THE TREATMENT OF APOPLEXY.

SUMMARY.—Apoplexy—Definition—Causes—Apoplexy by Rupture—Apoplexy by Anæmia—Apoplexy by Congestion—Treatment of Apoplexy by Hæmorrhage—Treatment before the Attack—Treatment of the Attack—On Blood Letting—Its Rôle—Inefficacy of Blood Letting—Treatment after the Attack—Pharmaceutical Means—Arnica—Strychnine—Electricity—Treatment of Apoplexy by Anæmia—Before, During, After the Attack—Treatment of Apoplexy by Congestion—The Apoplectic Temperament—Pathogeny of Cerebral Hyperæmia—Hygienic Treatment—Bad Influence of the Alcohols—Obesity and Apoplexy—Utility of a Normal Functional Condition of the Digestive Tube — Constipation — Purgatives — Aloes — Hæmorrhoids in Apoplectic Patients—Diagnosis—Conclusions.

GENTLEMEN—To devote a lecture to the treatment of apoplexy may seem to some of you useless, or even behind the times. This old word, apoplexy, to-day well-nigh obsolete, belongs rather to tradition than to clinical medicine; nevertheless, I persist in my resolution, and I will tell you why. If by reason of the progress of pathological anatomy we are able to know the pathogeny of the various cerebral apoplexies, and follow step by step the hæmorrhage from the production of the miliary aneurism to the rupture of the same, and even the necrobiotic alterations of the encephalon determined by emboli or thrombi, we are unfortunately unable in the living subject to establish a clear distinction between the effects of hæmorrhage and those of migrating or autochthonous clots, which give rise to symptoms so similar, although the causes are so dissimilar.[1]

Between the apoplectic attack resulting from hæmorrhage into the cerebrum, and that resulting from cerebral anæmia, there exist, from a clinical point of view, shades of difference but slightly marked, and save in exceptional cases, we are reduced, in making our diagnosis, to conjectures; conjectures which are often falsified by the post mortem examination. As this apoplectic ictus has been the subject of numerous discussions which have deeply concerned therapeutics, it has seemed to me desirable to retain the word, and to set forth the treatment, which is likely to give you the best results. But before going farther, let us come to an understanding about the signification of the word apoplexy.

That sudden suspension of cerebral action, complete or incomplete, persistent or transient, which characterizes apoplexy,[2] has had different interpreta-

[1] Recamier has established the following diagnostic signs:

In the case of hæmorrhage: Contracture of the paralyzed members, diminution of sensibility in the paralyzed parts, alteration of the intelligence.

In the case of ramollissement: Complete resolution of the paralyzed members, conservation and augmentation of sensibility, conservation of intelligence.

[2] Apoplexy, known from the most remote antiquity, described by Hippocrates, Galen, Celsus, Paul of Egina, Bœrhaave, etc., called *siderato*, *morbus attonitus*, *attonitus stupor*, etc.,

tions according to the prevailing medical doctrines. Under the domination of the doctrines of Hippocrates and Galen, the cause of the apoplectic phenomena was ascribed to sudden arrest of the vital spirits, or even to the presence of pituitous humor in the ventricles of the brain. But from the commencement of the seventeenth century the progress of pathological anatomy demonstrated that it was rupture of blood vessels which, in the majority of cases, caused the apoplectic stroke, and Morgagni brought clearly to light the preponderating importance of cerebral hæmorrhages. At the same time, authorities, while admitting the frequency of hæmorrhage in the pathogeny of cerebral apoplexy, did not, at the end of the last century, consider that as the sole cause, and while granting sanguineous apoplexies, they also affirmed the existence of serous apoplexies, and even certain essential apoplexies, called *apoplexiæ sine materia.* But from the beginning of this century, and especially since the work of Rochoux in 1814, apoplexy was regarded as nothing but the manifestation of cerebral hæmorrhage or congestion, and the notion of essential apoplexy was given up. The study of circulatory disturbances, and especially of the vascular changes produced by embolism, soon modified the exclusive view of Rochoux, and to apoplexy by rupture of blood vessels was now added apoplexy by embolism, or by cerebral anæmia. Therefore, I shall adopt with some modification the definition given by Schützenberger, and shall call cerebral apoplexy the

is constituted by an assemblage of cerebral phenomena, characterized by sudden loss of consciousness, with resolution or paralysis of the members, without arrest of the circulation or respiration.

It has been considered as the effect of the arrest of the circulation of the vital spirits in the veins, arrest of the vital force, repletion of the ventricles by a pituitous humor (Hippocrates, Galen), arrest of the sensitive or motor spirits (Avicenna), obstruction of the vessels by phlegm or atrabile; it is due to relaxation of the nerves, spasm of the meninges, (obsolete views).

In more recent times, by reason of anatomical examinations, apoplexy has been referred to compression of the brain and nerves by dilatation of the blood vessels, by effusion of blood or serum, etc.

Morgagni, Hoffmann, ascribe the principal cause to cerebral hæmorrhage; Hoffmann, at the same time, recognizes two forms:

Sanguineous apoplexy and pituitous apoplexy.

According to Rochoux and his pupils, apoplexy becomes the synonym of cerebral hæmorrhage, and for a long time this view has been adopted, and even now many authorities are of the same opinion as Rochoux, considering apoplexy as the equivalent of hæmorrhage, whether cerebral, medullary or pulmonary, etc.

This is to give an erroneous sense to the word apoplexy; as in the first place, all cerebral hæmorrhages are not accompanied by apoplexy, and the apoplectic ictus has been noted in many other maladies without the occurrence of cerebral hæmorrhage. The phenomena of apoplexy may manifest themselves in cases of effusion of serum in the ventricles (serous apoplexy); in other cases no appreciable encephalic lesion is found, as in apoplexies called nervous, the essential apoplexies (sine materia); the patients fall as if suddenly smitten down, just as in cases of epilepsy, or of eclampsia, and at the same time there is no cerebral hæmorrhage.

As has been seen, apoplexy may arise from many causes, and clinically, it must be considered only as a symptom; it is the expression of a cerebral perturbation, of a sudden suspension, temporary or permanent, of certain functions of the brain.

sudden suspension of cerebral action, produced by an internal cause affecting the circulation (vascular rupture, congestion or anæmia) acting directly on the encephalon.

I shall not enter here into the description of the symptoms of apoplexy, for which I refer you to your text-books,[1] but shall discuss the proper methods of treating this disease, unfortunately so common, giving reasons for such therapeutic hints as I shall offer But in order to understand the bases of our therapeutic endeavors, it is necessary that we should examine, as rapidly as possible, the pathogeny of the disease in accordance with the above definition.

[1] Cerebral apoplexy is rarely ushered in by prodromes; when these exist they consist in headache, vertigo, dazzling sensations, *muscae volitantes*, tinnitus aurium, formications, numbness; at times there is weakening of the memory, hesitation of the speech, etc.; often the invasion of the disease is sudden.

The patient is taken with vertigo, sensations of flashes of light; he makes several staggering steps, then falls suddenly, deprived of consciousness, of power of movement, of sensibility; he is in a state of complete resolution, but the beatings of the heart and the respiratory movements continue. The pulse is full, strong, regular; the breathing is painful, often embarrassed and stertorous; the face is often congested, the features drawn to one side; at each expiration the muscles of the cheeks and lips are puffed out; the eyes are turned to one side, like the head, and towards the sound side (conjugated-deviation of the head); the pupils contract under the influence of light, unless the hemorrhage affects the tubercular quadrigemina, the corpora geniculata or the optic commissure.

The patient is not always completely insensible; he groans in a characteristic manner when one pinches him; moreover, on lifting up the two limbs, lower or upper, it is observed that if one falls heavily and passively, the other falls less suddenly and its descent seems retarded by an instinctive, unconscious muscular contraction.

Often there are at the moment of the attack or a little after, involuntary evacuations of urine or fecal matters. Sometimes death is quite sudden, but this is quite rare; there is no such thing as *fulminating* apoplexy (apoplexie foudroyante), except in bulbar hæmorrhages. Generally in the apoplectic form of cerebral hæmorrhage, however considerable may be the loss of blood, the patients live several hours, sometimes days, and succumb either to the progress of the initial hæmorrhage or to the asphyxia brought on by the respiratory disorders.

In other patients one sees, at the end of a certain number of hours, consciousness gradually return, and the patient wakes out of his stupor, but he is hemiplegic, and the paralysis is seated on the side opposite to the lesion, *i. e.*, on the right side, when the hemorrhage is on the left, and *vice versa*.

With the paralysis there is sometimes anæsthesia more or less extensive; taste and smell are more or less altered. The hemiplegia is partial or total.

With respect to the face, the paralysis is often seated on the same side as that of the limbs, or it may be crossed and seated on the opposite side (crossed or alternate hemiplegia). One side of the face and of the forehead is immobile, without expression; the mouth is distorted, and the commissure of the lips is drawn in the direction of the sound side. At each expiration the cheeks are seen to be puffed out; the patient can neither blow nor whistle. The point of the tongue when projected from the mouth is often turned towards the paralyzed side, and sometimes does not seem to be touched by the paralysis. The speech of the patient is generally broken and confused.

The limbs are often unequally paralyzed; the arm is the most so. At first the temperature is higher on the paralyzed side; later the equlibrium seems to be restored; unless there be atrophy, in which case the temperature seems to be more elevated on the sound side.

The muscles of the pharynx, as well as all other muscles concerned in deglutition are paralyzed. Troubles in micturition and defecation are also frequent (retention, sometimes incontinence of urine and fecal matters).

The muscles of the abdomen and of the thorax do not, as a rule, participate in the paralysis.

Hemiplegia may supervene without any apoplectic stroke; it comes on during the waking hours and developes more or less rapidly; the patient, in full possession of his consciousness, has a little vertigo, observes flashes of light before him, then complains of formication and numbness and sees the paralysis gain, in whole or in part, one half of the body; at other times, having gone to bed well, he wakes out of sleep hemiplegic, without having lost consciousness and without having had any troubles of the intellect.

The duration of the hemiplegia is variable; sometimes it disappears after several days, weeks or months. Some patients even get completely well. In other cases we note the development of contracture of the paralyzed muscles, first of the lips, then of the leg, the flexors being affected rather than the extensors. There is at the same time atrophy of the limbs.

The hemiplegia does not often go away completely, and it may remain stationary indefinitely. When recovery takes place, movement reappears first in the leg, then in the arm, and restitution may be permanent. But in other cases, and especially when three weeks after the attack one notes exaltation of the tendinous reflexes, atrophy of the limbs is seen gradually to supervene, contractures of the paralyzed muscles and especially the flexor muscles, the extensors being rarely affected.

When the patients do not succumb to the progress of the central hemorrhage or as the result of a new attack, they are carried off, either by a consecutive cerebral affection, or by an intercurrent disease (such as pneumonia), or by the exhaustion consequent on the eschars, which so often form over the sacrum or trochanters in hemiplegic patients. (*a*)

Cerebral apoplexy is the result of three great factors: congestion, more or less intense, of the brain; sudden arrest of the blood destined to nourish a more or less extensive department of the encephalon, or, lastly, rupture of one of the blood vessels supplying a region of the brain; the latter constituting what used to be called sanguineous apoplexy. Once these affections were pathologically classed in the group of hæmorrhages, and supplementary, congestive, dyscrasic apoplexies were described. To-day we can expunge all these divisions, thanks to the discovery of miliary aneurisms made by Bouchard and Charcot. These pathologists have pointed out this capital fact, that rupture of the cerebral vessels is always preceded by the alteration of the walls of those vessels, veritable aneurisms, and that it is the rupture of these aneurismal portions which causes irruption of blood into the mass of nervous tissue, and all the consequent symptomatic and anatomo-pathological disorders which characterize the intracranial hemorrhage.[2]

[2] These aneurisms are undoubtedly the most frequent causes of cerebral hæmorrhages, especially in the hæmorrhagic apoplexies of old age. Charcot and Bouchard found them in *foyers* and in the ochreous cicatrices of ancient hæmorrhages, in the convolutions, corpora striata, optic thalami, and the pons. Visible to the naked eye, they appear in the form of

(*a*) Todd, Clinical Lectures on Paralysis, London, 1856. Grisolle, Pathologie interne. Monneret, Pathologie interne. Hardy and Behier, Pathologie interne. Durand Fardel, Traité Clinique des Maladies des Viellards, 1854. Rokitanski, Lehrbuch der Fatherlogischens Anatomie, 1856. Gendrin, Traite Philosphique de Medicine Pratique, 1838. Rochoux, Recherches sur l' apoplexie, 1814. Bennet, Pathological and Histological Researches on Inflammation of the Nerve Centres. Paget, on Fatty Degeneration of the Small Blood-vessels of the Brain, and its Relation to Apoplexy, London Medical Gazette, t. x. 6, 1850). Bouchard, Recherches sur la Pathologenie des Hemorrhagies Cerebrales, 1866.

Apoplexy by anæmia or necrobiosis, produces the same effects as sanguineous effusion, but by a mechanism altogether different. It occasions sudden cessation of the functions and death of a more or less circumscribed region of the brain, by reason of the arrest of the arterial circulation. We have here something comparable to the local gangrenes which accompany the ligature of the arteries of limbs, and this comparison is, moreover, so exact, that you know that the ligature of the carotid very often gives rise to an attack of apoplexy, with resulting hemiplegia of the opposite side.

This sudden stopping of the circulation in one of the arteries or arterioles of the brain, may have one or two origins: either it results from an alteration *in loco* of the arterial twig; chronic endarteritis of the vessel causing an autochthonous clot to be formed, which completely obliterates the lumen of the vessel: or else the vessel is sound, while the clot that obstructs it comes from a more or less distant point of the arterial system, or from the heart itself, being an embolus.

Between these two states, apoplexy from effusion of blood, and apoplexy from necrobiosis, or anæmia, is placed another group of apoplexies thus far badly defined, and which result from sudden and intense congestion of the brain, congestive apoplexy, or *coup de sang*. It is easily understood how an

little globular granules, whose diameter varies from two-tenths of a millimetre to one millimetre, and even more. They have a violaceous color, more or less deep, if the wall of the blood-vessel is thin, and the blood liquid; reddish brown or ochreous, or even black, if the blood is concrete; bluish or brownish, if the parietes are thickened by proliferation of the connective tissue elements of the adventitious membrane.

They are met with everywhere, but especially in the convolutions, either on the surface or at the point of reunion of the cortical and medullary substance, and at the same time in the pons, and great basal ganglia.

The number of these aneurisms is very variable; from one or two to a hundred or more.

Bouchard believes that these aneurisms (which are seldom or never seen before the age of sixty) are only the concomitant of an alteration of the blood vessels much more general, a sort of arterial sclerosis analogous to that which Rokitanski describes under the name of chronic periarteritis, but differing essentially from simple atheromatous and fatty degeneration. This vascular change affecting the entire arterial system of the encephalon, is especially conspicuous in the smaller intra-cerebral arteries, although the large trunks of the meninges and at the base of the brain may be pervaded by the same alteration, an alteration which consists in an exaggerated and often enormous multiplication of germinal elements in the interstices of the arterial trunks, and in the lymphatic sheath, and in atrophy of the muscular tissue. At the same time there is general dilatation of the vessel, with constrictions at various points where the circular fibres remain intact. In fine, we meet with abrupt dilatations in arteries thus modified, veritable aneurisms, whose rupture produces cerebral hemorrhage.

In the opinion of Charcot and Bouchard, the principal lesion in cerebral hemorrhage is peri-arteritis, or external sclerosis, while in the case of ramollisement, the lesion is endarteritis. or internal sclerosis. Zenker mentions an opposite view: that the lesion always commences by alterations of the adventitia. (*a*)

(*a*) (Cruveilhier, Anatomie. Pathologique—Bouchard and Charcot. Nouvelles Recherches sur la Pathogenie de l'hemorrhagié cerebrale, Arch. de Phys. 1868, p. 110—Bouchard, de la Pathogenie des hemorrhagies. These de Paris, 1869—Liouville, des anevrysmes miliares, Th. de Paris, 1871.)

afflux of blood, in great quantity, to the encephalon, or sudden stasis in the intra-cranial vessels, gives origin to a sudden but temporary arrest of the cerebral functions.

Having once these divisions well in mind,let us see how we can intelligently act when we have to deal with apoplexy from any one of these causes. I am assuming that we are in a condition to diagnosticate the particular lesion, a matter which I told you before is often involved in great difficulty.

As for cerebral hæmorrhages we are to take into consideration prophylaxis and the treatment during and after the hæmorrhage.

The rôle of prophylactic treatment is to prevent the production of these alterations of the walls of arteries which result in miliary aneurisms. It would seem that we are well nigh impotent in this regard. It is as much as ever if we can anticipate these aneurisms by heredity, and according to the laws which Dieulafoy has established, which go to show that the greater part of cases of cerebral hæmorrhage are witnessed in persons belonging to the same family.

In the majority of instances these miliary aneurisms do not reveal themselves by any symptoms, and their rupture is the first indication which we have of their existence. The most that we can do, in persons predisposed, in order to avoid the rupture of these aneurisms, is to make use of all those hygienic and medicinal means, which moderate the cerebral circulation; but I shall have more to say on this point when I come to speak of apoplexy from congestion.

The vascular wall gives way, the blood invades the cerebral mass, the patient has what is termed "a shock," he looses consciousness and becomes hemiplegic; you are called in haste; what are you to do? Twenty years ago the response would have been forthcoming; you would have made haste to take your lancet and bled the patient freely. This was one of the rules of therapeutics concerning which there was little or no variance of opinion.

Since the time of Aretæus, Paul of Ægina, Cœlius Aurelianus, Avicenna, Baglivi, down to Rochoux, Bouillaud, Monneret, Valleix, Grisolle, etc., the whole world was agreed that bleeding (or rather bleedings) was the proper way to treat apoplexy from blood effusion, and it was as much as ever if any extenuation of this procedure was granted in the case of anæmic and debilitated subjects.[1] The divers indications were pointed out for general bleeding, and

[1] In ancient practice, as soon as the patient has had an apoplectic shock, he was bled on the spot, and this was considered the only proper treatment. Blood letting was divided into general, derivative, and revulsive.

General blood letting was practiced from the arm, the jugular vein or the saphena. It was a subject of debate whether they should take blood from the sound side or the paralyzed side.

Aretæus used to bleed from the sound side. Baglivi from the paralyzed side.

Valsalva recommended to take blood from the jugular vein, but it was soon recognized that bleeding from this vein was dangerous practice.

Chauffard preferred the saphena, claiming that derivation from this vein has a revulsive as well as a spoliatory action.

The number of general blood lettings was often considerable; it was directed to continue them till the pulse was no longer hard and bounding.

Derivative blood letting.—This consists in the application of wet cups and leeches to the

for derivative and revulsive bleeding. It was a matter of serious discussion whether it were preferable to practice phlebotomy in the jugular, as Valsalva taught, or in the saphenous, as Chauffard advised, or even in the pituitary membrane, rather than at the end of the elbow, as Cruveilhier had proposed. One bleeding was not enough; you must bleed several times; at the same time Rochoux advised not to bleed more than four times in the generality of cases. They also discussed whether it were not better to bleed the paralyzed side than the sound side. Finally, they even went farther, and Bell, Nyemann, Zuliani and Gatherwood, taking their stand on hydraulic principles, more or less disputable, substituted arteriotomy of the temporal arteries for phlebotomy. Claudius Barbier, of Lyons, went ahead of all his contemporaries and predecessors in this practice; comparing the cranium to a tight cask, he advised that when the vein was to be opened the skull should be trephined.[2]

To-day, knowing better the mechanism of apoplexy, we ought to repudiate this kind of treatment, because nothing goes to show that we can at once arrest the hæmorrhage which results from rupture of some blood vessel, and to accomplish this result by bleeding, you would have to bleed the patient to syncope, a very dangerous procedure. Moreover, who is the physician that would now think of checking hæmorrhage from a small artery by blood letting? So then blood-letting is not to be thought of in the apoplectic seizure, for it is useless, and may be dangerous. It cannot prevent the consequences which result from rupture of the miliary aneurisms; it weakens the patient; places him in conditions of hydræmia and anæmia which oppose the clotting of the blood in the ruptured vessels and the obliteration of those vessels, and on this very account favors new hæmorrhages. It is plain then that we are impotent at the moment of the attack to combat the effects of the rupture of the cerebral arteries, and that our rôle consists in attending to symptoms as they arise. Observe if the alvine evacuations are free, if the functions of the bladder are regularly performed, place the patient in the best hygienic conditions possible.

Either the hæmorrhage is considerable and will prove fatal, or has affected only a limited part of the encephalon; the ensuing paralysis, then, is sharply defined, and, according to the extent of the hæmorrhagic *foyer*, it gradually disappears. In cases of this kind we may do much to prevent the inflammatory phenomena which accompany the disorders produced by the presence of the clot in the brain, and also to hasten the resorption of the clot.

To fulfill the first indication blood-lettings may do us some service. Much as I am opposed to this spoliative treatment at the moment of the attack, I am quite prepared to recognize its utility in the case of encephalitis. So, whenever after an apopleptic seizure, you note the appearance of fever and the other symptoms which characterize inflammation of the cerebral substance, you ought to apply leeches over the mastoid processes, or even take blood from the arm.

You may also apply ice to the head. Lallemand thinks that cold applications may do good during the attack itself. I do not agree with him, and believe that the only benefit which you can derive from this means is obtained in the inflammatory stage which succeeds the hæmorrhage.

In order to avert new hæmorrhages, and with this end in view, to combat congestive tendencies, drastic purgatives are indicated. It has been advised to give iodide of potassium on the ground that this medicament in small doses has the property of diminishing congestion of the encephalon; on data, more theoretical than practical, is also based the employment of ergot of rye in such cases.

As for the paralysis, consequent on destruction of cerebral tissue, it decreases in the ratio of absorption of the products of the hemorrhage. Many measures have been counselled to hasten the return of muscular movement. Some have proposed massage and stimulating frictions; others, from purely empirical considerations, certain medicaments, such as arnica; others still, in accordance with more exact physiological data, have proposed the use of strychnia and electricity.

I will say but little of frictions and massage, and I see little that is disadvantageous in the employment of these means, except it may be the danger of provoking traumatism of the paralyzed parts, already the seat of trophic disturbances often sufficiently intense to cause phlegmons and gangrene. As for arnica and all stimulating medicines, such as melissa, mentha, and lavender, they do not play any active part. It only remains to consider strychnia and electricity.

Strychnia was especially employed by Magendie and by Bradsley. It was hoped that the contractions determined by this medicament would hasten the return of muscular movements. You should be very careful in the employ of

occiput or to the angle of the jaws. As many as twenty leeches would be placed over the tract of the jugulars; these were repeated two or three times after general bleeding. (Monneret.)

Cruveilhier advised to bleed from the pituitary vein, and for this purpose he used a special instrument which he invented.

Revulsive blood letting.—This consists in the application of leeches to different parts of the body, more or less distant from the seat of the hæmorrhage, as the malleoli, the thighs, anus, etc.

Attempts have been made to formulate the application of blood letting in apoplexy. Hints were obtained from the state of the pulse. When the pulse was full but intermittent, it was necessary, according to Shauffer, to refrain from blood letting. The same precaution was necessary in the case of feeble old men. VanSwieten cautions against bleeding in grave cases, for, he says, if the patient dies after the blood letting, the physician would be blamed for his death.

It was generally admitted that in hæmorrhages which seemed to accompany active cerebral congestion bleeding was especially indicated, as being palliative and preservative. (*a*)

[2] Sir Charles Bell believed that arteriotomy was preferable to phlebotomy in the case of extravasation of blood in the brain; reasoning from doubtful hydraulic principles, he thought that the former operation diminished, while the latter increased the afflux of blood to the brain. Barbier would even bore a hole in the cranium, by means of a simple drill, to avail himself of atmospheric pressure in depleting the brain. (Bell—The Different Effects of Arteriotomy and Phlebotomy in Apoplexy—British Medical Journal, January, 1843. Barbier—A New Mode of Treating Apoplexy—Jour. des Conn. Med., Juillet, 1843.

(*a*) Aretæus, De curatione apoplexiæ. Chauffard, Des avantages de la saignë revulsive dans la plupart des maladies de la tête. (Arch. Gén. de Med., Mars, 1832, p. 2871.) Monneret et Fleury, Compendium de Medecine Pratique, t. I p. 278. Cruveilhier, Dict. de Méd. et de Chir. Pratiques, t. III, p. 255.

strychnia in hemiplegia, for this alkaloid produces congestion of the cerebro-spinal axis; a congestion always injurious when the patient is suffering from the effects of cerebral hemorrhage.* On the whole, it is better to rely on electricity.

Electricity accomplishes two purposes; it opposes the trophic disturbances which accompany hæmorrhages into the brain, and above all it combats the consecutive alterations in nerves which find their central termination in the part of the brain which has been destroyed. Bouchard has described for us the march of these secondary scleroses, which engender persistent disorders in the paralyzed parts. You can employ here the galvanic current, or the faradic current. When you wish particularly to influence the nutrition of paralyzed parts, you employ galvanism; when you wish to provoke muscular contraction and prevent atrophy in paralyed muscles you use faradisation. But you should exercise great caution in the use of electricity and not resort to this therapeutic agent till all the congestive or inflammatory symptoms have disappeared. Electricity, when too long applied (or in too strong currents) induces in the corresponding parts of the brain an excitation more injurious than useful.

In the case of apoplexy from embolism or thrombosis, we can do little in the way of prophylaxis before the attack. It is necessary to avoid all circumstances which favor the production of an embolus in the left heart and in the arterial system, or those alterations of the encephalic vessels which give origin to autochthonous clots. Here, however, we are well-nigh powerless. When the apoplectic seizure has taken place, and a clot has caused obstruction of an important blood vessel, inducing necrobiosis of the part of the encephalon supplied with its nutrient blood by that vessel, our rôle is still an impotent one, We cannot by any therapeutic measure re-establish the interrupted circulation, and we are just as powerless to hasten resorption of the necrosed parts. As you see, then, when we are concerned with cerebral necrobiosis, we can only be spectators of the disorders produced without the ability by modes of treatment, more or less energetic, to arrest the evil at its commencement or modify its evolution.

It remains to consider apoplexy due to cerebral congestion. Here we can accomplish much more, and we can in a measure successfully oppose cerebral hyperæmia. It is to this group of apoplexies that we have seen applied the name apoplectic temperament or constitution. You know the description that has been given of typical cases; you have heard described the flushed countenance, the injected eyes, the short, thick, massy neck and large shoulders of apoplectic subjects who experience under the influence of slight excitements bursts of heat and flashes which bespeak the imminence of dangerous cerebral congestions.

[*In American practice, strychnia is much given in hemiplegia, but with most physicians, more from routine, and from some vague idea that it may be useful to arouse dormant motor power than from any experience of its utility. If given, it should be given very cautiously and in a commencing dose not exceeding one-twentieth of a grain. The dose may be gradually increased till some jerking or tension is felt in the paralyzed muscles. (Vidè Stillé, Therapeutics, vol. 2, p. 155.) A good form is the liquor strychniæ; dose five to ten drops. TRANS.]

There are a great number of affections of which cerebral hyperæmia is a symptom. The description above given applies to arthritic patients with congestive tendencies, to individuals affected with mitral diseases, to certain emphysematous subjects, in all of which cases you will observe the congestive or apoplectic facies above set forth. Cerebral hyperæmia arises from a great number of causes, and the treatment ought to vary according to the affection of which it is an expression.

Already when treating of diseases of the heart I told you what you ought to do to combat congestive cerebral tendencies, and I need only remind you that the most of the therapeutic measures which have been proposed for apoplexy are directed not to the hæmorrhage, but to the hyperæmia, and this is the end of both hygiene and prophylaxis. A long time ago Lancisi showed the importance of hygienic therapeutics in these cases, and your attention ought to be directed principally to these two points: diet on the one part, and regularity of the bowels on the other.

As for diet you ought rigidly to proscribe all those aliments and all those beverages which are likely to induce cerebral excitation. Your patients must abjure altogether wines, generous liquors, in fact alcohols in general, which have a doubly deleterious action in apoplexy, for they not only produce hyperæmia of the brain, but they also engender alterations of the blood vessels, the result of which is rupture of those vessels or obturation. You should caution your patient not to use too highly-seasoned food, and should put him on a regime consisting principally of white meats and fresh legumes and other vegetables; fatty and feculent matter should be excluded, and, in short, everything which can augment the production of fat in the economy.

Do not forget that the hyperæmia, and the apoplectic tendencies which are its consequences, are often connected with obesity. Polysarca in hindering the play of the diaphragm, and especially in opposing the regular functionating of the heart by fatty accumulation around that organ, and by the alteration of the heart muscles which attends it, almost constantly, gives rise to hyperæmia of the brain; and it is for this reason that the hygienic treatment of obesity is quite applicable to congestive apoplexy. Therefore, you should establish by daily exercise a relation always exact between nutrition and combustion, and you should combat by appropriate means all the symptoms produced by slackened nutrition.

You should also endeavor to promote a healthy state of the digestive tube. Constipation ought to be avoided at any cost, and you ought always to keep up a certain state of diarrhœa in these congestive and plethoric patients. Here the purgative mineral waters are indicated—Pullna, Freiderichshall, Hunyadi Janos, Ems, Sprudel. Here you may give aloes with excellent effect, which, besides unloading the intestines, causes congestion of the rectum and produces hæmorrhoids. A great many apoplectic patients have piles, and these latter act as safety valves to the head, both by reason of the congestion which they determine in the region of the anus and the fluxes of which they are the seat.

The alkaline waters[1] are good in these cases, not because they anæmiate

[1] Carriere has proposed the internal use of alkaline medicines as a rational treatment

the patient, but because they regulate nutrition.[2] You should watch the urine of your patient, for many apoplectics are gouty, and you ought always to keep the urinary secretion abundant.

As a pharmaceutical means, besides purgatives and diuretics which are always of some utility, you may make use of arsenic, iodide of potassium and aconite. The latter diminishes in a notable manner cerebral congestion. Dry and wet cups, leeches, and sometimes even venesection may be valuable auxiliaries. With regard to venesection: much as I am opposed to blood-letting in the period of hæmorrhage, I am convinced that in cases of great hyperæmia of the brain, with flushing and lividity of the countenance, blood-letting may temporarily do some good.

Such are the therapeutic rules applicable to the three varieties of apoplexy to which I have called your attention. Our treatment would be more accurate and scientific were we always able to distinguish the one from the other of these three varieties. Unhappily this is not an easy matter.[3] If we can generally diagnosticate cerebral hyperæmia, it is really impossible to distinguish hæmor-

in cases of predisposition to apoplexy. He employs the bicarbonate of soda, of which he gives nearly two drachms a day. But when there is apoplectic congestion, he replaces the bicarbonate of soda by ammonia, giving from five to ten drops of the spirits every hour. (*a*)

[2] Picquot in Bull. de Ther. t. 61 lxi., p. 321, has advised the arsenical treatment as prophylaxis. This is the formula:

R	Arceniate of Potash	00.05
	Tinct. Aconite	10.00
	Tinct. Digitalis	5.00
	Distilled water	300.00

M. Dose, a tablespoonful morning and evening in half a tumbler of sweetened water two hours before meals.

[3] The diagnosis of cerebral hæmorrhage is sometimes attended with difficulty, by reason of the number of diseases which may present similar symptoms at the commencement; apoplectic and hemiplegic symptoms. Meningeal hæmorrhage, cerebral congestion, acute ramollissment, saturnine and uræmic encephalopathies and pernicious fever with comatose symptoms may be accompanied with apoplexy. In uræmia and pernicious fever the study of the temperature will help to clear up the diagnosis; in the commencement of the hæmorrhage the temperature is lower than natural, then it goes on increasing till the fatal termination; in pernicious fever, the central temperature is always superior to the normal; in uræmia, with comatose symptoms, there is a progressive lowering of the central temperature.

In cerebral congestion the diagnosis is determined by the march of the disease; at the end of a variable period, all the morbid symptoms disappear.

Syncope, asphyxia, alcoholic intoxication may lead into error. But in syncope, the suspension of respiration and circulation; in asphyxia, the respiratory troubles; in alcoholic intoxication, the odor from the breath, will serve to clear up the diagnosis.

In intraventricular hæmorrhages, or in those which take place in the substance of the pons, there are often convulsive seizures simulating epilepsy; the aspect of the patient, the tongue which presents fissures or cicatrices, the dilatation of the pupils, the temperature and then the march of the disease will remove all doubts. It is the same for certain apoplexies which are only attacks of epilepsy and to which Trousseau called attention.

The diagnosis of meningeal hæmorrhage is more difficult: according to Boudet, this

(*a*) Carriere, on the rational treatment of cerebral congestion and of apoplexy by alkalies and in particular by bicarbonate of soda. Paris, 1854.

rhagic apoplexy from apoplexy by anæmia, and we can only make hypotheses and conjectures when we come to actual experience at the bedside. This very circumstance should lead us to be chary with regard to any active treatment during or after the apoplectic seizure. Better always to do nothing than to do harm.

In my next and final chapter, I shall consider the treatment of spinal myelitis.

kind of apoplexy is likely to be attended with contracture as an initial symptom. In the ordinary form of hæmorrhagic apoplexy, contracture is a late symptom.

Chronic ramollissement is distinguished from cerebral hæmorrhage by its march. During its progress it is accompanied by cephalalgia, vertigo, intellectual enfeeblement, numbness which is habitual, and sometimes by contractures.

More difficult is the diagnosis of acute ramollissement. The *debut* may be apoplectiform, and without prodromes. At the same time the loss of consciousness is apt to be more transient and less complete in ramollissement. Moreover, in ramollissement by embolism, lesions may often be noted in other organs, which, produced by the same cause, shed light on the diagnosis (pulmonary apoplexy, infarctus of the spleen, hematuria, etc.) After the attack the hemiplegia is alike in both diseases, and it is almost impossible to distinguish them. You must be guided by the evolution of the disease.

In softening, and especially senile softening, from endarteritis, the symptoms often present oscillations; the paralysis is less complete at certain hours of the day. Aphasia is a symptom less common in apoplexy from hæmorrhage than in ramollissement.

According to Charcot, conservation of consciousness with sudden invasion, belongs rather to ramollissement; variable hemiplegia belongs to it exclusively, and aphasia almost exclusively.

Hæmorrhage often coincides with hypertrophy of the heart and Bright's disease. Cerebral ramollissement by embolus occurs in the same patients, and accompanies visceral infarctus.

Ramollissement by thrombosis is seen especially in individuals who are a prey to tuberculous or cancerous cachexia. Ramollissement by senile endartitis is frequently accompanied by atheromatous degeneration of the arteries of the extremities.