condition, in which occasionally a similar pigmentation is found, is that depending on malignant disease. One form, that of melanotic cancer, is itself highly pigmental, and the glands in the neighbourhood of the lesion may become absolutely black. Last year Dr. Legg recorded a case of melanosis following melanotic sarcoma of the choroid. The pigmentation was most marked in the face and neck, less so on the hands, and was not so obvious on the rest of the body. There was no melanæmia and no pigment in the urine. The darkening was of a uniform character, not in patches. In melanotic sarcoma, however, black molecules do sometimes occur in the vomit and in the urine.

Apparently, even where no portion of the abdominal glands or viscera are affected by malignant disease, some pigmentary conditions may be met with. The cachectic look of a cancerous person is itself a slight general pigmentation, and is independent of the seat of the cancer. But in a case lately observed by the writer, pigmentation was more marked. The patient, an elderly woman, sank from malignant disease affecting a large portion of the left lung. Some months after the development of this lesion, the whole abdominal surface became the seat of numerous small pigmental sarcomata, varying in size from a lentil to a threepenny piece. In another patient, now under observation, where the malaise and general debility lead to the fear of some malig-

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nant disease, she has lately developed a large crop of raised pigmental growths, varying in size from a millet-seed on the chest and upper part of the abdomen to tumours the size of a sixpence on the lower part of the abdominal walls, especially laterally. The colour of these growths is amber on the chest, and darker lower down, until, on the lowest ones at the side of the abdominal walls, the colour is a deep black.

Is not the first stage of such a condition a toxæmia, the effect of which takes the same course as the malarial and syphilitic poison, owning also with them the element of chronicity.

This view may be entertained equally by those who consider cancer as primarily a local affection, from which the rest of the system becomes poisoned, or by those who, with constant observation of hereditary influence and of the frequent return of the disease after operation, even where there has been no affection of glands, look on malignant tumours as only localised phenomena of a general morbid crasis.

In the form of pseudo-leukæmia, Hodgkins' disease, where the spleen is hyperæmic, there is a copious amount of pigment in this organ, and this at times may be deposited in the skin. In a recent case the nipples in a man were surrounded by a coal-black areola.

But in a large proportion of all cases of pigmentation the irritation is a reflex one. The pigmentation of face that occurs as a sequence of abdominal tubercle is not due to the morbid state of blood, but

to the direct irritation of the tuberculous growth or ulceration, as the case may be.

In a case lately under observation, an elderly man was the subject of intestinal obstruction from cancer of the descending colon, just above the sigmoid flexure. The lesion had evidently had a chronic course. The whole of the under surface of both thighs was profusely covered with brown moles, mainly of the same size as ordinary freckles, but in some cases larger. These pigmentations had originated only four months before the death of the patient. The scrotum was the seat of a larger and darker pigmentation. The whole of the anterior portion of the scrotum lying on each side of the penis was almost black. There was no discolouration of the portion on which the penis lay. The patient had not remarked this peculiarity himself, but his wife stated that it had only existed for twelve months.

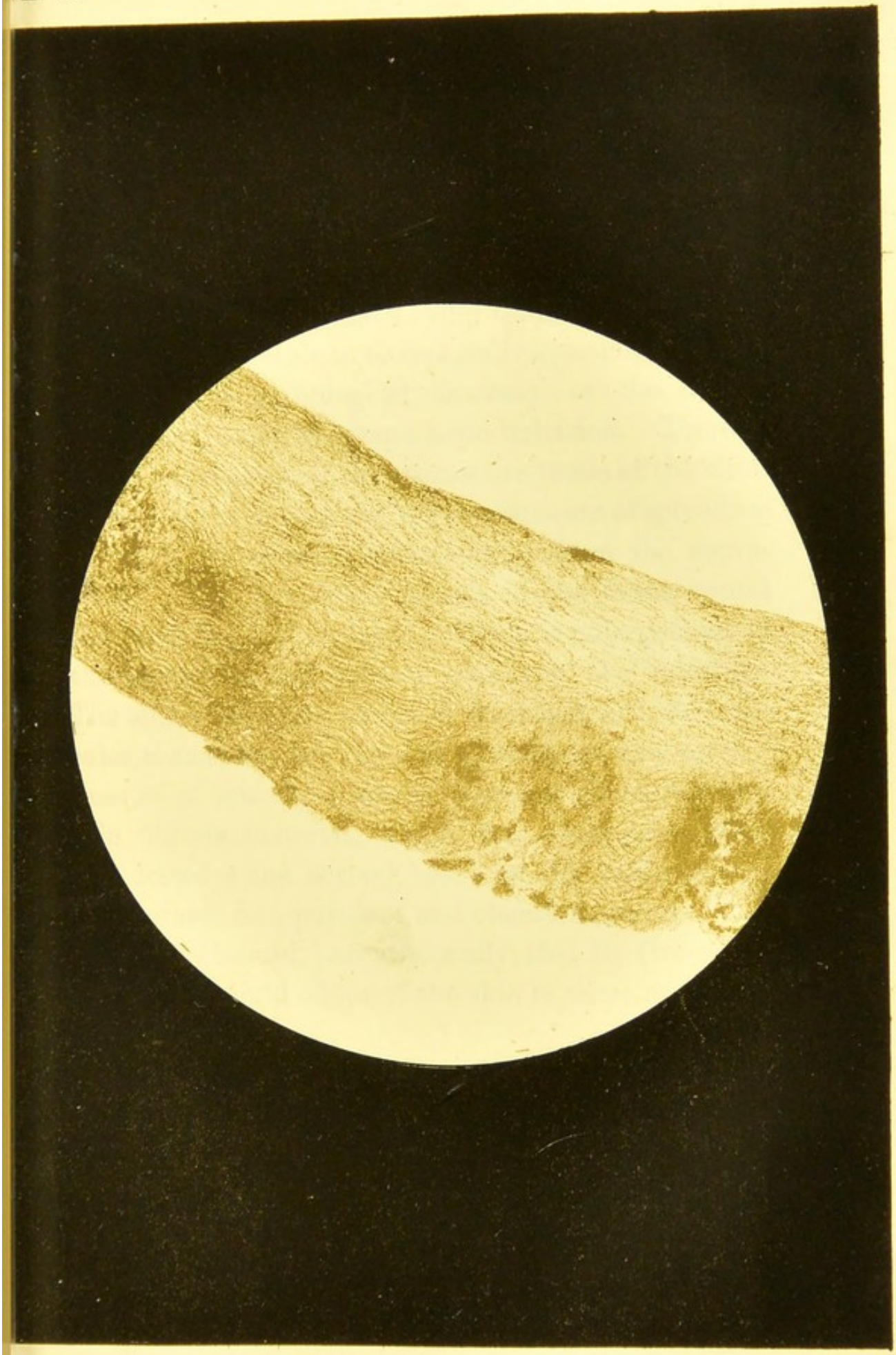
It is one of the conclusions of Dr. Laycock's paper on pigmentation, that structural diseases of the abdominal viscera and peritoneum exercise an influence through the nervous system upon the local deposit of pigment in the skin.

It is as yet an open question whether the pigmentation of Addison's disease is due to the dyscrasia of the blood or to the reflex influence of an injured sympathetic. In this disease pigmentation is a late symptom. The early phenomena are lumbo-abdominal pain, digestive troubles, great general feebleness, frequent vomiting, and pallor of skin. So great is

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this initial pallor, that sometimes the disease has been taken for idiopathic anæmia. In fatal cases with acute course, the capsules may be found very large, but no bronzings have occurred. This acuteness, of course, will probably explain the cases of capsular disease without bronzing, so largely reported in the medical journals. The bronzing, when it occurs, elects by preference the parts that lie uncovered. Sometimes the hands, especially the finger-nails, various patches of the neck and face, the skin behind the ear, portions of the bust, and later on patches on the trunk anteriorly and posteriorly; in very marked cases the greater part of the body will be affected, and the colour may finally become almost black.

Does the pathology of the disease differ from that of pigmentation in general? Brown-Séquard formed a peculiar theory of the accumulation of pigment in Addison's disease on experiments in which he removed the capsules. All the animals died, and their blood was found charged with pigment. He thought that the capsules had as their function that of destroying a substance destined to be transformed into pigment. This has been disproved. Schmidt and Jaccoud regard the affection as a disease of the whole of the abdominal sympathetic. The supra-renals often produce the phenomena, because they are so rich in nerve-elements. The irritation may be transmitted to the cord, or may stop short at the solar plexus, and be reflected on the abdominal plexuses, especially the splenic; or it may be carried up to the thoracic and cervical ganglia. The pecu-



PIGMENTATION OF THE MUCOUS MEMBRANE
OF THE LOWER LIP IN ADDISON'S DISEASE.

liar bronzing has, for instance, been seen by the writer accompanying exophthalmic goître.

Kölliker considers the cortical part of the capsule as constituting a vascular blood-gland, and the medullary as a nervous apparatus depending on the abdominal sympathetic; and Leydig thinks the medullary portion of a capsule to be especially a ganglionic nerve centre. The pathological anatomy of the disease points markedly to sympathetic irritation. There is found hyperplasia of the connective tissue of the semilunar ganglia and nerves, the consequence of spreading to them of chronic inflammation from the suprarenals. The lymphatic glands in the tissue round the capsules are enlarged. There is enlargement of the agminated and solitary glands of the intestine. The spleen is often enlarged, dark and soft, the capsules themselves are the seat of inflammatory exudation of a low type, which becomes converted into firm fibrous material, which first encroaches upon and destroys the normal tissue of the capsules, and degenerates into purulent and cheesy material.

It may be said, parenthetically, that Dr. Greenhow speaks of a fœtid odour of the skin in these cases, like that of the negro.

The nerves at some distance from the inflamed tissue are often involved.

Some cases are on record which prove that irritation of the abdominal sympathetic may excite the phenomena of Addison's disease without the intervention of the suprarenals. Thus, in Dr. Paget's case of lymph-adenosis, there was brown pigmentation of the

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skin. The semilunar ganglia and solar plexus were involved in a closely aggregated mass of enlarged lymphatic glands.

Eulenberg and Guttman have collected twenty cases, with more or less lesion of the abdominal ganglia.

Riegel considers the pathological order to be—1. Lesion of sympathetic nervous system. 2. Inflammatory processes in the connective tissue, &c. 3. From products of the inflammation ensues a paralytic condition of vasomotor fibres of the sympathetic, and consequently an imperfect distribution of food. 4. On this are to be saddled all the phenomena of the disease—anæmia, disturbance of nutritive function, bronzed skin, and a secondary affection of the blood.

In Dr. Goodhart's case the semilunar ganglia showed increase in the amount of fibrous and nuclear tissue, with diminution in the number of nervous elements, chronic neuritis of abdominal ganglia. The suprarenals were mere fibrous bands.

Dr. Davy has met with a similar case.

Dr. Coupland thinks that the vomiting and pigmentation of Addison's disease may be compared with those of pregnancy. In one case of Addison's disease, in a man, vomiting, evidently of a nervous kind, exactly resembling that of pregnancy, was a prominent symptom. In this case manifest changes, not only in the capsules but also in the sympathetic nerve and ganglia in the neighbourhood, were found post mortem.

Lubimoff finds various morbid lesions in the sympa-

thetic ganglia, and believes the relation of cause and effect as to sympathetic lesion to be nearly a certainty.

Dr. Semmola considers Addison's disease a disorder of the ganglionic centres, independent of the supra-renal capsules; one case recovered proper colour after employing the constant current between the side of the neck and the epigastrium. Whilst, then, the general consensus marks the abdominal sympathetic as the seat of morbid irritation in Morbus Addisonii, in connection with the fact of enlargement of spleen in the majority of cases, it seems at least highly probable that the formation of pigment is due to the effect on the spleen of the reflexly irritated sympathetic; though, as Dr. Greenhow remarks, so intimate is the relation in the abdomen between the vagus and the sympathetic that it is impossible to discriminate their separate influence; the depressed action of the heart, the small compressible pulse, the feeble gasping respiration, retching and sickness, are all due to reflex irritation of branches of the vagus.

The pigmentations that depend on irritation of the pelvic organs are somewhat varied. The areola round the nipple and the linea alba are the parts most especially liable to this discolouration. The face is affected in very various ways. Most frequently the forehead shows patches of a yellowish brown more or less symmetrical; or these may appear only below the lower lids, or above the eyebrows, or less frequently on the lips or cheeks. The discolouration may, however, be more universal over the face.

Dr. Swayne published a peculiar case in the

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'Obstetrical Transactions.' The subject was a blonde of rather florid complexion, with brown hair and blue eyes. At the time of her confinement there was a peculiar appearance of the skin of both forearms and hands. There was a very general discolouration of the skin of the forearms, more marked on the dorsal than on the palmar aspect. On the dorsal aspect it occupied all the surface of the arms, and existed in patches on the hands, the knuckles, and all the fingers. The skin in these spots was of a rich yellowish-brown colour, or as dark as the skin of a mulatto. The skin had been similarly affected in each preceding pregnancy, and the dark colour first appeared about the end of the third month, and increased pari passu with the development of the areola, until it attained its acme at the time of labour. After delivery it soon began to diminish in intensity, and in about three months had entirely disappeared. Her mother had two children, and in each of her pregnancies both the arms and the neck were spotted in a similar way; and, being a very fair woman, the discolouration was still more evident than in the daughter.

It is tolerably certain that these various pigmentations depend primarily on changes in the female sexual organs. They never make their appearance before the time of puberty; they disappear after the climacteric period; they are found either connected with menstruation or with pregnancy, and that, too, most frequently in persons in whom the sexual organs are more or less in a pathological condition, even if these regions are not the seat of coarse morbid changes.

They own a similar series of conditions—cachexia consequent on the pregnant state, menstruation, internal tumour, and the like; uterine irritation; the transmission of this uterine irritation to the solar plexus; the consequent formation of an abnormal amount of pigment from paresis of the splenic nerves; and lastly, the further transmission of irritation to some of the vasomotor nerves, determining in various positions the vascular congestion and stasis necessary to the deposit of the pigment.

Dr. Murphy has recorded a case of a lady, with light hair and grey eyes, in whom patches of dark brown pigment appeared, extending from the roots of the hair, across the forehead, and down the cheeks. The entire surface was of a dusky olive brown, like the bronzing in Addison's disease. All the parts of the body examined presented the same general appearance, mottled with dark patches in places, and then fading off at irregular intervals. Dr. Murphy thought that the pigmentation depended on irritation of the sympathetic nervous system, resulting in intestinal catarrh. At the eighth month parturition occurred. The mother convalesced fairly; but her skin, which before pregnancy was fair, still presented traces of pigmentation, and the dark splotches were plainly discernible.

Dr. Sturge has recorded a case of a lady in whom, after a severe illness following her confinement, muscular atrophy of the legs appeared, with a change of colour of the skin of the legs, whilst the hair changed from light auburn to an almost black colour.

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Dr. McLane mentions a case in which, during pregnancy, there was a general deposit of pigment all over the body in patches from one inch to six inches square, the largest being on the neck, the back, and the thighs. The patient was a blonde, with fair hair and blue eyes, and the total change of colour, making her resemble a negress, was the more remarkable.

In his interesting American address, Dr. Barnes mentions several instances. In a case of multilocular dropsy of the ovary of some years' standing, in a woman 38 years old, there was well-marked melasma of the face and abdomen.

Le Cat refers to a case in which the left leg became black during each pregnancy. The mammæ of the Samoyede women are black; and Dr. Latham, who notices the fact, thinks it may be due to a peculiar mode of sexual excitation.

Following the same law, and occurring under the same reflex conditions, are the instances of stearrhœa nigricans recorded by Dr. Neligan, Dr. Mericourt, Mr. Teevan, Mr. Yonge of Plymouth, and others, occurring most frequently round the eyelids. Conradi and Fontanelli describe a blue and black pigmentation of this kind.

Perhaps it would hardly be scientific to look for an emotional centre in the brain. It may be nearer the truth to consider that emotion is the outcome of the action of one layer of cells in the cortex uncontrolled by the other series of cells; and if so, emotion may own as its anatomical seat some portion

of the whole of the cortical structure of the brain. Be this as it may, emotion is seen to paralyse the vasomotors of various parts of the body, to dilate the pupil, to materially interfere with the action of the heart, to influence perspiration, the amount of urine, the catamenial function, the sexual feeling. It is difficult, therefore, not to look at emotional pigmentation as especially a sympathetic disorder.

It is not uncommon to meet with cases in both sexes of partial pigmentation of the face, due to anxiety. One such case under the observation of the writer was so marked as to give rise to fear of Addison's disease. The pigmentation passes away when the anxiety is removed.

In the well-known case quoted by Laycock, a woman during the French Revolution incurred the anger of the Parisian mob, and with difficulty escaped being hanged in the streets. Her terror caused a gradual black discolouration of the whole body, and this remained with her until her death, thirty-five years afterwards. The tint was deeper on the neck and shoulders than on the face; on the face and chest the tint was the same; it was less deep on the abdomen and legs; the joints of the fingers were blacker than other parts; the soles, palms, and folds of the skin in the inguinal region paler. In this case it is observable that the change was gradual.

Laycock's eleventh case was a so-called hysterical woman, who, under pressure of grief, showed melasma of the forehead, eyelids and face, with hyperæsthesia of the affected surfaces. This condition

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recurred during successive pregnancies; and it is therefore questionable how far this is a reflex case, or a purely emotional one.

There remain two classes of cases of dark pigmentation not often met with. One, that of black tongue, has been described by Mr. G. Stokes last year in the 'British Medical Journal.' In his case the scraping from the tongue showed greatly hypertrophied epithelial fringes from the fungiform papillæ and a diffuse staining of the cells. No pigment-granules could be detected. He mentions four cases reported by M. de St. Germain, in which the middle of the tongue was intensely black:

1. A girl, aged 13, with increasing emaciation and paraplegia.

2. A girl, aged 11, convalescent from enteric fever.

3. An asthmatic old lady, aged 70, whose health was not otherwise impaired.

4. An old man in fair health.

The other class of case is seen as xerodema pigmentosum, to three examples of which Dr. Radcliffe Crocker has called the attention of the profession. The disease had been also previously described by Kaposi. From the point of view of this paper, the pigmentation is an interesting feature. In the first stage red blotches or spots appeared, which faded, but left lentiginous pigment spots; or the freckles might be the first noticed, and tended to increase in number, size, and colour. In one of Dr. Crocker's cases papillomatous growths had developed; in one

of Kaposi's, epithelioma; and with reference to this Kaposi remarks that it is well known that epithelioma very often becomes developed from a pigmental wart, and it is therefore reasonable to suppose that the disturbance of nutrition, which in xerodema affects, in the first place, the papillary layer, the formation of pigment and of epidermis, was a cause of the epithelioma, the elements of which are related histologically to the structures named.

In the life history of pigmentation it is of the utmost importance to notice the examples of a want of pigment and the cause of this deficiency. The following case has been lately reported: 'A married woman, aged 35, with mydriasis and partial cycloplegia of the left eye, some dilatation of the retinal vessels, with thickening of the coats of the retinal veins and dilatation both of veins and arteries, showed patches of ivory-white morphœa on the corresponding temple, the side of the nose and upper lip, and similar slighter change in the skin of the forehead and front of the scalp, with thinning of the hair. She had recently been confined, and had been the subject of old uterine troubles. A patch of morphœa existed also at the angle of the left scapula. The skin affected by the morphœa had become atrophied. The morphœa was strictly confined to territories supplied by the first and second divisions of the fifth nerve, whilst the eye symptoms pointed to affection of the branches of the third nerve to the interior of the eyeball. The case, therefore, may be

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compared with those cases of herpes of the fifth nerve in which the third or other motor nerves were also affected.'

A case has been recorded by Darwin where the hair of a criminal brought out for execution turned white in the presence of spectators. Many authentic instances have been met with where a similar whitening of the hair has followed periods of terror or of great anxiety.

Dr. Banks speaks of a young woman in whom half the lashes of one of her eyes became white, which she attributed to the annoyance suffered from the persistent gaze of a wall-eyed admirer, who had white lashes on that eye. It seems highly improbable that the change of colour in the eyebrow of the young woman could have resulted from the contemplation of a similar disfigurement in her admirer. But the reflex influence on pigmentation is beautifully shown in some experiments and observations of Ponchut. A young turbot varies in colour with the colour of the rock or of the sand on which it rests. These changes depend on the greater or less absorption of light by the bottom (whether of sand, rock, &c.), so they must be regarded as true reflex acts, having, Ponchut believes, their centre in the brain and their starting-point in retinal impressions. His experiments prove that it is the great sympathetic which governs the chromatic functions. It forms the route of transmission for the influence going from the brain to the cutaneous chromoblasts; indeed, the retinal impressions transmitted to the corpora quad-

rigemina may be directly reflected on the vasomotor centres.

But, putting aside this class of case, there remains some that evidently depend on loss of nerve power on the part. An observation of Dr. Althaus bears upon this. The sense of smell, he says, depends on pigment in the olfactory cells and the presence of pigment on the integrity of the nerve. The absence, therefore, of pigment, and thereby of the sense of smell, are results of the loss of nerve energy.

In a case lately observed, the pressure of a tumour on vertebræ and ribs, and, ipso facto, the destruction of two intercostal nerves, led to a large patch of vitiligo in the course of the affected nerves.

The nervous influence of this defect of pigmentation is seen also in its being hereditary. In Mr. Godlee's case of vitiligo of the scalp the same condition existed in the mother, two aunts, an uncle, grandfather and great-grandfather, and four first cousins, all on the mother's side.

That morphœa is something more than an absence of pigment is of course evident; but the absence of all normal colour can only be explained by the defect of innervation.

It would scarcely be right to mention xanthelasma plana as an instance of defective pigmentation. It consists in the presence of cell infiltration undergoing fatty degeneration. But it is remarkable that this peculiarity sometimes affects families. In a case now under observation, the patient could speak of the condition affecting not only herself, but a sister,

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two brothers, her mother, a maternal aunt and uncle, and commencing in each instance at the age of nineteen.

It has been seen that in all cases of pigmentation there is the element of chronicity. Under certain circumstances, at any rate, enlargement of spleen, destruction of red globules to an excessive degree in it, an abnormal increase of pigment, have been found post mortem. In a large number of cases the condition of the spleen may pass unnoticed by the patient, and the effects of it, as seen in pigmentation of viscera or of skin, may only appear after the lapse of some time.

The observation also of absence of pigmentation shows that the influence of a sensory nerve is a necessary element.

The other factor requisite for pigmentation is congestion. Not only is this rendered exceedingly easy in the spleen and bony marrow by the relation of the capillary to the venous circulation before alluded to, but it is seen to be the condition preceding pigmentation in all irritants of the skin, blisters, the scratching of scabies, the various chronic irritative eruptions in leprosy, in malignant disease, as well as in reflex pigmentation. And to effect this, the same exciting causes, whether direct or reflex, that partially paralyse the solar plexus and its derivatives can be carried upward, probably through the cord, to influence universally in a paretic sense vascular tone everywhere. Whether its influence is so universal, except in some cases of malignant disease, of Morbus

Addisonii, or in very intense emotional storms, is doubtful.

Why, in cases of partial pigmentation, this paretic state of the vaso-constrictors obtains in one place more than in another is a great difficulty. Is it from any peculiarity in the arrangement of the vessels of the forehead, of the mammæ, of the linea alba?

It is a necessary factor for pigmentation of skin that the cutaneous papilla should be healthy. A pigmental wart shows a certain difficulty in the reflex of blood by the veins; but the papillæ are otherwise normal. When they are atrophied three structures are more or less wanting that would be essential to pigmentation: 1, the sensory nerve-fibril; 2, the full calibre of vessels; 3, the minute arc for reflex action, composed of the vasomotor fibrils and proximate sympathetic ganglion.

It is this atrophy of papilla that is the reason of absence of pigmentation in parts that have undergone pressure. It may be the result of exudation or other sequelæ of the inflammatory process, which cause atrophy of papilla by internal pressure. This probably accounts in part for the loss of pigment in cases of true morphœa. But the pigment may disappear from a part in the very early stages of inflammation, before any exudate has been poured out, in accordance with what Dr. Saviotti, of Turin, observed in a frog. He found that the pigment-cells of a frog collect under irritation round the outside of capillaries and gradually penetrate their walls, and

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those of the small veins partially or wholly, and are swept away by the current.

The pied negro, in whom pigmentless patches exist from birth, probably owes this appearance to a morbid condition of fœtal life. The same phenomenon occurs in white races, but does not strike the eye so vividly. It is an interesting matter for further investigation, to see whether the subjects of leucoderma of various forms lay up collections of pigment in the internal organs, like the masses of melanotic deposit found in grey horses.

The conclusions that may be drawn from the whole subject are:

1. That morbid development of pigment, or unusual position of it, will only be abnormalities of a normal condition.

2. That the exciting cause may be—1, a diseased condition of the blood, acting directly; 2, irritation acting in a reflex manner from a distant organ, and by preference from pelvic and intestinal viscera; 3, emotion.

3. That the irritation, whether direct or reflex, primarily affects the solar plexus, and through it partially paralyses the splenic plexus.

4. That the effect of this is, first, enlargement of the spleen, which may or may not be remarked; and secondly, an increase in the formation of pigment.

5. That, except in some instances of intense emotional storm, chronicity is an invariable element in pigmentation.

6. That, for the abnormal deposit of pigment in the skin, three factors are necessary: 1, a well-developed papilla; 2, the healthy influence of the sensory nerve; 3, a dilatation of local vessel.

7. That the latter factor is generally induced by a diminution of vascular tone, caused by paresis of the vaso-constrictors.

8. That this paresis, like the effect on the splenic nerves, can be effected by the direct action of morbid blood, by reflex irritation, and by emotion.

9. That the opposite condition, viz. one of absence or deficiency of pigmentation, is induced by loss of innervation of the sensory nerve of the part and atrophy of the papilla, however caused.

10. That when loss of pigment seems to depend on emotional causes, it does so by their acting as paralysers of the cerebro-spinal nerves.

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CHAPTER XVII.

DIABETES INSIPIDUS. NEPHRALGIA.

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THE chief symptom of this condition is an excessive flow of water, a flow so great that it is not uncommon to find fifteen pints passed daily, and in some cases very much more, amounting even to forty pints. The specific gravity is very low, 1008, 1006, even 1003; but the amount of solids excreted in the twenty-four hours at least equals that passed in health, and somewhat exceeds it. It is a noticeable point that thirst follows the excess of urine passed, the amount of urine not depending on the taking of a large amount of fluid. The quantity of fluid is taken by the mouth to restore the loss of water by the kidneys, and any increase of the fluid thus taken only shows itself in the urine slowly, but the effect is rather persistent. There is frequently a diminution of other secretions as a result of the urinary excess.

The rationale of the polyuria is vasomotor paresis, affecting the renal vessels. It resembles the condition (mutatis mutandis) that is seen in diabetes mellitus. In the latter disease the vasomotor paresis is of the hepatic vessels, but the modus operandi is the same. The vasomotor centre in the medulla oblongata is

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the centre of a reflex arc. The eisodic nerve, that which carries the irritation, is generally the vagus; the exodic channels are the vasomotor nerves that run to the kidney. The irritation need not be of the vagus. As in diabetes mellitus, lesions of various portions of the encephalon can affect the inhibitory action of the vasomotor centre in the medulla. The effect of cold on the surface of the body may lead to this reflex paresis through the medium of the peripheral nerves. The nerves of the kidney leave the medulla oblongata by the spinal cord, and the spinal cord by the branches to the upper dorsal ganglia, and so, through the splanchnic, reach the renal plexus. Dilatation of the renal vessels must be either from affection of the centre in the medulla oblongata, or from implication of the renal plexus itself. Sections of the nerves in any other part of their course tend to diminish the amount of blood in the kidney, because no such section could be made, either of cord or of splanchnic, without involving the vasomotors of other abdominal viscera, and so filling their vessels with excess of blood, and thus keeping back any excess of blood from the kidney itself.

Dr. Flatten has recorded the case of a man, aged 22, who had sustained a severe injury to the left side of the neck and the occipital region, with temporary loss of consciousness, variable diplopia, and the impairment of hearing on the right side. Almost immediately after the accident polydipsia and polyuria set in, and later on boils made their appearance. When seen by Flatten the man was found to

be suffering from complete paralysis of the left external rectus and partial paralysis of the right external rectus. Hearing was lost at the external meatus of the left side, whilst sounds were conducted through the structures of the head. The urine passed amounted to 423 ounces a day. Flatten's diagnosis of the locality of the lesion was that it was situated close under the nucleus of the left sixth nerve, which it destroyed, while it extended across the middle line and affected the nucleus of the right sixth nerve; but the diagnosis did not account for the peculiar disturbance of hearing.

Ebstein has collected ten cases of diabetes insipidus in which cerebral lesions were recognised, and eight in which lesions of the medulla oblongata were seen to be the cause. In several cases Mosler satisfied himself of the existence of organic disease of the nervous system, in one of which a gliosarcoma was found after death in the fourth ventricle, whilst in another there was unilateral atrophy of the medulla oblongata and the pons. In Murrell's case, a child, two years old, became the subject of diabetes insipidus after a fall on the head. Gayet records a case of extreme interest, in which, in the course of diabetes insipidus, paralysis of the right abducens nerve supervened; this implication of the abducens nerve has also been observed in a case of diabetes mellitus reported by Hæmitz, but here the lesion was a fracture of the skull, and the connection of the two lesions is not so remarkable. Dr. Stephen Mackenzie, in a lecture full of the best information on

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this disease, states that the whole excreta from the kidney must be strictly proportionate to the blood brought to the kidneys by the renal arteries and the relative pressure in the renal artery and capillaries. Anything which relaxes the pressure in the renal artery increases the pressure in the renal capillaries, whilst increased pressure in the renal artery, by increasing the pressure on the cardiac side of the artery, diminishes the pressure in the renal capillaries and veins. The renal arteries are governed by nerves, the vasomotor nerves. The renal nerves have two sources; the greater number of the fibres of these nerves originate from the greater splanchnic nerve through the intermediation of the solar plexus, while, according to Claude Bernard, a certain number of the fibres of the renal nerves are furnished by the lesser splanchnic nerve, and are distributed direct to the kidney, following the course of the vessels. In Dr. Dickinson's and Professor Houghton's cases, whilst it would appear that the paralyses of the vaso-constrictor nerves of the kidneys were caused by implications of the splanchnic nerve or solar plexus by abdominal tumour, dilatation of the renal arteries may, of course, have been caused directly by the pressure of the tumour leading to vasomotor paralysis; but such cases are open also to the explanation that the tumour irritates branches of the vagus, and that the vasomotor paresis is the outcome of a reflex act reflected down from the medulla oblongata. In diabetes insipidus the essential feature of the disease consists not in any malassimilation of food, but in paralysis

of the vaso-constrictor nerves of the kidney, by which too much blood is brought to these organs, and the pressure being reduced in the arteries, the capillaries have to bear the pressure of the systemic arterial force, and this greatly exceeding the pressure in the uriniferous tubules, rapid and excessive filtration of water results. In a fatal case recorded by Mr. Cook the splanchnics and the semilunar ganglia were healthy, but the left was somewhat pigmented. It is probable that the divisions made by some authors according to the amount of solids passed in diabetes insipidus are not dependent on varieties of the disease so much as on variations in the amount and quality of food ingested. But Dr. Tessier, of Lyons, has recorded a series of cases closely resembling saccharine diabetes in the increased discharge of urine, the thirst, the neuralgic and rheumatic pains, the wasting and the secondary lung complications, but no trace of sugar could be found in the urine, and the constant phenomenon was a very considerable increase in the quantity of phosphoric acid excreted (15–20 grammes of earthy phosphates in 24 hours). He has given the name 'phosphatic diabetes' to this class of cases. He divides them into four groups: 1. Those in which nervous symptoms are predominant. 2. Those which accompany pulmonary consumption. 3. Those which alternate with, or coexist with saccharine diabetes. 4. Those which run a distinct course, resembling saccharine diabetes, but without sugar.

Dr. Ralfe, of the London Hospital, has recorded

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similar cases, in one of which the polyuria and increased elimination of phosphoric acid were associated with the presence of a small syphilitic gumma at the base of the brain, situated in the middle line under the floor of the third ventricle. This form of the disease is interesting in its relation to the fact that excessive elimination of phosphoric acid has been noticed in acute inflammation of the membranes of the brain, in the acute paroxysms of certain forms of mania, and after injuries to the head. Eckhard found by experiment that, after unilateral injury to the medulla, hydruria did not occur equally from both kidneys, but chiefly from the one opposite to the lesion; and he thought that this was due, not to a general effect upon the circulation, but to an action of the nerves which proceed to the kidneys. He believed that his experiments justified the assumption that the medulla contains a secretory centre for the kidneys, and that nerve fibres proceed from this centre downwards to the dorsal portion of the cord, from whence they make their exit through certain dorsal nerves, and, accompanied by sympathetic fibres, reach the thoracic aorta, and then go on their way to the renal arteries. Heidenhain objects to this view, and believes that there are no specific secretory nerves for the kidneys, but that the changes in the urinary secretion are all owing to changes in the circulation of blood in those glands. If by section of the upper portion of the cord the secretion of urine is arrested, this would, according to him, simply arise from the diminished pressure in the

aorta, which is the result of such an operation. It is a point as difficult of proof as of disproof, but the fact remains that, whether the medulla contains the centre of the secreting nerves of the kidney or no, it certainly is the main origin for the vasomotor of the kidney, and the essence of diabetes insipidus resides in the condition of the vasomotors.

When the influence of emotion in causing paresis of the vasomotors is considered, as in blushing, &c., it is not difficult to understand how temporary polyuria may be the result of emotion.

It is this view of the origin of diabetes insipidus in the medulla oblongata, either by direct or by reflex irritation of the vasomotor centre, that has suggested the use of galvanism to some observers. Thus Dr. Althaus considered that the localised application of the constant voltaic current to the medulla was primâ facie rational. He applied the current of fifteen cells of Becker-Muirhead's battery to the occiput, taking care that the medulla received alternately the influence of the anode and cathode, and regulating the finer degrees of voltaic force by means of the rheostat. The success was complete.

The vasomotor origin of this disease is rendered more probable also by the success obtained by the use of ergot. In many hands, the writer's among others, this drug has been found successful. Mr. Hammond Williams has recorded two cases of diabetes insipidus thus treated. In one case two drachms of the fluid extract of ergot were given three times a day, together with digitalis; under this

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treatment, in three weeks the quantity of urine excreted fell from 280 to 140 ounces per day, and in another month to 80 ounces. In the second case the daily quantity of urine was reduced from 300 ounces to 70 ounces in twelve weeks. In a case of Dr. Cole's, of Bath, of polyuria, associated with some species of blood-poisoning, ergot did no good; recovery supervened upon the use of valerianate of zinc. There seems to be much truth in Dr. Cole's remark, that it is quite possible that ergot acts on the disease by its general tonic influence on the arterioles, and yet that cases do occur in which a stimulating effect of a deeper and more powerful character is needed to bring back the nerve-centres to the full capacity of their regulating and controlling power. The zinc seemed to exert such action in this case, and faradisation probably acts in a similar manner.

Dr. Henry Kennedy has been satisfied with the results of nitric acid. From various points of view, and acting sometimes on the general health, sometimes on other secretions, sometimes on the renal arteries themselves, belladonna, bromide of potassium, iron, strychnia, jaborandi, arsenic, and cod-liver oil have also been exhibited, and with satisfactory results. But Dr. Stephen Mackenzie urges the employment of valerian, following the lead in this respect of Professor Trousseau, who gave it in doses of from two and a half to seven drachms in twenty-four hours.

Theoretically there is no reason against the existence of a renal neuralgia—'nephralgia'—especially

if a similar condition of the nerves of the liver, the stomach, and the intestine be recognised. Some observers believe in the idiopathic form of renal neuralgia. As a matter, however, of clinical experience, it is extremely rare to meet with acute renal pain, unless associated with organic lesion of these organs, the presence of stone in the kidney, or, as is most usual, of gritty material in the pelvis or the medullary portion of the kidney. If renal neuralgia exist without some one of these conditions, it is met with in gouty persons, and although there may be no definite uric acid deposit in the water, yet it is impossible to deny the probability of the presence of such deposit in some portion of the kidney itself.

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CHAPTER XVIII.

NEUROSES OF THE EXTREMITIES. SYMMETRICAL GANGRENE OF THE EXTREMITIES.

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THERE are certain vasomotor neuroses of the extremities that have lately been described by various observers; in a less degree they are being constantly met with and recognised as a condition of paresis, if not of paralysis, of the peripheral vasomotors; this may be associated with heart disease, as in the case mentioned by Dr. Semmola at the London International Medical Congress, of paralytic action of the heart due to bulbar injury, with a feeling of oppression, palpitation, sometimes even murmur, with marking of hands and forearms (paralysis of peripheral vasomotors), and showing post-mortem pigmental degeneration of the bulbar nuclei of the vagus and of vasomotor nuclei, and, as a consequence, fatty degeneration of cardiac muscles.

Long ago, Dr. Handfield Jones quoted a case of Graves, of neuralgia of the feet and legs, the disorder not confining itself to the cerebro-spinal nerves, but involving in a high degree the sympathetic nerves also; there were pain, heat, and vascular congestion of the feet and legs, alternating with pallor, cold, and absence of pain.

But the ailment has been fully described in various countries, and with especial accuracy by Dr. Weir Mitchell. He speaks of it as a disorder of the feet and legs generally; there is pain, especially when the foot is hanging down, but also, in many cases, in any position; it is sometimes associated with lesion of the spinal cord; there is flushing of the feet, both venous and arterial, and tenderness; it is generally relieved by the horizontal position, but, occasionally, exercise causes the feet to become cold, producing contraction of vessels; pain frequently precedes the vasomotor phenomena. Rest seemed to induce flushing in Sir James Paget's case—at any rate was followed by it. Dr. Mitchell thinks the disease is similar to one termed 'acrodynia' (pain in the extremities), which was epidemic in France in 1829–30.

Other American physicians describe a similar affection of the fingers: they become so cold as to resemble frozen rods, there is tingling and burning on putting them to the fire, the skin is red in patches, sometimes abscesses form at the tips of the fingers. Mitral stenosis has been found associated with it, perhaps as its cause. Dr. McBride speaks of the fingers becoming dead reflexly through the vasomotor system, and Dr. Allan Hamilton looks at the disease as excessive irritation of the local sympathetic vasomotor filaments.

In this ailment there are two chief varieties: one with phenomena of paralysis of blood-vessels, the other with symptoms of contraction, especially of vessels of the extremities.

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In diffuse paralysis of the vasomotors there is an intolerable sensation of pulses, and heat over the body, the face is red, especially the lips and the nasal mucous membrane. It is sometimes accompanied by profuse sweating, and, in some cases, the vasomotor phenomena show themselves exclusively in the extremities.

A partial cramp of vasomotors, confined to the extremities, or to the fingers alone, is seen sometimes in angina pectoris. In washerwomen this cramp is associated with itching and pain. 'The finger dies,' there is pallor of fingers or of the whole hand, diminution or loss of sensation to touch, stiffness of the fingers and local diminution of temperature. It may be cured by faradisation.

More rare is the diffuse cramp of the vasomotors, extending over all the extremities, especially at puberty. In this latter form, Seeligmüller says the cyanosis depending on venous stasis in the capillaries and veins is a consequence of deficient vis a tergo, and does not result from a primary paralysis of the nerves supplying the small veins. Dr. Sturge has recorded a case of vasomotor disturbance of the leg with diminution of reaction both to the constant and induced currents, when the rheophores were applied to the muscles; he also thinks it due to over-excitation of the vasomotor centre. His patient, a man aged 29, began to have attacks of redness and swelling, with a feeling of heat in the right great toe, about four years ago. They came on after he had been standing some time, lasted for two or three hours,

but went off more quickly if the foot were kept elevated; they increased in severity and eventually reached the thigh and buttock. Eighteen months after the first onset, the burning pain felt when the foot was put to the ground obliged the patient to give up work. The left foot and leg have begun to feel hot during the last six months. The attacks are brought on by hanging the leg down, sitting near the fire, wrapping it up warmly, or by much excitement. When first admitted to the Royal Free Hospital he rarely passed twenty-four hours without an attack. On the right foot the surface thermometer (taken by Stewart's surface thermometer) was below 75° (the lowest point in the scale), in the attack it was about 93°, always lower on the dorsum than on the sole.

Dr. S. Mackenzie reports a similar case. The patient, an engine-driver, had suffered with attacks of pain and heat in the foot, the veins of which swell up, and he is quite exhausted after walking only half an hour. The affection had lasted two years, and was probably induced by the different temperature to which his extremities were exposed in comparison with the rest of his body.

As these neuroses of the extremities differ somewhat, both in symptoms and in pathology, it is well to collect together instances of various kinds. Thus, Dr. Cavafy showed two cases before the Clinical Society of London. The first was a young woman, aged 22, who was first seen in March, 1882, when the condition had existed for three years. It began as a reddish mottling of the left shoulder, which

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gradually spread down the arm, and soon afterwards appeared on the right arm, the cheeks, and both thighs, gradually increasing in intensity. At the date of her first visit the skin of both cheeks was mottled with blotches and irregular rings and streaks of a bluish-red colour, most marked on the right side, not prominent, and covered by normal epidermis. Singular dull, bluish-red maculæ, and irregularly confluent blotches and streaks, forming reticulated annular and gyrate figures, occupied the exterior surfaces of both arms and forearms and the backs of the hands, being especially distinct over the left wrist. The front and outer surface of both thighs near the knees was similarly mottled, but in a much less degree. The blotches and streaks were not sharply circumscribed, and disappeared completely on pressure, leaving, in some spots, a delicate fawn-coloured pigmentation. The marking also disappeared from the arms when they were held up, and returned when they were allowed to hang down. The neighbouring skin was normal in all respects and the general health perfectly good. The girl had rheumatic fever a year before the mottling commenced, but the heart was unaffected; the only departure from perfect health was a liability to dead fingers and occasional dyspepsia. The markings were always intensified by cold; they never completely disappeared, and were throughout unaccompanied by pain, numbness, tingling, or any abnormal sensation. The second patient is a healthy young woman, aged 21, who has been five months under

observation, and in whom the affection had existed eighteen months. It began over the ankles and gradually spread to the legs and thighs; twelve months later the arms became affected, and, quite recently, blotching has begun in the waist: the face has remained free. The mottling is almost an exact counterpart of that in the first case, but more extensive, and of a deeper bluish-red colour over both legs and the front part of the thighs, especially near the knees. It is situated on the exterior surface chiefly, but also extends slightly over the flexor sides. The condition above described was only due to venous stasis or passive congestion of the skin, and appears to be an exaggeration of the marking often seen on the skin of children and young persons after exposure to cold; but in the above cases, although cold intensifies the marking, the congestion remains more or less evident at all times. It is probably due to a vasomotor neurosis, but the share taken in its production by arteries and veins is not easy to apportion. The affection appears to be quite harmless, and has not led to any changes except pigmentation, and that only slightly; but the disfigurement, especially where the face is attacked, is considerable. The cases differed from those of vasomotor paresis such as Weir Mitchell describes; the closest alliance was, perhaps, to local asphyxia, which may lead to symmetrical gangrene.

An interesting record that bears on this subject comes from Natal. Dr. McIntyre writes thus: 'On entering on the duties of medical officer in the Avoca

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circle, Natal, some years ago, I was for a long time perplexed by a disease from which some of the coolies suffered. They complained of excessive tenderness and heat or burning in the plantar surface of the feet; they professed not to be able to walk and sometimes even to stand; they are generally weakly and debilitated, but how far this might be the result of the inconvenience from the feet I could not tell. The temperature was normal, and, except that they were weak, there was nothing I could find to confirm their statement regarding the feet. Thinking it might be in some relation to specific disease, I tried iodide of potassium, then I tried tonics, quinine, iron, cod-liver oil, arsenic, local embrocations, &c. No treatment seemed to influence the feet in the least. At last I thought I would try the effect of nerve-stretching. I cut down on the posterior tibial nerve and stretched it. The first few cases were much relieved, but, after I had tried this treatment on four or five with apparent benefit, I tried to pull the nerve in both directions much more forcibly than in the first cases, and, since I began to do so, all the cases so treated have been entirely free from the heat and tenderness in the feet, and the general health has also improved. I am informed it is a disease which is not uncommon in Burmah, but is little known in India; I may say it is peculiar to the coolie, as neither Kaffir nor European has ever suffered from it, to my knowledge, here.'

This communication elicited some further information from an Indian medical officer, Mr. Allen,

then practising at Maritzburg, Natal. He says: 'I have come across many cases of the same affection, and from the frequency of its occurrence, the peculiarity of its nature, the difficulty of forming a diagnosis, and the absence of any cases recorded in any text-book that I possess, I have been induced to pay some little attention to the affection. My first experience of it was in China, amongst the natives there, where it is by no means an uncommon complaint, but only limited, I think, to a certain class of labourers; it was caused in them by standing on marshy ground where rice is cultivated. Gold-diggers are subject to the same complaint, and, I am told, so are persons engaged in the washing of sheep, &c. I am satisfied the affection is brought on by some occupation which necessitates standing in fresh water, or standing on damp or wet ground, while the rest of the body is overheated by a tropical sun. Coolies who arrive here during the summer months—the wet season—are more susceptible to this ailment. The affection is, in most cases, rheumatism of the plantar fascia; in other cases it is due to neuralgia of the plantar nerves and anæmia of the parts, from contraction of the capillaries and blood-vessels; in other cases, again, it is due to a local form of urticaria complicated with syphilis. In this form there is determination of blood to the parts, possibly by a temporary paralysis of the vasomotor nerves. The patient complains of an intense burning, itching feeling, which is only relieved by placing the feet in cold water. I have known a patient who could not

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sleep at night without placing his feet in water. The urticaria which affects the palms of some dyspeptic persons is very much like this affection. As regards treatment, I have found the following to answer successfully: Direct the patient to place his feet in hot water, to which a couple of spoonsful of mustard are added, the bicarbonate of soda being substituted in urticaria; then rub the feet dry with a rough towel, and in the interval anoint the feet repeatedly with warm cod-liver oil; also wrap the feet in cotton wadding and order boots and thick socks to be worn; damp and cold are to be avoided. Abstinence from fish, nuts, curry, and hot condiments, all of which the coolie consumes as his natural food, is necessary. A liberal diet must be supplied. In the rheumatic and neuralgic forms I have, in addition, given phosphorus and tonics. Dry cupping, Corrigan's button, and electricity to the soles of the feet are of great benefit.'

It seems certain, therefore, that this affection is an angio-neurosis, though occurring under different forms, associated sometimes with contraction, sometimes with dilatation of vessels. One result of this symmetrical contraction will be seen in the notice of symmetrical gangrene. But under other circumstances than those above detailed, the two hands, or the two feet, or both hands and feet together, may be involved in a state of vascular dilatation. A case has been recorded of a woman, aged about 35 (neurotic), who was tortured for more than a year by attacks of painful heat in the four limbs,

but especially in the legs and feet; they recurred almost every day, without regular periodicity. The feet and lower part of legs then became congested, the skin a dark red and very warm, the arteries of the feet pulsated forcibly and appeared dilated during the attacks, although in the intervals the pulses in them could scarcely be felt. She experienced a feeling of painful swelling, and walking was impossible; the only relief was derived from plunging the feet and lower part of the legs into cold water.

It is probable that this condition may sometimes have a central origin, but in the majority of cases the course is a reflex one, a symmetrical neurosis of the centripetal nerves of the extremities, causing, by reflex action, a vascular dilatation.

Duchenne, of Boulogne, met with a man, a worker in copper, who used to suffer from attacks of feebleness of the four limbs, in which the hands became red, and hotter and more sensitive than in the normal state. The congestion extended to the forearms and was accompanied by a sensation of tingling. Heat increased the symptoms, whilst they diminished under the influence of cold; rapid and considerable improvement followed the use of faradisation of the upper limbs.

Cahen considered that the congestions that appear in the course of neuralgia (as of the fifth nerve) are due to vasomotor paralysis; the congestion is generally either preceded by or associated with more or less neuralgia; but, in exceptional cases, the

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pains may be absent, so the phenomenon appears as a true congestive neurosis.

Ilio-lumbar neuralgia may thus excite congestion of uterus, and may so cause metrorrhagia, or even leucorrhœa, as results of vasomotor neurosis of the genital organs in the woman. Here, too, the sensory nerve is the part primarily affected, and the vasomotor disturbance secondary.

It may be presumed that the vasomotor phenomena sometimes found in the lower limbs of those affected with alcoholic paralysis are examples of the central origin of the symptoms: the centre need not be a high one. It may be one or more of those small centres near the periphery of the vessels; it may be rather higher up, or even in the lower portion of the spinal cord; but the origin of the symptoms seems to be in the circulation of impure blood in the vasomotor centres of the lower limbs, and there is met with a diffused red or, sometimes, violet colour, and perimalleolar doughiness, which almost constantly exists without the urine furnishing any satisfactory explanation. At other times there are local sweatings of the hands and feet, which appear suddenly and cease in the same manner, or there are alternating paleness and redness.

All these different varieties, however, can be more or less classed together, either as depending upon excitation, direct or reflex, of vasomotor centres, a condition more rare than its opposite, or upon a state of these centres, whether in the cord or, more usually,

in the limbs, that leads to paresis or paralysis of the vaso-constrictors, and that in its turn may be induced, either by direct or by reflex influences, in the vasomotor centre. As in the whole range of sympathetic phenomena, the existing influence is far more usually reflex than direct. Indeed, those instances in which direct influences seem to be brought to bear are not wholly free from suspicion, as where these vasomotor phenomena are associated with syphilis, or with alcoholism, it is not certain that the vessels may not have undergone some degenerative change.

The contractile form of the neurosis referred to in the preceding paragraph may determine symmetrical gangrene of the extremities. This condition may occur in both feet, sometimes also coincidently in both hands, sometimes in both ears, and in the nose. Neurotic people are most subject to the affection, and women especially, particularly during menstruation and from twenty to thirty-five. It may be set up by cold, and seems sometimes to depend on emotion. It is due to a spasm of the vessels themselves. There is no organic alteration in the heart or vessels. There is no arteritis, embolism, or thrombosis. The pallor and bloodlessness of the parts affected, long before the gangrene sets in, are evident proofs that the circulation is primarily affected. The vascular contraction is not quite regular. Red spots appear at first; these become cyanotic on a white ground, the blood appearing to be here and there imprisoned

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and not renewed. The anæmia prevents the nerves feeling external impressions. The early stage may be recovered from. If the contraction persists, reddish phlyctenæ appear, or the tissues shrink up and become black. It is a true tetanus of the vasomotors, and the centre affected may be spinal or cerebro-spinal, as the disease is certainly caused sometimes by moral causes. It is probable, however, in the slighter cases that the centre involved is either one or more of the vasomotor centres in the vessels themselves, or at most one of the ganglia of the sympathetic forming part of the gangliated prevertebral chain. The condition caused is a local asphyxia, or local syncope of the extremities. The skin is pale and cold, more or less insensible. There is a sensation of swelling and a difficulty of movement. The pallor extends beyond the roots of the fingers or toes. There are many variations of colour, from the capillaries or the veins being not quite empty of blood. If the anæmia persists, gangrene is a necessary consequence, and, as above stated, bullæ appear; or a desiccation of skin occurs with retraction of the tissues. The integument becomes like parchment and assumes a black colour. In appearance it closely resembles dry senile gangrene from obstructed arteries. Elimination of the mortified parts may take place. An important fact in this symmetrical gangrene, depending on vascular tetanus, is the presence of the pulse at the root of the segment of the limb where gangrene is produced. Raynaud thinks the obstruction to circulation is due to

excitation of the vasomotors, this excitation often being reflex, and not unfrequently having its starting-point in the genital apparatus. Some authors draw a distinction between local syncope and local asphyxia. In the former, the constriction affects the arteries and the veins; in the latter, the arteries only are involved. But, practically, one is only a variety of the other.

Perhaps the bulbo-spinal centre is sometimes at fault. This may be more probably the case when the hands and feet, the ears and nose, are coincidently affected. But this is not necessary. External cold may induce the first symptoms, making the small ganglia on the vessels themselves the centre of the reflex arc. The parts farthest from the heart suffer, and offer least resistance. This reflex influence may start from within the body.

Raynaud has found in some of these cases constriction of the arteria centralis retinæ. He considers symmetrical gangrene of the extremities as a neurosis, characterised by exaggeration of the excito-motor power of the central parts of the cord presiding over the innervation of the vessels; and he advises descending continuous currents down the spine, with the view of enfeebling the excito-motor power of the cord, and of diminishing thereby the reflex contraction of the vessels, which gives rise to a local asphyxia of the extremities.

There is often intense and radiating pain in the affected region. 'While Weber and a few others believe that the vessels themselves are primarily

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involved, Durosier, Raymond, Vulpian, Fischer, Poincaré and Fontarce think the disease is strictly nervous in its character, and seated in the vasomotor filaments, the contraction of the vessels being due to an irritation of the nerves distributed to their muscular coat. Sometimes this is apparently reflex in its nature, as in McBride's case. At other times the lesion appears to be a central one and involves the vasomotor centres in the cerebro-spinal axis. Vulpian and Poincaré believe it is in the cord, the latter placing it in the so-called vascular column of Clark. Vaillaud mentions a case occurring in a soldier, who had some localised tenderness along the lower part of the cervical spine, associated with a little fever. The symptoms in this case were more on the left than the right side, and later on this side became somewhat paralysed. Raymond, however, puts the centres affected in the cerebellum, and it is quite probable, indeed, that the localities affected are not always the same." An exceedingly instructive case has been communicated by M. Pitres to the Biological Society of Paris—a case of sphacelus from lesion of the peripheral nerves. The patient was a young woman, aged 24 ; she suddenly had unclean habits, her motions were involuntary. Coexistent with this condition, sphacelus appeared in both feet. During the last few days of her life a number of bed-sores appeared on the sacrum and trochanter. The necropsy showed an escape of blood in the cerebral ventricles and the pia mater to the grey substance. The medulla oblongata, the cerebellum and lateral ventricles were

normal, also the heart, aorta and arteries. The cord presented diffused sclerosis, especially in the posterior columns, leaving intact the grey substance. The spinal ganglia were healthy, also the principal nerves. To the naked eye the nerves innervating the gangrenous limbs appeared healthy; there were no neuromata, but an examination under the microscope demonstrated that they presented excessive degeneration, especially near the groove of elimination limiting the eschar. The sheath of the nerves was nearly normal, the lesion was very evident in the nerve bundle, but became less so towards the knee; on the upper part of the legs the sciatic and crural nerves were perfectly normal. The arteries and veins also were healthy. Thus, in the course of a diffused affection of the nervous system, symmetrical sphacelus supervened, independent of any vascular change due to lesion of the peripheral nerves. The reporter adds that this communication of M. Pitres is an additional demonstration to those furnished by M. Déjerine, that peripheral gangrene can exist and the spinal ganglia retain their normal condition; it also confirms the accuracy of his recent interesting researches made with M. Leloir on cutaneous affections coexistent with affections of the central nervous system. These authors have ascertained that peripheral lesions may be coexistent with central lesions, but that one is entirely independent of the other. Nerves present degeneration, and the spinal trophic ganglia remain healthy. In this case the lesion itself of the peripheral nerves may have been the starting-

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point of the irritation, which, carried up to a healthy vasomotor centre, was reflected down to the vaso-constrictors of the arteries of the feet. If cold, impinging on the termination of the peripheral nerves, can set this reflex arc in action, it is not difficult to understand that organic changes in the peripheral nerves themselves can act likewise.

In October 1884, Mr. A. H. Young brought before the Manchester Medical Society a case of symmetrical gangrene, which had been admitted, under his care, to the Salford Royal Hospital. The patient was a man aged 21. The parts which had become gangrenous consisted of almost the whole of the distal phalanges of the fingers on each hand. Beyond a general smooth and glossy condition of the skin, the thumbs were unaffected. The onset of the gangrenous conditions was characterised by intense pain and lividity; before this, however, the digits, which were usually cold and pale, became more markedly so; they were more or less numb, and there was some loss of power. The patient, who presented a strikingly anæmic appearance, had always been weak, and suffered from cold extremities, but in other respects appears to have been fairly healthy. Careful examination of organs and secretions failed to reveal any definite lesions; the blood, however, contained an excess of somewhat small white corpuscles. The family history pointed unmistakably to the probability of this patient being the subject of inherited syphilis; but, beyond slight deafness, he presented no definite indications of such being the case.

In the following case the gangrene might easily have been thought to have depended on emboli, had there not been an autopsy. The case was reported by M. Roques. Symmetrical gangrene affected all four extremities in a patient admitted for cardiac failure, in whom was noted intense pulmonary congestion, enlargement of the liver, albuminuria, and tumultuous cardiac action. Her vision was dim, but the ophthalmoscope showed the fundus to be normal, except that the retinal vessels were rather slender. For some time she had suffered from her fingers becoming pale and as if dead. After being a few days in the hospital her toes became livid and cold, and similar patches appeared on the legs, though the dorsalis pedis arteries pulsated, but perhaps rather feebly. The upper extremities soon presented similar phenomena, and ultimately the little finger of the right hand, and the toe of the same side, became gangrenous. The patient died of heart failure. At the necropsy, a ring of vegetation was found around the aortic valves; the heart was hypertrophied, the mitral valve was healthy; the right lung was hepatised, the liver nutmeggy, the spleen healthy; both kidneys presented an advanced degree of interstitial nephritis; the arteries of the extremities were normal and free from emboli; no infarcts were found anywhere. M. Roques imagines that this symmetrical gangrene is in some obscure way associated with or dependent upon the renal disease. In support of this hypothesis, he alludes to the vasomotor disturbances met with in Bright's disease, and instances œdema of

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one-half of the body, deadness of the fingers, and certain forms of pruritus.

It is not improbable that part of the higher vasomotor ganglia, or else the small ganglia on the arteries themselves, are irritated by the morbid blood in these nephritic cases, and that the constriction so caused cannot be overcome at the most distant arteries by the vis a tergo of the cardiac circulation, in view of the grave difficulties of circulation usually met with in chronic nephritis. In cases, however, in which no necropsy has been made, it would be bold to diagnose this pathological condition in the face of the very frequent alterations in the structure of the arteries themselves. A case recorded by Dr. M. Weiss is that of a member of an extremely neurotic family, of the upper class in society, who had from infancy been the subject of various nervous complaints. Some time after convalescence from a slight attack of typhus she began to experience, without any evident cause, shifting pains in the upper extremities; at the same time there occurred, suddenly coming and going, œdema of the forearm, hands and fingers; then a bloodless condition of one or more fingers, which became pale, waxy and cold, the seat of unpleasant sensations. From time to time also the ends of the fingers would present round spots of various sizes, from that of a pin's head, of a bluish-red colour, presenting a dry parchmentlike condition of the skin. The integument would become necrosed, and desquamating, leave an annular patch of cicatrising surface. The whole process lasted three

months. The terminal phalanges of the little and ring fingers of the left hand were lost, and the volar surface of the right middle finger was diseased in the same manner. Superficial gangrene of the skin over the sacrum also occurred. Then paretic symptoms of the left cervical sympathetic appeared, followed by wasting of the left side of the face.

Aphasia was twice noticed. During the process of mortification the sensibility of the hand, and its susceptibility to heat and electricity, were reduced. The activity of the muscles of the hand was diminished, as was also their faradaic excitability. The nails of all the fingers became furrowed, and exhibited a dark brown discolouration. The vascular system did not appear to be disturbed. Menstruation did not exert any influence upon the course of the affection. The pathogenesis of the case is, by the author, traced to disturbance of the vasomotor system, causing spasm of the minute vessels. By spastic ischæmia of the centres of speech aphasia was occasioned, and by the same condition in the anterior cornua of the spinal cord muscular nutrition would be interfered with, while the cutaneous lesions could be traced to a morbid condition of the posterior column.

This case is the more important, in that it is associated with other vasomotor phenomena, and also that the gangrene of the fingers was preceded by conditions showing vasomotor paresis. Such states are only intensifications of the blueness, pallor and coldness of the extremities in delicate people from the use of a cold bath. In such persons the action

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of the heart is often perfectly good, but the phenomena of the extremities are due to a more or less persistent constriction of the arterioles. Somewhat similar cases have been brought before the Clinical Society of London by Drs. Southey, Barlow, and Mahomed. In one of Dr. Mahomed's cases intermittent hæmatinuria had existed, and crystals of oxalates were found in the urine. A few male patients of his own had suffered from the disease in a more or less chronic form for seven or eight years, the fingers presented a gangrenous appearance, which varied with the weather, but was not improved by treatment. The tips of two or three fingers had been quite lost. In summer-time the hand was quite useful.

In the absence of post-mortem records, it is doubtful whether the rare cases of symmetrical gangrene of the abdomen can be placed under the same category as the above. A peculiarly strange case of this kind was brought before the Vienna Medical Society by Neumann. An anæmic young woman, aged 18, had been the subject of acute circumscribed gangrene on various parts of the body. The first symptom was a sensation of severe burning which lasted from five to ten minutes; the skin became reddened on a circumscribed spot, slightly raised, and the temperature increased. The part then became brown, and afterwards of a dull white colour. Sensation was lost in the periphery first, and after about half an hour in the centre.

A child, aged 5, was admitted into St. Bartholo-

mew's Hospital, under Dr. Southey, on the twenty-first day of illness (scarlet fever), and on the abdomen was noticed a large dark purple triangular patch, graduating off in bruiselike rings of colour at its margin, suffusing the skin of the abdomen. It left the umbilicus free, but was very symmetrical on both sides of the mesial line, extending from the costal arch to a little below the navel, the apex pointing to the pubes. A second ecchymotic patch of smaller size extended a little below the larger one. Three days after admission, the skin of these patches became gangrenous. As recovery in both these instances took place, the causation of the gangrene is doubtful, and it would be manifestly unfair to associate them with the pathology of symmetrical gangrene of the extremities, although such an association is not improbable.

The connection of this subject with frostbite is exceedingly close. In frostbite the effect of cold is primarily in the peripheral sensory nerve, but the almost immediate result is reflex constriction of the artery of the part affected. All circulation and sensation stops, and this so entirely that no reaction can take place in the obstructed part, and it thus becomes a mechanical obstacle to the free passage of blood. In a lesser degree chilblain is an example of the same thing. In chilblain the action of the cold is usually much less intense than in frostbite. The primary action is overlooked, and the results of the reaction constitute the complaint. The cold, instead of stopping entirely the chemical action in the fingers

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and toes, so checks the healthy changes that the blood becomes more or less retarded; congestion ensues, and thus the circulation is more or less completely stopped. This gives rise to increased tension in the vessels leading to the obstruction; the impulse of the heart is increased; it urges the blood more strongly forward to overcome the congestion; increased motion of the blood takes place around, increased oxidation is set up, and altered nutrition occurs in and around the chilblain: thus the healthy action of the part is usually restored, unless the increased oxidation leads to excessive cell formation, and then suppuration occurs.

It would seem to be against experience to connect perforating ulcer of the foot specially with the vasomotor system. Except in so far that no ulceration can take place without some morbid alteration of vessels, post-mortem records do not bear out the view that this lesion is connected with the sympathetic. When this form of ulceration depends on lesion of the nervous system it does so either in connection with neuritis of the peripheral nerves, without any lesion of the central nervous system, or, whilst the peripheral nerves are healthy, in association with locomotor ataxy, or, as in two cases reported by M. Christian in the 'Arch. de Neurologie,' v. 246, in connection with general paralysis. In one of these cases the perforating ulcer was found in one foot, in the other in both. But the causation is quite different from that of symmetrical gangrene of the

extremities: it cannot be considered a vasomotor neurosis of the extremities at all.

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CHAPTER XIX.

MYXŒDEMA. SCLERODERMA.

PATHOLOGY — Myxœdema

WHILST the pathology of a morbid condition like myxœdema is still under investigation, it may appear premature to refer to it, and especially under a heading that would seem to imply that the sympathetic system is involved. Doubtful, and at least unproved as this theory is, some observers are inclined to hold it, whilst others, if they deny that this system of nerves has anything to do with the causation of myxœdema, will allow that some of its phenomena depend upon a morbid state of the vasomotor nerves secondarily induced. In the difference of opinion as to the part played by the ganglionic nerves in myxœdema, it may be allowable to refer to the chief features of this remarkable disease, and to determine what share, if any, in its causation or in its symptoms is taken by the sympathetic.

The condition arises spontaneously, but it can be induced by experiment. The chief factor has only been recognised almost accidentally by the results of some operations on the thyroid body by Professor Kocher, of Berne. He observed that, after complete (not partial) extirpation of this body, great anæmia

resulted, the hair fell out, in young persons there was arrest of growth, in all the arteries were narrowed, there was loss of memory, a general hebetude, a doughlike condition of the face and hands and of much of the body, and œdema without pitting; and that the fœtal tissue mucin was found in abnormally large quantities.

In comparison with the results of extirpation of the thyroid body the spontaneous form of the disease is always associated with loss or great diminution of the thyroid body. The movements are slow and languid, speech slow, deliberate and indistinct, taste and hearing dulled, mental powers dulled even almost to extinction, if life is preserved (but for a long time the patients, though tranquil, are painfully aware of their condition); expression of face immobile; firm œdema of face, especially of the eyelids, which are pendulous, the face round, swollen-looking, waxlike, the nose broad, the lips thick, and often of a purple hue; the skin of the hands and arms waxlike and sometimes scaly, the shape of the hands spadelike. The skin is always cold, the axillary temperature about 95°; no perspiration. The same immobile condition seems to involve the internal organs. The stomach does not digest properly. A condition of atonic dyspepsia is met with. Constipation like that of aged persons is found, and, depending on the same cause, an inactive state of the intestinal muscular coat and a deficiency of the normal intestinal fluids. The heart sounds are distant from feebleness. The urine shows a low specific gravity, probably from a

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decrease of the amount of urea excreted, a fact that has been observed in some cases, and one that would probably be met with from imperfect digestion and almost total want of exercise. In Dr. Ralfe's case it fell to half the normal amount. The mucin is much increased. In a case of Dr. Ord's the mucin was fifty times as much as in health.

This mucin is the ground substance of connective tissue, not the fibrous element. Morochowitz teaches by his researches that all the tissues of the connective type have a common chemical origin in the embryo from collagon (which yields gelatine on boiling) mixed with mucin. In myxœdema there is probably a retrograde metamorphosis.

Dr. Ord has described the post-mortem appearances of a case. The whole connective tissue of the body had been found swollen and jellylike and œdematous with mucin. The swelling seemed sufficient to account for all the symptoms. The heart and arteries were obstructed by it, and hence the feeble blood-current, the deficient aeration of the blood, and the purple of the cheeks and lips. The tongue and palate were swollen with it, the intestines choked by it, the senses dulled, the functions of organs interfered with, and the patients died with all their tissues smothered by their own padding.

The symptoms of such a case of myxœdema are precisely similar with those of a patient in whom extirpation of the thyroid gland has been performed by operation. So far it is clear. The symptoms almost wholly depend on the presence of abnormal amounts

of mucin in the tissues. What, then, is the connection between this substance and the thyroid gland?

Dr. Cresswell Baber, of Brighton, attempts to answer this question by referring to some researches he has made under the direction of Dr. Klein. He has shown that not only is a homogeneous or finely granular viscid material (termed colloid) found in the vesicles of the thyroid gland, but that a substance morphologically identical with this is found in the lymphatics with which this organ is very richly endowed. It is therefore reasonable to conclude that a material is formed in the vesicles which is carried off by the lymphatics of the organ. He has further shown that the colloid material in the vesicles is largely formed by the destruction of red blood-corpuscles which have entered the vesicles, as red blood-corpuscles are frequently found in the vesicles in all stages of disintegration and discolouration—in fact, in such a state as to show beyond question that they had entered during life. He suggests that in myxœdema, if the elimination of colloid material or its constituents from the blood be prevented by loss of the thyroid gland, a transudation of these substances into the connective tissue generally may take place. This presupposes that on its way back to the circulation through the lymphatics, the colloid material becomes changed in character. But these observations of Dr. Baber do not explain why, when the organ that produces mucin is abolished, this substance should exist in abnormal amount in the tissues.

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In a very suggestive record of a case of myxœdema, associated with insanity, Dr. White formulates the following sequence of events, each bearing to that succeeding it the relation of cause to effect:

1. Some neurotic influence in a person predisposed to its agency. 2. Atrophic lesions of the cervical sympathetic ganglia, with paresis of their cardiac branches. 3. Retardation of the heart's action and blood flow, with alteration of the composition of the blood consequent thereon; the formation of mucin, as a retrograde derivation of blood albumen, this mucin being held in solution by the alkaline blood. 4. Transudation of mucin from the vessels, deposition of it in the external vascular tunic and in the connective tissue.

Here, probably, the atrophy of the thyroid gland is too much lost sight of. The experience of no single observer having as yet been large of this disease, it is the more necessary to collect any post-mortem records. Thus, Dr. Henrot found in one case hypertrophy of the trunks of the pneumogastric, the glossopharyngeal, the nerves of the brachial plexus, and especially the ganglia and cord of the great sympathetic. A tumour the size of a hen's egg was found at the base of the brain, flattening the optic chiasma. The main cord of the sympathetic and all its glands and efferent nerves were much hypertrophied, especially in the cervical region. The semilunar ganglia and solar plexus were enlarged. There was enlargement of the pituitary body and of the pineal gland. On these facts he founds a theory

that the disease consists in a return to the embryonic state of the subcutaneous, submucous, and interstitial connective tissue, and the formation of mucin under the influence of the enlarged pituitary and pineal glands which preside over this function.

It would be a very tempting conclusion that the inactivity and hebetude of the mental faculties depend on the pressure of the mucin round the cerebral vessels or on the layers of cerebral cells in the cortex. If this is granted, it must be that the brain becomes thus involved before the integuments, as it has been frequently observed that the nerve phenomena precede in point of time the solid œdema of the skin. The inertia, too, can be thrown off under the influence of anger, and both in speech and in movements can such patients then manifest unusual vivacity.

In a case of Charcot's, such a patient raised seven bushels of potatoes in each hand. On the other hand, when cerebral inertia has existed, if from any cause the swelling of the extremities has improved, the psychical condition has improved pari passu, the sensory perversions have gradually disappeared and all delirious ideas passed away, so that at least some of the nervous phenomena must depend on this pressure.

The constant triad of symptoms—profound anæmia, lesions of the integument, and nervous disturbances—may exercise a mutual relation on each other. Among the latter, motor disturbances are met with, abnormalities of general and of special sensation,

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symptoms of locomotor ataxy, psychical phenomena of every grade, from the slightest to the most severe. Is it fair to assign to the nervous system the causation of the malady? A word will have to be said with reference to the rôle played by the sympathetic in this disease; but for the moment, putting this aside, there seem to be only two ways of looking at the nervous phenomena in myxœdema—one, that some of them may be caused by a morbid condition of the centrospinal centres irrespective of the pressure of mucin; the other, by the interference with the circulation in these centres and with the due action of the nerve-cells by the compression exercised by the mucin. In general terms, the only evidence of the causative effect of the central nervous system lies in the fact that this disease seems to follow anxiety, domestic worry, &c. A nervous origin of the complaint is contended for by M. Verneuil, who has seen it follow injuries; the wound in these cases having some supposed influence on the central nervous system capable of producing the special phenomena of myxœdema. But there is no direct evidence of this connection. M. Blaise considers that the psychical disturbances may be independent of the lesions of the integument, inasmuch as the former are sometimes the first symptoms of the malady; but he thinks it not unreasonable to believe that in certain cases the alteration in the integument may become the origin of delirious ideas. Such a fact is only in analogy with the delusions set up in an insane person by gastric disturbance, and which disappear when

the stomach recovers itself. Thus, a lesion of the integument may set up a false idea in the brain of a myxœdematous patient; but probably this false idea could not be excited except in a brain disposed to receive it, and to transform it in a special and incorrect manner.

The cerebro-spinal system, therefore, being excluded from the causation of the disease, it remains to touch upon the sympathetic.

Kocher believes in a neuritis of the sympathetic. M. Ballet and Sappilli have seen morbid changes in the sympathetic in myxœdema. Dr. G. Stokes believes that the supervention of myxœdema after the operation for total extirpation of the thyroid gland results rather from lesions to the sympathetic nerves with which the gland is freely supplied, than from injury to or absence of the gland itself; that in the operation for extirpation, the sympathetic nerves and the large ganglion-cells in connection with them are wounded, and that, possibly, these cells have to do with the nutrition of the cervical sympathetic ganglia, which hence become atrophic, just as the ganglia in the posterior roots of the spinal nerves perform a similar office. He thinks this supposition is borne out by the following facts: First, though complete atrophy of the gland follows division of the isthmus, myxœdema does not; and certainly neither does myxœdema follow extirpation of one half of the gland, because nutrition of the cervical sympathetic ganglia is still carried on by the nerves and ganglion-cells of the opposite side. A point in favour of the

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influence of the sympathetic is the early presence of increased arterial pressure in myxœdema. Later on the kidneys become implicated, the cortical substance becomes involved by a nuclear proliferation, and albumen appears in the urine. But long before this the sphygmograph registers a high arterial tension; so much so that Dr. Mahomed, in 1881, asked whether these cases were not cases of chronic Bright's disease. 'In many cases of chronic Bright's disease there was no œdema, but in others there were all the symptoms of myxœdema without albuminuria, but with high arterial tension. In one case the subcutaneous tissues were found not to contain mucin. In the published cases of myxœdema, now twenty in number, three were not noted as to urine; in the seventeen others ten had albuminuria, of which four were fatal: they had affected kidneys, hypertrophied heart and thickened arteries, whilst many of the changes of the spinal cord were doubtless those of myxœdema of the cord. Thus, in all the four fatal cases, Bright's disease was present. Albuminuria was absent in many cases of Bright's disease, and, if there were such a thing as local œdema taking place, it would be a good explanation of nervous symptoms developed in myxœdema.' Without accepting these views, the high arterial tension is a vasomotor phenomenon.

Dr. Haddon believes that the disease is probably dependent on changes in the sympathetic nervous system, and that the thyroid is in some relation with the peripheral sympathetic nerve-fibres. Dr. Heriot

has found, as already stated, great hypertrophy of the main cord of the sympathetic, the ganglia, and efferent nerves.

Dr. Hadden would consider that probably, from vaso-constrictor lesion, a state of angiospasm obtains in myxœdema, affecting not the blood-vessels only, but also the lymphatics. Under such circumstances there would be a generalised lymphatic obstruction, or, in other words, an inability on their part to remove the products from the lymphatic spaces within the connective tissue. The result is the development of the solid œdema. Perhaps these accumulated products undergo certain changes resulting in the formation of mucin. He concludes—1. That in the early stages myxœdema is essentially a disease of imperfect nutrition, dependent probably on generalised angiospasm.

2. That the solid œdematous condition of the skin and connective tissue is due to a form of lymphatic obstruction which may also be ascribed to vasomotor influence, and that the accumulated products undergo changes which result in the formation of mucin.

3. That the condition of the thyroid gland is also to be explained on the vasomotor hypothesis.

4. That the more severe mental symptoms, such as insanity occurring in the later stages of myxœdema, are due to alterations in the brain itself.

5. That although myxœdema is a distinct morbid entity, it is probably allied to certain other disorders, such as sporadic cretinism and scleroderma.

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6. That the solid œdema which is universal in myxœdema may be localised in various parts of the body, as well as the tongue and extremities.

7. That the primary and essential lesion probably exists in the peripheral sympathetic system, and perhaps, too, in the supreme centre in the medulla oblongata, this last supposition being based on the occasional occurrence of bulbar symptoms in myxœdema. This theory of angiospasm is not only borne out by the high arterial tension, but by the good effects of jaborandi and of nitro-glycerine as remedies.

The following is the only well-marked case seen by the writer :

Miss D., 52, has been ailing three years. The mind sluggish for one and a half year. Memory fair. Is fidgety, worries herself; has lost confidence in herself. After any loss of blood or sleeplessness she is exceedingly fanciful and suspicious, almost incoherent, but this is for the time only; no fixed delusion. All mental effort is difficult. Sensation good, except in the hands. Locomotion very slow—no ataxy. No muscular atrophy. Sight good. Hearing not quite good, and some hallucination of hearing. She is decidedly deficient in tasting and smelling. Patellar tendon reflex strongly marked on each leg. Temperature always subnormal. No perspiration, but this secretion was always deficient in health. Rather subject to emotional blushing. Tongue enlarged. Solid œdema of fingers and hands and face. Immobile expression of face. Skin waxlike. Lips large and purplish. Neck swollen, so that the

condition of the thyroid is not easily made out, but it is probably atrophied.

Even if Dr. Hadden's vasomotor theory is accepted as explanatory of most of the phenomena, it does not determine the real causation of the disease. Can mental anxiety impress the vasomotor centre in the medulla oblongata so strongly as to cause spasm of all the vessels and lymphatics in the body? Why does this angiospasm induce complete atrophy of the thyroid gland, whilst no other organ of the body is atrophied? What evidence is there of the possibility of the material unabsorbed by the lymphatics being metamorphosed into mucin, either by way of advance or of degeneration? All these are questions that must be determined before the theory can be sustained.

But the vasomotor theory explains arterial tension, diminished urea, subnormal temperature, and early psychical phenomena, and carries the causation of the disease one step farther back towards a possible origin. Hitherto the ultimate causation has not yet been determined.

In Mr. Horsley's dissections of animals, in which after ablation of the thyroid gland myxœdematous symptoms were produced, the tissue of the sympathetic nerves was found absolutely normal.

Scleroderma is met with in two varieties, according to the age of the patient. The sclerema neonatorum first appears in the lower extremities, thence spreads over the abdomen, downwards to the anus, and upwards to the upper extremities and to the

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face. From immobility of the lips the child cannot suck or drink sufficiently. Death occurs in from two to sixteen days.

The first symptom is coldness of the surface, associated with œdema, and then with a sensation of hardness to touch in the parts affected. The proximate cause of this condition is a disturbance of the circulation in the capillaries of the peripheral parts of the body. The colour of the skin may be red, but is often livid and pallid. After a few days the œdema and resistance disappear, and the skin often appears shrivelled or wrinkled. The surface temperature gradually falls. Kaposi says that, in appearance and to the touch, the impression suggested is that of a child's corpse in a state of rigor mortis.

That the impaired capillary circulation produces the loss of temperature in the skin is only in accordance with universal experience in many morbid conditions. Opinions are somewhat at variance as to what induces the impaired circulation. Chronic intestinal catarrh, follicular ulceration of the mucous membrane of the intestine, atelectasis pulmonum, pleuro-pneumonia, chronic bronchial catarrh, malformation of the heart, patency of the fœtal orifice of the heart and great vessels, meningeal apoplexy, hydrocephalus, &c. have all been found preceding or else coincident with the affection. Many of these lesions may be simply evidences of a morbid state of blood or of nervous centres, such as might cause also the impaired circulation. Defective nutrition and syphilis have preceded the affection in some instances.

It is possible that internal lesions might induce reflexly a state of vessels that might give rise to sclerema, but it is far more probable that the vasomotor centres are directly affected by a morbid condition of the blood itself.

Post mortem, the only lesions found have been in an œdematous infiltration of skin and a dense rigid stearinelike panniculus adiposus. No increase in the connective tissue.

It is doubtful whether scleroderma of the adult is pathologically a close congener of the above. Addison's cases of keloid were cases of scleroderma, and to the four cases described by him Dr. Fagge has added three. The skin affected by scleroderma is of the same length as before, but owing to the loss of its elasticity it is relatively too short for the parts beneath. The lesion appears chiefly on the upper extremities, but various parts of the body are affected, and it may occur in isolated patches or stripes or in a diffused form. The patches are often more or less pigmented. The skin feels firm, and is not movable on the subjacent tissue, but, as it were, soldered to the muscular fascia or to the bone. The semi-flexed position of the arms therefore cannot be altered. The features are petrified when the face is affected, and no emotions can induce any alteration of countenance. The temperature of the affected region is from 1 to 3 Fahr. lower than that of the healthy skin. Sensibility is often normal; as a rule, sweat and the sebaceous secretions are absent, but this is not universally the case. A similar indurated condition

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is sometimes met with in the mucous membrane of the tongue, gums, soft palate and pharynx.

Scleroderma progresses in one of two ways—either by way of resolution, the skin recovering its normal condition, or the diseased patches undergo atrophy. Hebra, indeed, is not certain whether the various forms of this disease have a common parentage, or are distinct and separate maladies. The former view is the more probable, from the fact that occasionally the different forms coexist in the same individual. It does not seem to be fatal: patients who die with it die from other causes.

As to the pathological anatomy, a dense cell accumulation has been observed round the vessels of the scleromatous skin, filling the adventitious space around the vessels. Kaposi found the papillæ of normal size and shape. The corium was remarkably thick at the expense of the reticular layer and of the tela cellularis, the subcutaneous tissue being filled by a dense collective tissue network. The vessels were numerous, though diminished in calibre and closely surrounded by connective tissue. Great accumulation of cells existed in the perivascular lymph space, and sometimes also in the meshes of the network of the corium.

The pathology of scleroderma is not inflammatory. Kaposi speaks of its fundamental nature as a diffused thickening and stasis of lymph in the cutis—this thickening of the lymph a result of a generally abnormal state of the nutritive process. Should the flow of lymph again become free, then also the infil-

tration disappears completely, and the cutis returns to its normal condition. Should the stagnation continue for a longer time, then out of the accumulated superfluity of nutrient material the previously normal connective tissue is formed in excess, becomes denser and increased in quantity. But his expression, 'a generally abnormal state of the nutritive processes,' is hardly explanatory. Vidal, considering the prominent part which the vessels must play in it, its symmetrical appearance and its origin, beginning like local asphyxia, thinks without doubt that the disease is primarily an affection of the vasomotor centres in the anterior columns of the cord, and he believes also that his views are confirmed by Charcot and Luys, who found sclerosis of this part in an autopsy they made in a case of scleroderma. This also is the view adopted by Grasset.

The distinction between the two diseases or two forms of the disease was drawn by Foerster of Würsburg. In both cases the skin loses its softness, elasticity and mobility and becomes as hard as a board. This condition in newly-born children is due to an infiltration of serous liquid in the meshes of the cellular tissue, and probably some chemical alteration of the fat. In the adult it is due to an excessive growth of cellular tissue, by which the stems of the meshes are thickened, the meshes themselves filled up, and the connection of the cellular tissue and the corium and subjacent parts, viz. the muscles, tendons, and fasciæ, are tightened. In a case given by him, the papillæ were found post mortem to have

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disappeared; the epidermis was thin; the deepest parts of the rete mucosum, elsewhere pigmental, were colourless; the connective tissue of the corium was thickened and hardened and the subcutaneous cellular tissue was firm like the corium. Very few nerves were seen. Dr. W. Pepper gives an unusual case in the 'American Journal of Medical Science' for 1871. A man, aged 48, had had inflammation of the feet eight years ago, involving the skin of the feet and legs up to about a hand's-breadth below the knees. There is now redness, swelling, heat and tingling pain. The sensation of the feet has been gradually impaired, and is now very complete. The skin is blue, its consistency is greatly increased; the temperature is reduced; the whole limb is of a hidebound, indurated character. The nails became brittle, broken, discoloured, and fell off. There is a tendency to ulceration about the joints of the toes. He could walk perfectly in darkness, although unconscious of touching the ground, and he felt as though walking through air. He could co-ordinate muscular movement perfectly. The nature of the case was sclerosis of skin, in which proliferation of lymphoid cells had actively advanced, and had induced such pressure on the cutaneous nerves as to cause anæsthesia.

A case peculiar from the amount of pigmentation was recorded by Mr. Gaskoin in 1877. A married woman, aged 40, experienced a mental shock when six months advanced in pregnancy, and was subsequently affected with certain stains or patches on various parts of the body, which had generally the

character of morphœa. These patches were extremely hard. A certain proportion of them were marked by a notable subsidence of the central portion of the patch. There was besides a temporary œdema and condition of tenderness. During the course of the past year these patches have gradually disappeared, and have given place to an intense staining of the general integument, so as to rival the colour of a negress. The limbs are like logs of wood, and the sclerema affects not only the integument, but the muscles, aponeuroses, and tendons; the joints are painful and contracted, and there is also pain in the bones. Dr. Tilbury Fox, commenting on this case, stated his belief that scleroderma must be regarded as a diffused, and morphœa as a localised scleriasis.

Dr. Harley records a case of slowly advancing sclerema with cardiac and gastric disorders. He concludes that the sclerema is merely the direct result of atrophy pari passu of the blood-vessels and of the lacunæ and canaliculi of the connective tissue, converting thereby the soft, moist, open, and well-nourished network into a hard, comparatively dry, close, and ill-nourished tendinous tissue. The atrophy Dr. Harley believes to be due to depression of sympathetic (vasomotor) nerve-power; that the functional disturbance of the heart and stomach is due to the same cause—in a word, that the case is one of slowly advancing paralysis of the sympathetic nerves generally, and that the affection is manifested at the periphery by the atrophy of the connective

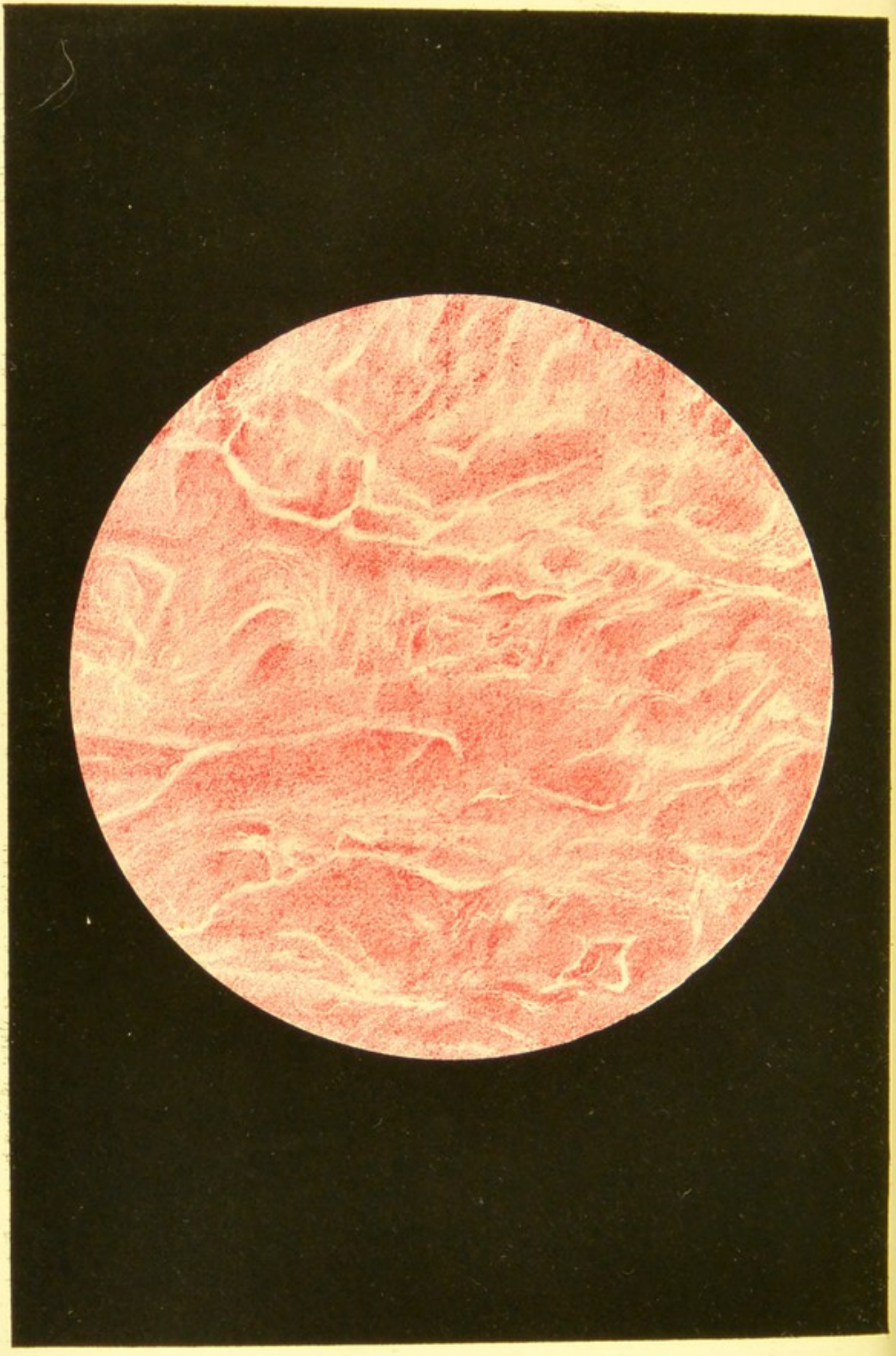
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tissue, and nearer the centre by the grave disturbance of the circulating and digestive functions.

A somewhat similar case was recorded by Dr. Harley in 1878. Very many examples of this condition have now been collected by various observers, probably nearly 200. Dr. Radcliffe Crocker has met with one of a girl, aged 13, in whom the condition existed over nearly the whole body, even the face, especially beneath the lower jaw, the glands being enlarged all round. The skin was as hard as frozen fat, the greatest induration being of the flexor surfaces. There was no pigmentation. The noticeable features in the case were, the rapid onset (the process being complete in less than a fortnight), the almost universal diffusion of the induration, the association with acute rheumatism and cardiac disease, the repeated attacks of pericarditis, the high temperature, and the œdema.

In a case recorded by M. Blachez, a man, aged 34, after having experienced a feeling of numbness in the hands, and nervous disorder, for two or three months, found himself suffering from growing puffiness of the hands and feet. This œdema, temporary at first, became permanent, and lasted from four to six months. Then only did the hardening of the skin begin, which manifested itself especially in the hands and feet, then in the legs, the belly, and later in the face. During the last fifteen days only some pigmentary spots had appeared on the hands. To sum up, this man had passed through three distinct phases—first, nerve disorder and numbness; second,

Pl. X



THE SKIN IN SCLERODERMA

a period of œdema and effusions; third, a period of localised induration.

M. Vidal has found little good in electricity. Warm douches to the spinal column had appeared to succeed.

Mr. Sympson, of Lincoln, gives the history of a little girl, aged 5, in whom scleroderma affecting the left lower extremity had come on gradually after an injury to the knee. The tendon of the tibialis anticus was rigidly contracted, and the foot was drawn up to an angle of 30 with the leg. The tendons of the tibialis anticus were divided subcutaneously—but the knee was left perfectly stiff and the skin resumed its former denseness. Subsequently by degrees the skin became more pliable and more easily pinched up. A well-marked band of pigment appeared along the inner aspect of the limb; the foot was held at a greater angle to the leg (about 45°) the movement of the ankle was more free. It was not unusual for the child to assume a position in bed with the affected limb so turned up alongside of the trunk that the foot was placed behind the back of the neck.

Mr. Sympson has also had prepared sections from the skin of a gentleman, aged 50, in whom, with the exception of the head and neck, the disease was universal. In it are well-displayed increase in the number of cells in the rete malpighii, enormous increase in the amount of connective tissue, and atrophy of the hair bulbs and of the subcutaneous fat.

Dr. Collins, of Nottingham, has lately met with a

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case which gives the main features of this condition in a graphic manner. The integument is thick and adherent to the subjacent tissues. There is evidently considerable increase of connective tissue. The face is as flaccid as a mask, from inability to call the muscles of expression into play; it is very thin. She cannot purse the lips so as to blow out a candle. The jaws can be separated about a quarter of an inch. The tongue can be protruded with difficulty and it feels leathery. Mastication is attended with considerable inconvenience, and at times with pain. The hands are skinny; the arm is adducted to the side; the forearm is contracted at right angles to the arm; and the hands are clawlike and rigidly flexed at the second and third joints, with the skin over them discoloured, thickened, and very tightly contracted.

This contracted skin and adventitious tissue appear to have caused absorption of all the muscles and fat of the hand; even the finger-bones are wasted, for they taper spindlelike. A similar compression and wasting is found over the greater part of the body. The knees are more or less fixed and the toes are distorted, so as to render progression difficult. There are patches of brown discolouration about the armpits, on the hands, neck, and on the abdomen. The surface temperature of the extremities 'feels perceptibly below normal.'

Illustrative cases might be recorded in large numbers. It suffices to say that the post-mortem appearances, that account for most of the phenomena

are not themselves the essence of the disease, but the results of the primary lesion; that this primary lesion is vasomotor paralysis, that the condition of the vasomotors is the precise opposite of what is met with in symmetrical gangrene of the extremities, and that this paretic state of vasomotors may be induced either by a traumatic lesion, by the effect of certain constitutional states on the vasomotor centre—states like anæmia, rheumatism, &c.—or sometimes by definite injury to important vasomotor centres, as by sclerosis of the anterior columns of the spinal cord.

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