A study of some points in the pathology of cerebral haemorrhage / Translated from the French, with notes by T.J. Maclagan.

Joseph Cook

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A

STUDY OF SOME POINTS

IN THE

PATHOLOGY OF CEREBRAL HÆMORRHAGE.

BY

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TRANSLATED FROM THE FRENCH WITH NOTES

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TRANSLATOR'S PREFACE.

THE pathology of diseases of the brain, from the importance of the subject, as well as from the obscurity with which it is surrounded, has always attracted much attention both in this country and on the continent. To no part of the subject has so much attention been paid as to the pathology of cerebral hæmorrhage; and yet our knowledge of the mode of production of that accident is still most unsatisfactory. The greatest variety of opinion exists as to the exact cause of the hæmorrhage, and as to the connection which exists between it and cerebral softening, atheroma, cardiac hypertrophy, kidney disease, &c. The names of Abercrombie, Todd, Rochoux, Durand-Fardel, Rokitansky, Leubuscher, and of many other illustrious men, are inseparably connected with the literature of the question, and are a sufficient guarantee that the cause of this confusion is to be sought for rather in the difficulty of the subject than in want of ability or competence on the part of those who have investigated it.

The subject still demands attention as well from its obscurity as from its importance.

The most original and important of all recent works on the subject of cerebral hæmorrhage is unquestionably that of which I now give an English translation.

It was published in Paris early in 1867, and at once attracted much attention, both from the interest attaching to the subject, and from the novelty of the views therein expressed. I happened to be studying in Paris at the time, and naturally had my attention directed to the work. I had, moreover, the great advantage, especially at the clinique of Professor Behier at la Pitié, of seeing the miliary aneurisms which M. Bouchard has described, in various cases of cerebral hæmorrhage, and can bear personal testimony to the accuracy of his description, as well as to the minute care and exactness with which they are represented in the plates.

The importance which is attached at the present day to the state of the minute arteries in health and in disease, forms one of the most striking features of modern medical research. One can scarcely take up a journal without seeing a reference to some question in physiology, pathology, or therapeutics, in which the condition of these vessels forms the point of interest. In therapeutics especially great advance is being made regarding our knowledge of the mode of action of many drugs, and some of our most valuable remedies are now believed to owe their good effects to their stimulating or restraining action on the vaso-motor system of nerves.

The importance of the physiological action of the minute arteries in regulating the supply of blood to a given organ or part of the body cannot be over-estimated. And it can readily be understood that any pathological change in their contractile elements, such as that described in the following pages, must have a

serious effect on the organ whose vessels are so altered, and must also render the person in whom it occurs less amenable to the action of those remedies which act through the vaso-motor system.

The interest of MM. Charcot and Bouchard's researches is centered in the miliary aneurisms which they have so well described, and which they have shown *par excellence* to be *the* cause of hæmorrhage into the substance of the brain.

If it should hereafter be found (as, from the observations of M. Liouville, seems not at all unlikely) that similar changes take place in the minute arteries of other organs of the body, the observations of MM. Charcot and Bouchard will have a wider sphere of application than is at present accorded to them, and may throw much light on the pathology of other diseases than cerebral hæmorrhage, and especially of those chronic ailments which chiefly occur in advanced life.

It is possible, too, that changes in the minute arteries (functional or organic) may play a not unimportant part in the pathology of other forms of cerebral disease, and even of many of those mental ailments which are unconnected with appreciable organic change in the proper tissue of the brain.

The great importance of the subject, and the fact that the labours of MM. Charcot and Bouchard have not attracted in this country such general attention as they certainly merit, have induced me to publish this translation.

At the suggestion of Dr Bouchard I have considerably curtailed those cases which I have given, and have omitted many altogether.

I but carry out his expressed wishes in saying that the work to which his name alone is attached is the result of labours which were shared by M. Charcot, to whom he is anxious that there should be accorded an equal share of the honour which the authors of such an original and able investigation certainly merit.

At the further suggestion of Dr Bouchard I have added as an appendix a more recent joint paper by M. Charcot and himself, which was published in the "Archives de Physiologie," and in which are embodied the results of their further investigations into the nature of the miliary aneurisms, and their connection with hæmorrhage.

DUNDEE, *July* 1872.

CEREBRAL HÆMORRHAGE.

INTRODUCTION.

THE multiplicity of the causes which seem to have an influence in the production of cerebral hæmorrhage, and the diversity of the lesions which play an important part in its pathology, have induced many authors to regard effusion of blood into the brain as only an accident or complication of different diseases.

This opinion, which undoubtedly applies to a certain number of cases, appears to me much too absolute. The study of the pathology of sanguineous apoplexy can alone lead to a solution of this important question. Without pretending to treat fully in this work of all the points pertaining to that study, I shall endeavour to show, by examining in succession the influence of the various lesions which act as proximate causes of cerebral hæmorrhage, that there are cases, in my opinion the most numerous, in which hæmorrhage into the substance of the brain presents itself as a distinct and separate disease, characterised by changes peculiar to itself, and really meriting a special place in nosology.

In order to attain this end, the importance of which is evident, and to the difficulties of which I am quite alive, we must carefully examine the opinions already enunciated regarding the pathological modifications of the local circulation in the brain, and the morbid changes of the intra-cranial vascular system.

Researches carried on during two years at the Salpêtrière, under the direction of M. Charcot, enable me to bring to the support or refutation of these opinions the evidence of facts. As to the special changes which appear to me to be the real cause and the specific lesion of the disease *cerebral hæmorrhage*, I think that there will be recognised in them at least a certain novelty. The verification of these is the result of labours carried on during the course of the past year, labours in which M. Charcot kindly allowed me to participate; and I should be wanting, both in gratitude and in truthfulness, did I not point out at the commencement of this thesis the large share that he has had in its production.

Plan and Divisions.

The various pathological conditions included under the common denomination *cerebral hæmorrhage* are certainly all due in the end to rupture of some part of the intra-cranial vascular system. I do not in any way wish in this work to enter on the study of those vascular ruptures which may lead to extravasation, either outside the dura mater, or in the false membranes which may be developed on its internal surface. Neither shall I consider the hæmorrhage which appears to be due to rupture of the sinuses or large arteries of the base, nor that which has its origin in aneurisms of these arteries, or of the large meningeal branches. I shall leave out of account also extravasations produced primarily in the subarachnoid space, and in the meshes of the pia mater, as well as those which may be produced either on the surface or in the interior of certain intra-cranial tumours. It is true that in some of these cases the surface of the brain may be torn, and the effused blood clear a way for itself into the depth of the cerebral substance; but I wish to limit myself to the study of cerebral hæmorrhage properly so called, that is to say, to the hæmorrhage which results from the rupture of vessels situated in the substance of the brain itself.

A survey of the long list of causes, as numerous as various, to which the production of this hæmorrhage has been attributed, enables us to group them under three great heads:—

Rupture of the vessels results—

1. From abnormal tension of the blood contained in the vessels.

2. From change in the consistence of the surrounding tissue, which is primarily affected, and no longer affords sufficient support to the vessel.

3. From diminished resistance of the walls of the vessels which have lost their natural cohesion.

It may appear doubtful under which of these three proximate causes, the only ones possible, some cases of cerebral hæmorrhage should be ranked, especially those which seem to be connected with change in the blood, and which occur either during the course of certain chronic dyscrasiæ, in fevers, or in septic diseases. As we shall see further on, it is to the third order of causes that they appear to be due.

Before considering the influence exercised by the different causes which have thus been classed under these heads, I would remark that this distinction is altogether theoretical, and that a special examination of the facts will not fail to show that a variety of causes, often different in their nature, habitually lead to the same result. It is exceptional to find cerebral hæmorrhage produced by increased blood-tension alone, and perhaps not a single fact could be adduced capable of establishing the dependence of the extravasation solely on diminished consistence of the surrounding tissue, or weakness of the vessel itself. The complex nature of the facts is little in accordance with these theoretical divisions, and in cerebral hæmorrhage in particular, classifications tend rather to make known than to explain the facts. I shall not attach equal importance to the various points just indicated, and on which the principal divisions of my work shall bear. We shall see in particular that causes of the first order frequently combine with those of the two last, and that they generally intervene as determining causes. We shall have to return to that order of causes in the two last chapters, and shall now only briefly consider the pathological influence which may be exercised by tension of the blood within the vessels.

CHAPTER I.

ON EXAGGERATION OF THE TENSION OF THE BLOOD IN THE VESSELS OF THE ENCEPHALON AS A CAUSE OF CEREBRAL HÆMORRHAGE.

Some cases of cerebral hæmorrhage appear to be due neither to altered consistence of the brain substance, nor to change in the walls of the vessels. The rupture is produced by the accession of an undue quantity of blood to the vessels of the brain, or by the more forcible impulsion of that fluid through the vessels.

Various causes indicated by authors may be referred to the first head. Increase in the total quantity of blood, producing distension of the cerebral vessels, as well as of those of other organs, has been cited by some in explanation of the so-called plethoric apoplexy. I shall not stay to discuss facts which do not appear to be less problematical than the interpretation which has been given of them.

In some cases partial displacement of the mass of the blood produces an accumulation of that fluid in certain internal organs, and chiefly in the brain. This is what occurs when sudden spasm of the cutaneous vessels drives the blood from the surface. Some cases in which cerebral hæmorrhage has suddenly struck down young people in a cold bath, and in which no change was found, either in the vessels or in the brain substance, might be referred to this cause. But, while admitting that in such cases there may have been no congenital weakness of the vessels of the brain, and no disease of the heart or lungs, it is not clear that, at the moment at which the accident occurred, a more energetic contraction of the heart, or pulmonary spasm interfering with the return of the blood, may not have played a part in the complex etiology of the phenomenon in question.

Other partial displacements in the mass of the blood have an altogether mechanical cause. I would instance compression of the thoracic and abdominal organs, which may be produced either by external violence, or by strong efforts of defæcation, coughing, or vomiting, as an immediate consequence of which cerebral hæmorrhage may result. But, apart from violence and child-birth (in which the uterine contractions acting on the trunk of the fœtus may forcibly and for many hours drive the blood towards the partly disengaged head, and that, too, in a brain whose young capillaries possess but feeble powers of resistance), such hæmorrhage nearly always has some other cause aiding its production, and the effort which displaces the blood becomes the determining cause of a rupture, for which the way had already been prepared by a pre-existing morbid process.

I would also refer here, but without insisting on it, as I shall have to return to the subject in the following chapter, to the increased tension of the blood which is mechanically produced in the collateral vessels when a neighbouring arterial trunk becomes obliterated. We shall, by-and-by, see what part this cause is called upon to play in the production of the capillary apoplexy which occurs around those softenings which result from arterial obliteration.

Some influence in the production of cerebral hæmorrhage has been ascribed, as I have said, to the more forcible impulsion of the blood which flows through the vessels of the brain.

This impulsion may be increased by two causes: by increased energy of the left ventricle, and by diminished elasticity of the arteries. Hypertrophy of the left ventricle has been cited by Legallois, by Corvisart, by Bricheteau, by M. Bouilland, and by Ménière, in explanation of the rupture of the vessels of the brain in hæmorrhage. Its influence has been altogether denied by Todd,* by M. Grisolle,† by M. Monneret,‡ but appears, on the other hand, to have been accepted without reserve by Rokitansky§ and by Leubuscher.‖

* Clinical Lectures on Paralysis. London, 1856. P. 116.

† Traité de Pathologie Interne, t. 1er, 1862. P. 729.

‡ E. Monneret. Traité Elémentaire de Pathologie Interne, t. 1er, 1864. P. 32.

§ Rokitansky. Lehrbuch der pathologischen Anatomie, band ii. 1856. P. 450.

‖ R. Leubuscher. Die Pathologie und Therapie der Gehirn-Krankheiten. Berlin, 1864. P. 220.

MM. Behier and Hardy thus express themselves regarding this agency:—"Without absolutely denying the influence of hypertrophy of the heart, we believe that Legallois, and those who have supported his view, have exaggerated the importance of this anatomical fact, which indeed may be no more than a simple coincidence."*

Statistics, to which it is customary to have recourse in researches of this kind, would, however, appear to be opposed to these conclusions. In 83 cases of cerebral hæmorrhage collected by M. Durand-Fardel,† and in which the condition of the heart was noted, he found hypertrophy of that organ 42 times, and in 27 of his own cases that lesion occurred 14 times. But hypertrophy of the heart rarely exists as an isolated lesion; and it would be difficult to ascribe the hæmorrhage to that cause when the thickening of the ventricular walls has resulted from aortic constriction. When this hypertrophy complicates a state of rigidity of the main arterial trunks, or some obstacle to the flow of the blood, the increased tension of that fluid consequent on these lesions perhaps plays a more important part than the cardiac affection in the production of the hæmorrhage. M. Gendrin‡ had already pointed out the frequent coincidence, in those who die of sanguineous apoplexy, of arterial incrustations and hypertrophy of the left ventricle, "of which they (*i.e.*, the incrustations) are often the cause." So long as the arteries possess their normal elasticity, and the blood flows freely along, hypertrophy of the heart seems only to increase the rapidity of the blood-current, without notably augmenting its tension. Hasse§ had already made this observation, which has been repeated and extended by Eulenburg.‖ This latter thus expresses himself:—"Hypertrophy of the left ventricle can favour the production of cerebral hæmorrhage only when it

* Traité Elémentaire de Pathologie Interne, t. iii. p. 413.

† Durand-Fardel. Traité Clinique et Pratique des Maladies des Vieillards, 1854. P. 292.

‡ Gendrin. Traité Philosophique de Médicine Pratique, 1838. P. 483.

§ Handbuch der specialien Pathologie und Therapie, von Virchow, band iv. 1855. P. 383.

‖ Ueber den Einfluss von Herzhypertrophie und Erkrankungen der Hirnarterien auf das Zustandekommen von Hæmorrhagia Cerebr. (Archiv für patholosische Anatomie und Physiologie und klinische Medicin). Band xxiv. 1862. P. 361.

increases the mean normal tension of the aortic system; and this occurs only when the hypertrophy depends on obstacles to the peripheral circulation, and especially on atrophy of the kidneys and diffuse arterio-sclerosis, never, on the other hand, in cases of hypertrophy compensating for valvular lesions of the left heart. It may be stated generally that hypertrophy of the heart accompanies cerebral hæmorrhage much more rarely than degeneration of the arteries." It is evident that the part played by cardiac hypertrophy is thus singularly limited. I would merely refer to the opinion of Ménière, who ascribes to hypertrophy of the heart in pregnancy the cerebral hæmorrhage which sometimes occurs in pregnant women.*

Incrustations of the large arterial trunks, by the resistance which they offer to the lateral effort of the blood, augment, as we have just seen, the tension of the arterial system. The loss of their elasticity modifies also the continuity of the stream of the blood, which reaches the brain in a jerking manner. This seems to be a more potent cause of hæmorrhage than hypertrophy of the heart. But these are only secondary agencies, incapable by themselves of producing rupture of the

* Dr Quain, in the Lumleian Lectures delivered at the College of Physicians of London in March 1872, has described a form of hypertrophy of the heart in which there is no increase, and often a diminution of the muscular fibres, the greater size of the heart being due to increase in the connective tissue, by which the muscular fibres are surrounded and more or less compressed. Such a heart is dynamically weak, and any signs of apparently increased strength which it may show during life are the result, not of increased cardiac force, but of increased effort on the part of the embarrassed (and possibly diminished) muscular fibres to overcome the restricting action of the fibrous tissue which surrounds and hampers them in their work. It is obvious that the recognition of this form of cardiac enlargement, which may very readily be mistaken for true hypertrophy, has an important bearing on the question of the relation which exists between hypertrophy of the heart and cerebral hæmorrhage, and that it must have a modifying effect on such statistics as those of Durand-Fardel and Eulenburg, this effect being still further to diminish the percentage of cases in which cardiac hypertrophy can be regarded as even an indirect agency in the production of cerebral hæmorrhage. If further investigation should show that this cirrhotic hypertrophy (if I may so term it) is the form which usually occurs along with chronic Bright's disease, the question will naturally arise whether the change in the cardiac tissues may not be one local sign of a general disease of which the kidney disease forms another. See also footnote at page 28. T. J. M.

vessels of the brain, and at best can only be looked upon as aiding causes.

Side by side with those causes of increased blood tension, which result from exaggerated impulse, or from rigidity of the large arterial trunks, must be placed those which interfere with the free flow of that fluid outside the cranial cavity. These obstacles to the return of the blood may be tumours pressing on the cerebral veins, obliteration of the sinuses or jugular veins by phlebitis or thrombosis, tumours of the neck, and traumatic lesions capable of producing pressure on the veins of that region, diseases of the heart and lungs accompanied by venous stasis, asphyxia, and, in short, all the common causes of cerebral hyperæmia. With the exception of disease of the sinuses of the dura mater, which we shall not now consider, as we shall have to return to that subject in the following chapter, there is perhaps not one of these causes which by itself is capable of producing cerebral hæmorrhage. I believe that even less importance should be ascribed to that relative increase of the blood tension which would result from that so-called tendency to vacuum, which has been regarded as one of the consequences of cerebral atrophy. This congestion *ex vacuo* is, however, accepted by Leubuscher * and by Niemeyer† as a cause of cerebral hæmorrhage, but has been formally denied by Hasse.‡

Upon the whole, the local and general changes which lead to increased tension of the blood contained in the vessels of the brain play but a very limited part in the pathology of cerebral hæmorrhage. We shall presently see whether the same may be said of the causes which tend to diminish the resistance of the vessels.

* Die Pathologie und Therapie der Gehirn-Krankheiten, p. 211.

† Eléments de Pathologie Interne et de Thérapie : traduction de Culman et Sengel, t. ii. 1866. P. 201.

‡ Handbuch der speciellen Pathologie und Therapie, von Virchow, band iv. p. 385.

CHAPTER II.

ON DIMINISHED CONSISTENCE OF THE TISSUE OF THE BRAIN AS A CAUSE OF CEREBRAL HÆMORRHAGE.

THE opinion which has ascribed to altered consistence of the brain tissue an important part in the production of cerebral hæmorrhage is not of very old date. It was promulgated for the first time in a doubtful manner by Rochoux* in 1814, though similar ideas had already been enunciated by Pariset† in 1811. Since then this idea, developed on many occasions by Rochoux himself, has been adopted by a good number of pathologists. It has been modified by M. Durand-Fardel,‡ and by Todd;§ and, without speaking of the work of M. Cruveilhier‖ on the identity of the nature of capillary apoplexy and softening, modern researches into the hyperæmia which frequently accompanies cerebral necrobiosis, have appeared to renew this opinion. Whatever view may be taken as to the extent to which it should be received, this idea deserves at least to be discussed. Of all the authors who have written during late years on the pathology of the brain, Leubuscher¶ appears to me to be the only one who has failed to appreciate the influence which a preparatory softening might have in the production of hæmorrhage.

Rochoux in his first work expresses himself in the following terms:**—"The walls of apoplectic cavities are surrounded by a layer of cerebral matter from one to three lines in thickness, of a canary-yellow colour, pale, very soft, scarcely of

* Recherches sur l'Apoplexie, 1814. P. 88.

† Journal de l'Empire, 7 Février, 1814.

‡ Traité Clinique et Pratique des Maladies des Vieillards, 1854. P. 294.

§ Clinical Lectures on Paralysis, p. 126.

‖ Anatomie Pathologique du Corps Humain, livr. xxxiii.

¶ Die Patholog. und Therap. der Gehirnkr.

** Loc. cit.

better consistence than some creams, and but slightly miscible with water." And further on he says, "So far as I am aware, such a change has not been described by any one. Is it simply the effect of the continued presence of the blood, or does it precede the hæmorrhage? The latter opinion seems to me the more probable."

Such a hypothesis, thus presented, and quite devoid of proofs, was not readily accepted.

Moreover, what was this so-called softening? Rochoux never said. In the article *Apoplexy* in the "Dictionaire de Médecine," in which his opinion was developed in 1833,* he says that this pre-hæmorrhagic softening (*ramolissement hémorrhagipare*), the real nature of which is unknown, is different from the softening described by Lallemand and by Rostan. He had not met with it in cases in which hæmorrhage had not already occurred.

We need not, then, be surprised that this cause has been doubted or denied altogether by most pathologists. M. Gendrin,† who believed that he had discovered the source of the hæmorrhage in the rupture of numerous arterioles, whose existence he points out on the surface of the apoplectic cavities, and whose torn extremities were involved in the substance of the clot, thought that that was a primary lesion, and concluded that softening as a cause of hæmorrhage had no real existence. M. Durand-Fardel,‡ without expressing himself in so decided a manner, has at least denied its influence in the production of the majority of cases of hæmorrhage; while Hasse§ denies not only its influence, but even its existence.

And yet, if we peruse the numerous works which have appeared in late times on the subject of ischemic softening of the brain, and in which a description is given of the singular phenomena of hyperæmia which frequently occur in these cases, and from which result capillary apoplexy, red softening, and even extravasations forming more or less considerable

* Dictionaire de Médecine, t. iii. 1833. P. 486.

† Traité Philosophique de Médecine Pratique, p. 481.

‡ Traité Clinique et Pratique des Maladies des Vieillards, p. 394.

§ Handb. der spec. Path. und Therap., bd. iv. p. 384.

cavities, we see that the authors of these works refer to these softenings as being those which Rochoux had already described. The name *ramolissement hémorrhagipare* of Rochoux has been revived, but diverted from its true meaning. As understood by the physician of Bicêtre, it indicated an error; to-day it is applied to facts which are real, but which Rochoux himself was careful to point out bore no relation to the new name which he proposed.

Instead of the pre-hæmorrhagic softening imagined by Rochoux, M. Durand-Fardel* has endeavoured to find the cause of most cerebral hæmorrhages, at least of those which occur in old age, in a different change of the brain tissue. He has described a condition of interstitial atrophy of the brain—a state characterised, according to him, by a large number of very small cavities, which is not to be confounded with cellular infiltration, and which differs also from the cribriform state, but may coincide with it. No very exact account of what M. Durand-Fardel intended to describe has ever been given, and that condition which, according to him, is frequently met with either around recent hæmorrhagic cavities, or in brains in which hæmorrhage, though imminent, has not yet taken place, is still involved in doubt and obscurity. I have never been able positively to verify it, and Hasse† denies its existence altogether.

According to Todd‡ almost all hæmorrhages occur in the substance of a patch of softening, and on this theoretical view he has based some very ingenious clinical deductions. It is thus he thinks that hemiplegia remains simple, without being complicated with rigidity or contraction so long as the blood remains within the limits of the softened portion. But if the blood breaks through the thickness of the softened bed which primarily surrounded it, and lacerates the healthy cerebral tissue, then appear the phenomena of spasm, indicative of the irritation caused by the blood in the tissue of the brain. But what is this softening? Is it an encephalitis? Or is it, on the other hand, the true softening with which we are familiar since the labours of Rostan and Abercrombie? Todd tells us that

* Traité Clinique et Pratique des Maladies des Vieillards, p. 295

† Loc. cit. p. 384. ‡ Loc. cit. p. 126.

it is accompanied by no appreciable change either in the colour or structure of the cerebral tissue! From this we might conclude that it is a softening which does not exist.

On what, then, is based the hypothesis which has induced some authors to admit the existence of this so-called pre-hæmorrhagic softening? It rests on the difficulty which is often experienced in finding out the proximate cause of cerebral hæmorrhage, and on the fact that softening is very frequently found in the walls of apoplectic cavities. But this softening, which may be produced by many causes, is secondary to the sanguineous effusion. This opinion, which I shall presently try to establish, has already been opposed by Rochoux.* "If," he says, "we can admit the influence of imbibition when the disease has lasted four or five days, we cannot do the same for cases in which death supervenes in a few hours, and in which yellow softening is found with all its characteristic appearances." It might be objected to this view of the matter, that if the softening could not be produced during the few hours which elapse between the occurrence of the hæmorrhage and death, that effect might be produced by the same cause and in a more marked manner during the period which intervenes between death and the autopsy. The objection may also be raised that it is not uncommon to find the walls of the cavity of normal consistence in cases of very recent apoplexy, provided always that the effusion is inconsiderable, and completely coagulated into a non-diffluent clot. Indeed, without even appealing again to the result of histological examination, I would remark that one of my cases proves in a decided manner that it is the imbibition of the serous part of the effused blood which modifies the external characters of the cerebral tissue around the sanguineous cavities. The case is that of a woman who died in M. Vulpian's wards after repeated attacks of cerebral hæmorrhage coming on suddenly during an attack of jaundice. All the tissues were more or less coloured by bile. But this coloration was most marked in the substance of the brain, around each of the apoplectic cavities to the depth of about half a centimetre. The cerebral tissue at these points was likewise diminished in consistence,

* Dictionaire de Médecine, t. iii. p. 485.

but presented no histological changes. It would be difficult in this case not to attribute the coloration of the cerebral tissue around the cavities to the imbibition of the serum of the effused blood.

Imbibition, then, appears to me to play an important part in the production of that diffluent condition of the cerebral pulp which is found around apoplectic effusions. When the effusion is quite recent microscopic examination fails to discover any change of structure. The small vessels only show some peculiarities to which I shall presently refer, and which are the result and not the cause of the rupture. If, on the other hand, we had to deal with hæmorrhage occurring in the substance of true softenings, and not of these supposititious softenings of Rochoux and of Todd, we ought to find, to say nothing of the cause of this softening (vascular obliteration), a more or less advanced state of necrobiosis of the cerebral tissue in the part in which the effusion had occurred. It cannot, indeed, be maintained that the interference with the nourishment of the brain from which the softening results, is capable of diminishing the consistence of the cerebral tissue to such an extent as to produce rupture of the vessels before the products of the retrograde change have become apparent. We know, indeed, that in softening fatty granules appear in the affected part within the first twenty-four hours; and that granular bodies and an atheromatous appearance of the small vessels may be met with in the course of the second day. If the histological changes do not precede diminution of consistence, they accompany, or at least follow it very closely. Now, neither granular bodies nor fatty granules are found in those softenings which surround hæmorrhagic cavities when death takes place one or two days after the attack. It is even rare to find them, and they are never met with but in very small numbers, when death supervenes during the first four or five days. But at last they appear, and the tissue then presents a much more marked degree of diffluence. It appears to me that this necrobiotic change which is thus secondarily developed ought to be regarded as the result of a secondary degeneration, which transforms into fatty granules the portion of the tubes which the rent in the cerebral tissue had separated from their

trophic centres. This degeneration, as we know, may be traced far from the primary lesion, even into the medulla.

To these two causes, imbibition of the serous portion of the blood and retrograde destruction of the torn nerve tubes, there ought, perhaps, to be added a third which was apparent in one of the cases which I have collected. I allude to a sort of inflammation which springs up in the torn portions of cerebral pulp, and which, in the case to which I have referred, was characterised by a very remarkable multiplication of the neuclear elements which in the normal condition are found scattered in the white tissue, and which appear to be essential parts of the connective tissue of the nervous centres.

I believe that I have shown that the softening which is frequently found around hæmorrhagic cavities is secondary, that it results chiefly from the imbibition of the serum of the blood, but is also due in part to atrophic degeneration of the torn nerve tubes, and sometimes also to slight inflammation of the lacerated cerebral tissue ; it does not, therefore, merit the name of *pre-hæmorrhagic* softening. I come now to the consideration of vascular ruptures occurring in softening properly so-called—that which results from necrobiosis of the tissue of the brain consequent on interruption of the local circulation.

We know how commonly there is found after obstruction of one of the cerebral arteries a punctated or uniform red coloration of the parts of the brain which correspond to the divisions of the obliterated vessel, and which become the seat of those retrograde changes which are characteristic of softening. Red softening appears, indeed, to be more frequent than white softening, or that in which the normal colour is preserved; this red coloration is due not only to the accumulation of the blood in the dilated capillaries, but results also from more or less complete extravasations of that fluid. The causes of this hyperæmia and of these vascular ruptures are difficult to estimate, and have recently been the subject of both critical works * and experimental studies. † Some points appear to

* V. Proust. "Des différentes formes de Ramolissement du Cerveau." Paris, 1866. P. 39. Poumeau. "Du Rôle de l'Inflammation dans le Ramolissement Cérébral." Paris, 1866. P. 131.

† Prevost et Cotard. Etudes Physiologique et Pathologiques sur le Ramolissement Cérébral. Paris, 1866. P. 38.

have had considerable light thrown upon them by these researches, but as a whole the question still remains very obscure.

In these hyperæmias and extravasations it is necessary to distinguish those which occur around, from those which are found in the depth of the softened patch. The hæmorrhagic dotting (*pointillé hémorrhagique*) of the periphery, which Oppolzer believed to be due to a sort of eliminatory inflammation, has, on the other hand, been attributed by Rokitansky to increased pressure of the blood, which is driven through the collateral circulation into those vessels which arise beyond the point of obstruction. This interpretation, altogether mechanical, which is supported by Meissner * and O. Weber, † appears to be justified by the laws of hydrodynamics, and by the observations of MM. Prevost and Cotard. ‡ The hyperæmias and hæmorrhages which are so frequently found in the substance of the part to which the branches of the obliterated trunk are distributed, are much more difficult of explanation; they are in all cases real, and do not differ from what is observed in infarctus of other organs,—the spleen or the kidneys, for example,—when some arterial branch of these viscera is obstructed by a clot, by a foreign body introduced into the circulation experimentally, or even by a ligature. We cannot plead here collateral flow; nor can the surmise of Virchow, that they are due to venous reflux consequent on suppression of the *vis a tergo*, be accepted as satisfactory. The almost immediate production of these dilatations and ruptures after the obstruction of the artery scarcely permits of their being attributed to a change commencing in the structure of the small vessels. Finally, local asphyxia, paralysis of the muscular coat under the influence of blood, which loses by stagnation its normal qualities, is no more than a pure hypothesis.§ I shall not venture on a new explanation, but shall confine myself to

* "Zur Lehre, von der Thrombose und Embolie" (Schmidt's Jahrbücher, 1861, No. 1). Meissner believes that the collateral flow is accompanied by dilatation, then by altered nutrition of the vessel, and finally by its rupture.

† Handbuch des Allgemeinen und speciellen Chirurgie, von Pitha und Billroth. Erlangen, 1865.

‡ Etudes Physiologiques et Pathologiques sur le Ramolissement Cérébral, 1866. P. 40.

§ Poumeau, loc. cit.

the observation that hæmorrhage may occur during the progress of the softening, and that it results from the altered state of the cerebral tissue, or from the change in the circulation consequent on the obliteration.

The hæmorrhage which accompanies softening is generally very limited in extent, and rarely produces true hæmorrhagic cavities. More extensive extravasation occurs, however, in some cases, especially in those in which the softening results from obstruction of the sinuses. But then the obstacle to the return of the blood suffices to explain the phenomenon. In most cases the hæmorrhage consists only in the intimate mingling of the blood with the nervous tissue,—this is red softening properly so called; or in a punctated appearance, which is called capillary apoplexy, and which is really only a minor degree of the preceding condition.

Before entering on the study of capillary apoplexy, on which I must dwell at some length, I shall relate a case of red softening with considerable hæmorrhage, consequent on obliteration of the veins of the pia mater. The case was communicated to me by M. Charcot.

CASE I.—*Obliteration of some of the veins of the pia mater. Red softening, with cerebral and meningeal hæmorrhage.*

A female, aged 65, had pneumonia in 1858, and again in 1863.

On the 16th June 1865 she was admitted into hospital a third time. For three days she had been suffering from sickness and a sharp pain in the right side and breast, and extending to the right shoulder. Auscultation revealed nothing abnormal; temperature in rectum, 39°½.

August 10*th.*—For some time the patient has complained of very acute pain in the right side of the head, in the forehead and occiput, but not in the face. The abdomen is somewhat large: there appears to be a little ascites. The urine is very high coloured; contains neither sugar nor albumen.

August 21*st.*—Abdomen considerably distended; patient has very anxious expression; pulse very quick; skin hot; no stool for two days. At evening visit I learned that she had had a seizure, and found her paralysed on the right side. Sensibility

and reflex movements were awanting on the paralysed side, but were normal on the left, the limbs of which she moved very well. Patient was quite intelligent, answered questions pretty well, but with a slight embarrassment of speech. There was considerable œdema of the right leg; temperature in rectum, $38°\frac{2}{5}$.

August 23*d*.—At two o'clock this morning she became comatose; at time of visit the breathing was stertorous, the pulse small and frequent, the skin warm. The limbs were all flaccid. Reflex movements were abolished on both sides. Died at 4 P.M.

Autopsy.—The superior longitudinal sinus was adherent to the vault of the cranium. The walls of the sinus were thickened, and a small quantity of blackish blood was found in its interior. There were no false membranes on the inner aspect of the dura mater. There was sub-arachnoid hæmorrhage over almost the whole extent of both hemispheres, but more abundant on the right than on the left. Both lateral ventricles were filled with dark-coloured, very friable clots. There was yellow softening of the whole of the floor of the left lateral ventricle. The optic thalamus of the same side presented on its surface a reddish pulp formed by a mixture of blood and softened cerebral tissue. On the left hemisphere, behind the convolution which bounds the fissure of Rolando, was found a patch of red softening. The grey matter was of the colour of wine lees, and the subjacent white substance was softened and slightly yellowish. On the right hemisphere, behind the fissure of Rolando, was found a new patch of red softening not quite so extensive as the preceding. The arteries of the base were not atheromatous. On the surface of the clots which covered the inner aspect of both hemispheres were seen stretched two veins which emptied themselves into the superior longitudinal sinus; they were of a yellowish colour, and contained old clots.

There was fatty deposit at the base and in the interventricular septum of the heart; no lesion of the orifices.

The peritoneum contained a large quantity of yellowish serum; peritoneum itself healthy; liver small, yellowish and granular in appearance; examined under the microscope it was

seen to be the seat of fatty degeneration. With the exception of a small cyst in the right kidney the other abdominal organs were healthy.

Let us now return to the study of capillary apoplexy, which merits attention for a little. It is not a disease, but an anatomical condition common to various diseases; not a special form of hæmorrhage, but a peculiar disposition of the extravasated blood, no matter how the extravasation may have been produced; and if I insist on this, it is because the anatomical condition which characterises and constitutes capillary apoplexy has been described, not as a result, but as a cause of the hæmorrhage.

As contrary doctrines appear to be very generally held, it might be well to premise the consideration of these propositions by certain details. The opinion expressed by M. Cruveilhier, in his "Anatomie Pathologique du Corps Humain," regarding the identity of capillary apoplexy and red softening, was reproduced by him* in 1862; and quite recently in the last edition of Valleix, M. Lorain,† in accepting this view, concludes as follows: "In truth, hæmorrhage and softening of the brain are two modifications of the same pathological state, that is to say, that they are governed by similar pathological conditions." Dance,‡ and afterwards M. Diday,§ held an opinion diametrically opposed to this, and regarded apoplexy as a disease *per se*, having no connection with softening. M. Fleury,‖ in a critical article on capillary apoplexy, appears to me to have been nearer the truth, when, in his conclusions, he says that "nothing warrants us in according to sanguineous infiltration the same individuality and distinct existence (*individualité et essentialité*) which we accord to senile softening, for instance."

* Traité d'Anatomie Pathologique Générale, 1862, t. iv. p. 214.

† Guide du Médecin Praticien, 1866, t. ii. p. 86.

‡ Archives Générales de Médecine 1832, t. xxviii. p. 325.

§ Mémoire sur l'Apoplexie Capillaire (Gazette Médicale de Paris, 1837, t. v. p. 242).

‖ What place in nosology should be accorded to the cerebral change described under the names—capillary apoplexy, capillary hæmorrhage, sanguineous infiltration of the brain?—*Journal de Médecine de Beau*, t. 1er, 1843. P. 121.

If we examine the nervous tissue which surrounds a patch of recent red softening, or that which forms the walls of an apoplectic effusion into the corpora striata or optic thalami, we find in both cases a red dotting which is characteristic of capillary apoplexy. If we tear the cerebral tissue with needles close to these red points, we can generally satisfy ourselves that they correspond to the section of a vessel, which may be followed and isolated for a certain distance. If the vessel thus isolated be placed under the microscope, it is seen that the blood which fills it is enveloped only by a very thin structureless membrane, having none of the structural appearance of the vascular walls. But with a little care the vessel is soon found in the interior of this tubular membrane, either perfectly healthy or variously altered. Rupture of this vessel, somehow produced, has caused extravasation, and the blood has filled the lymphatic sheath which limits the effusion externally. We know that the arteries of the brain are surrounded by an extra coat which is not adherent to their substance, but is separated from them by a space more or less considerable, filled with a colourless fluid, and that this tunic ramifies with the arteries, accompanying them in all their divisions up to the point at which they terminate in the capillaries properly so called.* In capillary apoplexy this space, in which the arteries float freely, is filled to a greater or less extent by the effused blood. In some cases this arrangement may be seen even with the naked eye in the large sized vessels which ramify on the walls of the lateral ventricles. This injection of the lymphatic sheath may so distend the bounding membrane that it gives way; small cavities may then be produced in immediate contact, and sometimes intimately mingled with the cerebral substance. Capillary apoplexy may then

* The perivascular system, here referred to, was discovered by M. Robin in 1855. Its existence forms a most important element in the pathological investigation of diseases of the brain, and has, at least until very lately, scarcely had bestowed on it in this country the attention which it certainly merits. Recent researches seem to indicate that it is not confined to the vessels of the brain, but exists also around the vessels in other parts of the body.

See Bastian's translation of His's paper in the "Journal of Anatomy and Physiology" for 1867; and an important paper by Mr Wagstaffe in St Thomas' Hospital Reports, vol. ii., 1871. T. J. M.

precede true hæmorrhage; it is the first stage of an effusion which ultimately forms a true hæmorrhagic cavity. The blood which is effused into that sheath results from the rupture of the vessels contained in it; that rupture may be the result either of increased tension or altered nutrition, as may be the case in cerebral softening; or it may be due to tearing and laceration of the vessel, such as occurs when cerebral hæmorrhage consequent on the rupture of a large vessel lacerates the brain tissue, and with it the small arteries which it contains. It is evident that capillary apoplexy has no independent existence, that it accompanies red softening as well as the cavities of sanguineous apoplexy, and that, on the other hand, it may, in some cases, be the first stage of a true cerebral hæmorrhage.

Let us now return to the study of the question which forms the chief object of this chapter. It appears to me that neither the softenings of Rochoux and of Todd, nor the interstitial atrophy of M. Durand-Fardel, should be regarded as causes of cerebral hæmorrhage; and, moreover, it does not seem to have been shown that the sanguineous extravasations which accompany softening at its outset result from diminished consistence of the surrounding tissue. It remains for us to inquire whether old softening, by the diminished support which the diseased tissue affords to the vessels which run through it, may lead to secondary ruptures, to hæmorrhage occurring later on, forming a sanguineous cavity in the substance of a patch of softening.

Hæmorrhage into the interior of an old softening may indeed occur; but it is extremely rare, and when we find, what is not very frequently the case, cerebral hæmorrhage developed in an individual in whom there already existed patches of former softening, the sanguineous effusion is generally found in other localities than those in which the necrobiotic change has taken place. The existence of softening appears then to exercise no influence in determining the seat of an ulterior hæmorrhage; and if such influence did exist, the question would still remain for inquiry, whether it is the diminished support of the surrounding tissue, or change in the vessels consequent on the softening, which favours the rupture. Be that as it may, I shall relate two cases in which

extravasation occurred in a pre-existing softening. One was observed by Pestalozzi, the other was communicated to me by M. Charcot.

Pestalozzi's case* was that of a female lunatic, aged 51, Ursule Hammer by name, in whom there was found after death a cellular, gelatiniform softening (*plaque jaune*) of the convolutions of the middle portion of the right hemisphere, and in the substance of which the presence of some recently extravasated blood was demonstrated. This woman presented at the same time a red softening of the right corpus striatum. In the internal carotid artery of the right side were found some stratified deposits; the arteries of the upper part of the body were atheromatous, and the spleen was the seat of recent red infarctus. The patient had presented some symptoms of heart disease, but the autopsy showed no valvular lesion.

Microscopic examination demonstrated the existence of *dissecting aneurisms* in the recent extravasations of the old softening, as well as in the substance of the recent red softening. As we shall see in the next chapter, the dissecting aneurisms of Pestalozzi are nothing more than that hæmorrhage into the lymphatic sheath which has been described above as constituting capillary hæmorrhage. At the time when Pestalozzi wrote, the existence of the lymphatic sheath was not known, and the presence of blood between the vessel and this sheath appeared to be the result of the detachment of the adventitious membrane, such as is observed in dissecting aneurisms of the large arteries. Moreover, the description given by Pestalozzi of the so-called aneurisms observed in this case, leave no doubt as to the correctness of the interpretation which I offer. He says, in fact, that the largest of these arteries simulated elongated clots surrounded by the adventitious membrane, and that by pressure and the action of water the vessel could be seen reduced to its two internal coats, and, without the least dilatation, floating in the distended cavity of the adventitious membrane. He adds that when the adventitious tunic was stretched more than a line, or a line and a half, it gave way, and that the clot was then no longer en-

* Ueber Anevrysmata spuria der kleinen Gehirnarterien und ihren Zusammenhang mit Apoplexie. Wurzburg, 1849. P. 13.

veloped by a membrane. He says again, and this is another argument in support of my view, that in the vessels of the old softening the granular bodies occupied the same situation between the middle and adventitious tunics. Now, I believe that I have shown* that these granular bodies are situated precisely in the lymphatic sheath.

On the whole, this case of Pestalozzi's, which demonstrates the existence of hæmorrhage in a patch of softening, proves in addition that the effusion of blood into the lymphatic sheath, which constitutes capillary apoplexy, is also observed in hæmorrhage and in red softening.

CASE II. — *Cerebral softening with numerous cavities. Secondary cerebral hæmorrhage into one of the softened patches. Escape of effused blood into the arachnoid cavity. Epileptiform convulsions.*

A female, aged 59, was admitted, under the care of M. Charcot, on 30th May 1862.

About a couple of months previously she had been found unconscious in bed. When admitted she lay on her back; could not stand; presented symptoms of paralysis with contraction, chiefly on the right side, and was '*complétement gâteuse.*'

On the 26th August she was suddenly seized with epileptiform convulsions, complete loss of consciousness, and stertorous breathing. The convulsions continued almost uninterruptedly for ten hours, when death occurred.

Autopsy.—Encephalon: the cavity of the arachnoid on the left side was found to contain a black rounded clot, evidently compressing the surface of the corresponding posterior lobe of the brain. This clot penetrated to the interior of a large cavity, which occupied almost the whole extent of the posterior lobe. This cerebral cavity communicated with the cavity of the arachnoid by means of a jagged opening of the size of a franc, surrounded by torn and soft edges. Neither the parietal nor visceral arachnoid presented any trace of false membranes in

* "Des Dégénérations Secondaires de la Moelle épinière" (Arch. Gén. de Méd., Mars 1866). See also Proust. "Des différentes formes du Ramollissement du Cerveau," p. 59; and Poumeau, du "Rôle de l'Infl. dans le Ram. Cérèbr." p. 25.

the neighbourhood of the sanguineous cavity. The entire clot, that is to say, both the extra-cerebral and intra-cerebral portion, was about the size of a hen's egg. About one-third of the whole clot occupied the arachnoid cavity; the remainder was contained in the abnormal cavity to which reference has been made. The walls of this cavity were softened throughout almost their whole thickness, to such an extent that the whole posterior lobe might be regarded as the seat of a softening, which extended to the very surface of this lobe. This surface, moreover, presented throughout its whole extent, but especially in the neighbourhood of the fissure of Sylvius, a yellow coloration in keeping with the existence of softening. The lateral ventricle did not appear to communicate with the softened patch. The softening, then, involved only the extra-ventricular portion of the posterior lobe. The corresponding optic thalamus and corpus striatum were quite distinct. They appeared to be comparatively healthy; yet without being actually the seat of softening, they were of very soft consistence. The corpus callosum and fornix were likewise soft.

All the parts of the anterior lobe situated in front of the fissure of Rolando were comparatively healthy in appearance. But on raising the pia mater, that membrane carried along with it fragments of the cortical layer of cerebral tissue. This cortical substance presented, moreover, throughout its whole extent a dark tinge; besides, here and there, at different points, were observed light red spots, which corresponded to the points of superficial softening. At these points the softened portions were readily removed by a jet of water.

The central white substance of the anterior lobe appeared almost healthy. The lobule of the insula was likewise healthy,

The right lobe presented many points of yellow or white softening either on its surface, or in the depth of the posterior lobe. The corpus striatum of the right side, and the surrounding cortical substance, at the junction of the anterior with the two posterior thirds of the corpus striatum, presented a hard linear cicatrix surrounded by softened portions of an ochrey tint, and altogether resembling old cysts, the remains of hæmorrhagic cavities. The anterior lobe was healthy, though generally soft; the lobule of the insula was likewise

healthy. The pons Varolii, the cerebellum, and the medulla oblongata were somewhat soft.

The basilar artery was three or four times its natural size: it was tortuous, and indurated with some hard patches. The arteries raised along with the pia mater, when examined afterwards, presented at many parts of their calibre thrombi about a centimetre in length, hard, in a great measure colourless, adhering pretty closely to the walls, and distending the vessels; these thrombi were situated in vessels a little larger than a crow quill. The name of the obliterated vessel could not be given, as the examination was made after the vessels, detached from the brain with the membranes, had lost their connections.

The arteries, in general indurated, presented here and there small atheromatous patches.

To sum up, the pre-hæmorrhagic softening of Rochoux and of Todd appear to me to be pure hypotheses: as to the interstitial atrophy of M. Durand-Fardel, its influence, and even its existence, appear to me to be only problematical. The proximate causes of the extravasations which occur in softening, due at its outset to deficient blood supply, are yet unknown to us, but these extravasations appear to occur too early to permit of our attributing them to diminished consistence of the softening tissue. Indeed, old cerebral softening does not appear to me to dispose to hæmorrhage in the affected part; and when, in exceptional cases, such hæmorrhage does occur, it may just as well be attributed to change in the vessels as to diminished support of the surrounding tissue.

Increased tension of the blood, the pathological signification of which I endeavoured to estimate in the first chapter, appears to me to be of but secondary importance in the production of cerebral hæmorrhage, and, except in some very rare instances, to intervene only as an aiding cause. The causes which we have just considered in this chapter appear to me to have only a very doubtful influence; it remains for us to estimate the value of the different lesions of the vessels of the brain, to which alone we must now look for an explanation of cerebral hæmorrhage.

CHAPTER III.

ON THE CHANGES IN THE VESSELS OF THE ENCEPHALON TO WHICH THE PRODUCTION OF CEREBRAL HÆMORRHAGE HAS BEEN ATTRIBUTED.

BEFORE entering on the study of these anatomical changes in the vessels of the brain which appear to diminish the resistance offered to the lateral pressure of the blood, I must say a few words regarding the vascular weakness, and especially the friability of the capillaries, which appears to exist in some cases in which, even with the aid of the microscope, nothing abnormal can be detected in their walls. This condition has been regarded as a possible explanation of those very rare cases of sanguineous apoplexy in which young people are suddenly struck down in the midst of perfect health, under the influence of an exaggerated cardiac impulse, or an intense congestion, no matter how produced. This condition, very hypothetical though it be, may be congenital. Rokitansky admits the frequency of weakness of the vessels in young women. Virchow,* who has reproduced the opinion of Rokitansky, refers also to weakness of the capillaries in newly-born children. This defective resistance may be observed in a general way in all young vessels, whether developed physiologically or pathologically. Virchow, besides, is not aware how long this weakness of young vessels in process of development may last. A similar state of friabilty of the capillaries, and more particularly of the small vessels of the brain, appears capable of being developed in the adult, under the influence of certain morbid conditions in which some change takes place in the blood. This vitiated nutrition of the vessels, which declares itself only by diminished resistance, without appreciable histological

* Handbuch der speciellen Pathologie und Therapie, band i. P. 240.

change, is, according to Virchow, Leubuscher,* and Niemeyer,† the cause of those very rare cases of cerebral hæmorrhage which occur in certain fevers, and in septic and infectious diseases, such as typhus, pyæmia, scurvy, and certain chronic 'dyscrasiæ.' May not the supposed changes in the blood, which have been attributed to retention of the menses, or to the accumulation in that fluid of extractive or other materials, derived from the liver, spleen, or kidneys, be also the cause of like changes in the vessels of the brain?

It is impossible to reply, even doubtfully, to that question. The cerebral hæmorrhage which may occur in the course of albuminuria is the only form which has been studied from this point of view, and we shall presently see that the greatest uncertainty prevails regarding even that subject. Of the sanguineous apoplexy which may occur in jaundice, I have only one instance, which shall be reported farther on; it will be seen that the hæmorrhage was due to vascular changes which had been going on for a long time, and which led to rupture on the occasion of some disturbance of the circulation, or some congestive accidents presented by the patient, rather than to deranged nutrition consequent on the action of blood charged with the materials of the bile.

In hæmophilia, which appears to present in a high degree this condition of congenital friability of the vessels, hæmorrhage is very rare: Virchow‡ relates only one case, and that is a doubtful one. It is not so in purpura. Cerebral hæmorrhage has also been observed in chlorosis, but, as we shall presently see, this accident appears to be connected with an appreciable change in the capillaries. M. Duchassaing,§ in a work in which he points out this complication of chlorosis, refers also to apoplexy occurring in scorbutic old women who came under his notice in the Salpêtrière. It is necessary to guard against these so-called scurvies of old people, and especially to be precise regarding the nature of the cerebral lesion which is disclosed

* Die Pathol. und Therap. der Gehirn-krankh. p. 222.

† Eléments de pathologie interne et de thérapie (édition Française), t. ii. p. 201.

‡ *Loc. cit.*

§ Mémoire sur certaines affections Cérébrales qui dépendent de la Chloro-anémie (Journal de Médecine de Beau, t. 1er, 1843, p. 353).

by the apoplexy. It is quite common among the old women at the Salpêtrière to see petechial spots on the limbs having the appearance of scurvy; these women are also frequently seized with apoplexy. But these scorbutic spots are nothing more than infarctus of the skin and subcutaneous cellular tissue, and the apoplexy results from a softening connected, like the cutaneous affection, with a very marked atheromatous change of the arterial system. *L'état scorbutique* of MM. Becquerel and Rodier, which is characterised by the lowering of the total quantity of the fibrine below 2 per 1000, may also, according to Virchow, produce this change in the nutrition of the capillaries. The same author also refers to a change of the blood, characterised by a gelatinous transformation of the fibrine, as capable of leading to the same result, but adds, with reason, that nothing positive is known on that subject.

How is cerebral hæmorrhage brought about in albuminuria? Without reckoning weakness of the small vessels, which is certainly a very convenient explanation, it is necessary in this case to allow for other lesions. It is not uncommon to find hypertrophy of the left ventricle, with or without valvular lesions accompanying Bright's disease. This hypertrophy is secondary according to Traube,* primary according to Bamberger.† On the other hand Bright, Gregory, and Rayer have pointed out the frequent occurrence of atheromatous change in the large arterial trunks in those suffering from albuminuria. There are two opinions regarding the part played by these changes in the production of the cerebral hæmorrhage which sometimes occurs in such cases. S. Kirkes,‡ as well as Traube, thought that the atrophy of the kidneys impeded the circulation through these organs, that the diminution of the urinary secretion produced a sort of repletion of the arterial system, that the heart became hypertrophied in its endeavours to overcome this resistance, and that the hypertrophy of the ventricle increased the tension of the vessels of the brain to such an extent as to

* Ueber den Zusammenhang von Herz und Nierenkrankheiten. Berlin 1856.

† Ueber die Beziehungen zwischen morbus Brightii und Herzkrankheiten (Virchow's Archiv, band xi. Janv. 1857. P. 12).

‡ Medical Times and Gazette, 1855.

cause rupture. Barlow * gives quite a different explanation; he supposes that Bright's disease alters the blood, which irritates the lining membrane of the heart and vessels, especially those of the brain, causing them to thicken and retract, and that apoplexy may result from these lesions by extravasation, or by sudden arrest of the circulation. This last opinion does not seem very probable, though it is, perhaps, more so than that of Todd, who, admitting poisoning of the blood, thought that this blood vitiated the nutrition of the brain, and that the hæmorrhage resulted from a sort of pre-hæmorrhagic softening. In a question so obscure I shall not try to choose between three interpretations, no one of which appears satisfactory.†

* A Manual of the Practice of Medicine. London, 1856. P. 486.

† Dr George Johnson has shown that the cause of the obstruction to the flow of blood through the minute arteries is hypertrophy of their muscular walls. These vessels, in the performance of their proper duties as regulators of the blood supply, resist the entrance of the blood into the kidneys in proportion to the destruction of the secreting cells. In time they become permanently hypertrophied. With the advance of the renal disease the blood becomes more impure, and the minute arteries of the whole system are excited to increased contraction. This increased obstruction to the flow of blood demands a corresponding increase in the cardiac force, which results in hypertrophy of the heart. From this again results increased tension of the blood in the vessels situated on the cardiac side of the point at which this "stopcock" action of the hypertrophied arteries is produced. As a consequence of this strain a vessel may give way and hæmorrhage result.

It is doubtful if such an accident could occur if the vessels were healthy; and it is rendered probable by the observations of MM. Charcot and Bouchard, that in cases in which the cerebral hæmorrhage is apparently due to this increased cardiac effort, the accident ought chiefly to be attributed to the existence of diffuse peri-arteritis with miliary aneurisms,—increased blood tension occupying only a secondary place in its somewhat complex etiology.

The tendency of the present day is to regard chronic renal disease not as an isolated lesion, but rather as a local manifestation of a constitutional disease, of which possibly the vascular changes may be only another sign; this view is strengthened by the fact that, along with these changes in the kidneys and arteries, there is often found an altered condition of other organs, the lungs and liver for example.

The whole subject of the influence of increased tension of the blood in producing rupture of the vessels must henceforth be studied in the light thrown upon it by Dr Quain's observations on hypertrophy of the heart (see foot note at page 7). It may possibly be found that the enlargement of that organ which so frequently accompanies renal disease is not true hypertrophy of the muscular substance, but is caused partly, if not wholly, by an increase

I approach now the study of the appreciable changes in the structure of the vessels of the brain which may diminish their resistance, and lead to the production of cerebral hæmorrhage. First, I shall consider that which is connected with fatty change of the capillaries and small vessels.

Paget,* in 1850, first pointed out fatty change in the capil-

in the connective tissue,—that it is, in fact, a sort of cirrhosis of the heart, resulting, possibly, from the same cause which gives rise to the renal disease, the arterial degeneration, the pulmonary emphysema, and the cirrhosis of the liver which are occasionally all found in the same subject.

Since the above was written, Sir Wm. Gull and Dr Sutton, in a communication read to the Medico-Chirurgical Society of London, have advocated the view that chronic Bright's disease with contracted kidney is a general disease, and that other organs may suffer before there is any evidence of the kidneys being affected. They specially regard hypertrophy of the walls of the vessels as the most characteristic of the local lesions which occur; they hold, however, that the hypertrophy takes place, not in the muscular coat, but external to it. The thickening, as described by them, is due to the presence of a "hyalin-fibroid" formation in the outer coat of the vessel; this change, they say, occurs in the minute arteries of all parts of the body. Dr Johnson, however, maintains that what Sir Wm. Gull and Dr Sutton regard as a pathological change in the tunica adventitia, is really a post-mortem change resulting from the mechanical distension of that tunic by endosmotic imbibition of the fluid in which the sections of the vessels were placed for microscopic examination.

As regards the production of cardiac hypertrophy, it does not much matter whether the obstruction to the flow of blood through the minute arteries is caused by thickening of the middle or outer coats, if only the calibre of the vessels be equally diminished in both cases; but as regards the more general question of the mutual relation of the renal disease and the vascular changes, and the connection of both with a general disease of the system, the distinction is one of the utmost importance. Dr Johnson's theory is so beautifully simple, and is so in keeping with what we know of the physiological action of the minute arteries, that it is not likely to be hastily abandoned. Whether that of Sir Wm. Gull and Dr Sutton is founded on accurately observed facts, or on such an error as Dr Johnson indicates, remains for further research to show; meantime, there can be no doubt that their communication forms an important addition to the literature of the whole question of renal disease and its connection with pathological changes in other organs. It is very possible that neither theory may admit of general acceptation to the total exclusion of the other. If the condition described by Sir Wm. Gull and Dr Sutton is proved to exist, one cannot fail to see a possible connection between their "hyalin-fibroid" changes in the adventitia, and the chronic peri-arteritis of Charcot and Bouchard. T. J. M.

* On fatty degeneration of the small blood-vessels of the brain, and its relation to apoplexy (London Medical Gazette, vol. x. p. 229).

leries and small arteries of the brain, and its connection with apoplexy Though M. Robin* had already, in the preceding year, called attention to fatty degeneration of the arterial system, and especially of the small arteries of the brain, yet he did not indicate this as a cause of hæmorrhage. That opinion, however, has been attributed to him, and has been connected with his name by almost all French authors who have studied the vascular changes which occur in cerebral diseases. Since that time numerous works have been published on this subject; but in most of them there is a confusion, which is to be regretted, between *primary* and *secondary* changes,—two great classes of facts which are essentially distinct, and which must be clearly separated if we would avoid coming to erroneous conclusions in studying the pathology of many different affections of the nervous centres. An inaccurate knowledge of the normal anatomy of the small vessels of the brain has been the chief cause of the confusion to which I refer, and the uncertainty which has long prevailed regarding the seat of the fatty granules, which, according to some, are situated outside the vessel, and according to others in the substance of its walls. These two apparently contradictory opinions, with which are connected the names of Bennett† and of Paget, appear to me capable of reconciliation; and M. Robin's discovery of the lympathic sheath of the small arteries of the brain enables us, by modifying the interpretation of these two authors, to show how, by the accurate observation of identical facts, they have arrived at such opposite conclusions.

Billroth‡ appears to me to have been the first to understand that fatty degeneration of the small arteries of the brain may not always be a primary lesion, but may be dependent on change in the surrounding tissue. This is very like the opinion of Bennett, that in these cases there is rather an envelopment

* Sur la structure des artères et leur altération sénile (Compte Rendu. Société de Biologie, 1849. P. 33).

† Pathological and histological researches on inflammation of the nervous centres.

‡ Ueber eine eigenthümliche gelatinäse Degeneration der Gehirneinde nebst einigen Bemerkungen über die Bezichungen der Gefasserkrankungen zur Kronischen Encephalitis (Archiv der Heilkunde. Dritter Jahrgang, p. 47).

of the capillaries by fat than fatty infiltration of their walls. I have arrived at a similar conclusion from a study of the changes presented by the vessels in the parts of the nervous centres which are the seat of secondary degeneration.

The vascular lesions which occur in the diseased nervous tissue in cases of cerebral softening appear to me to be of the same nature, and to be brought about by a similar process. Their examination has enabled me to conclude, that in all cases of necrobiosis of the brain substance, whether due to suppression of the action of the trophic elements, or to stoppage of the action of the blood, similar changes are secondarily produced in the vessels of the affected part. This opinion appears to me to have been very generally adopted by those authors who have recently studied the anatomy of cerebral softening. I would briefly indicate the characteristic features of that change which takes place in vessels situated in the midst of brain tissue which is undergoing necrobiotic transformation. A knowledge of these facts will be of great use to us in the discussion and estimation of the opinions already expressed regarding fatty change of the capillaries and small vessels of the brain.

In the most simple case, that of secondary degeneration, in which the local circulation is not at all disturbed, in which there exists no disease in the tissue of the part about to change, but in which only the nerve tubes which traverse that part, and which have been torn at a distant point, are transformed into fatty granules because they have lost their connection with their central (*originelles*) or trophic nerve cells, in this case fatty granules are first seen scattered about in the tissue. These granules become more and more numerous, and are seen here and there to form more compact groups. These groups may have no connection with the vessels,—they are the granular bodies. Others range themselves along the capillaries; this is what has been called fatty or atheromatous degeneration of the capillaries, but which, I think, would be more correctly termed atheromatous appearance. These accumulations of fatty granules, which are sometimes sufficiently numerous to form opaque tracks, concealing the vessel which traverses them, are chiefly situated in the interior of the lymphatic

sheath. The vessel itself is not diseased, and its walls are not incrusted by fatty granules.

When a cerebral artery is obliterated by a clot formed in the vessel itself, or by an embolism, the same changes take place in the part of the brain to which the branches of the obliterated artery are distributed; the molecular fatty granules which are formed there from the necrobiotic destruction of the nervous elements, also accumulate by preference along the vessels in the interior of the lymphatic sheath.

Nevertheless, it would be an exaggeration to pretend that in ischemic softening the vessels alone do not participate in the altered nutrition which destroys all the other elements which surround them.

It is thus that the true capillaries (capillaries of the first order of M. Robin), on which the existence of the lymphatic sheath has not been demonstrated, present here and there in their walls some refracting granules, especially around, and even in the substance of the longitudinal cells. The same change may take place in the internal coat of the small arteries; as to the muscular coat, it appears to undergo fatty degeneration, as that has been seen, but differently interpreted, by Moosher* and Brummerstädt. Be that as it may, when, often after a considerable time, the fatty granules are reabsorbed, the arteriole, looked at through its lymphatic sheath, no longer presents any transverse nuclei, though the longitudinal ones remain distinct.

Similar changes may be observed in the capillaries of the walls of a hæmorrhagic cavity, when necrobiotic softening, such as has been described above, invades these walls.

It is evident, therefore, that this accumulation of fat on the vessels, this secondary fatty degeneration of the arterioles and capillaries, has often been noticed, both in softening with extravasation and in simple hæmorrhage; but it is also evident that we must not look on that altered condition of the vessels as the cause of a hæmorrhage of which, on the contrary, it is often only the result.

The details which have just been given will enable us also

* Ueber das pathologische Verhalten der kleinerer Hirngefœsse. Wurzburg, 1854.

to understand how Bennett, who saw the healthy vessel in the midst of this atmosphere of fatty granules, believed that he had to deal, not with a true degeneration of the coats of the vessel, but with an enveloping of the arteriole, not exactly by free granules, but by cells swollen and infiltrated by refracting granules ; for he looked upon these granular masses as exudation corpuscles,—identifying himself with that inaccurate opinion which, since Gluge, tends to identify the granular bodies, products of the necrobiosis of the nervous tissues, with inflammation cells, in other words, with white cells become granular.* It need not be matter of surprise that Paget, seeing these granular accumulations limited externally by the lymphatic sheath, and not knowing of the existence of that sheath, should have believed in degeneration of the walls with persistence and elevation of the adventitious tunic. Nor is it to be wondered at that Leubuscher,† in this question, should have supported the views of Paget and Bennett regarding the situation occupied by the fatty granules in this altered condition of the small vessels of the brain. Indeed, the persistence of this so-called adventitious tunic enveloping the fatty granules, and, on the other hand, the integrity of the calibre of the vessel, made those who regarded this change as primary, and who were ignorant of the existence of the lymphatic sheath, admit that this degeneration took place at the expense of the middle tunic. This is indeed the opinion enunciated by Moosher, by Leubuscher, by Hasse, and by Todd.‡ In this fatty change in the small vessels the fat occupies the same situation that the blood does in capillary apoplexy ; this remark has already been made. Moosher says, indeed, that the fat may distend the adventitious coat to the point of bursting, and that this change presents the greatest analogy to the dissecting aneurisms of Pestalozzi ; now, it has already been said that these so-called dissecting aneurisms of Pestalozzi are only hæmorrhage into the lymphatic sheath. This analogy, moreover, did not escape Pestalozzi or Kölliker,

* Regarding the distinction which should be established between the cellules of inflammation and the granular bodies, see Poumeau, "Du role de l'inflammation dans le ramolissement cérébral," p. 128.

† Op. cit. p. 218., en note.

‡ Clinical lectures on paralysis, p. 126.

under whose direction his researches were carried on. He indeed makes this remark,* with reference to the observation which has been cited above, that in the vessels of old softening, the granular bodies occupy the same situation between the middle and adventitious coats, that the blood does in recent hæmorrhage, and he is led to believe that these granular bodies result from the transformation of the elements of blood long effused between these tunics; and this in turn leads him to conclude† that the process of hæmorrhage and that of softening are identical. I do not think it would serve any useful purpose now to insist on showing how erroneous is this opinion, which results from a false interpretation of facts correctly observed.

I believe that I have shown that the secondary fatty change in the small vessels of the brain corresponds exactly to that which the different authors, to whom I have referred, have described under the name of fatty change. As this condition is only developed subsequently to the cerebral lesion, it cannot be regarded as the cause of hæmorrhage into the brain. I may add, that I have never met with it in recent sanguineous apoplexy not complicated with softening.

I believe, then, that I am right in formally denying the part which many authors, notably Paget, Hasse,‡ Todd,§ and Eulenburg,‖ have ascribed to it in the production of hæmorrhage. I am aware, however, that the true fatty degeneration which is secondarily developed in vessels situated in the midst of a patch of softening, may not be without influence, as Meissner ¶ has pointed out, in producing extravasation in the interior of the already established softening. Indeed, I have already made this reservation above. In these cases, when fatty degeneration leads to rupture of the vessel, we may have hæmorrhage into the lymphatic sheath, a so-called dissecting aneurism of Pestalozzi, as Hasse** appears to have observed.

* Op. cit. p. 19.

† Pestalozzi. Ueber Anevrysmata spuria, etc., p. 22.

‡ Handbuch der spec. Path., band iv. p. 382.

§ Op. cit. p. 126.

‖ Archiv. für Path. Anat. und Physiol., band xxiv. p. 361.

¶ Zur Lehre von der Thrombose und Embolie (Schmidt's Jahrbücher, 1861, No. I.)

** Loc. cit.

Primary fatty changes in the capillaries of the brain have, however, a real existence; but as they appear to have been confounded with the preceding, their history is but little advanced in a pathological point of view. I would remark, however, that they are always much less marked, and that the fatty granules do not form considerable masses, but are more or less separated from each other, being more numerous only on a level with the nuclei. This condition is not very rare among old people, but it does not appear to be sufficiently marked to permit of its being regarded as a potent cause of hæmorrhage. I would add that hæmorrhage frequently occurs in individuals who present no trace of this change.

Independently of old age, primary fatty change in the capillaries may be developed in certain diseases, and may sometimes be accompanied by ruptures and extravasations. Virchow * says that he has seen this degeneration in cases of chlorosis, and especially in one case in which cerebral hæmorrhage supervened. There is one source of error in the investigation and estimation of the influence of premature fatty degeneration of the small vessels against which we must be on our guard. It sometimes consists only of an accumulation of fatty granules in the lymphatic sheath, due to some influence, the exact nature of which is unknown, but which, at all events, is not connected with any affection of the tissue of the brain.

I have pretty frequently met with it in children affected with chronic diseases, especially with diabetes mellitus, and even in young people who had died of acute inflammation of the lungs. This remark appears to me to diminish the importance which has been attached to a case, related by Moosher, of fatty degeneration of the capillaries of the brain in a cachectic child. Moreover, there was no hæmorrhagic complication in this case any more than in those which I have observed.

I come now to the study of another order of vascular changes, which have been regarded as capable of producing cerebral hæmorrhage; I refer to aneurisms of the vessels of the brain.

I exclude at once, as not coming within the precise limits

* Handb. der Spec. Path. und Therap., band i.

to which I wish to confine the study of cerebral hæmorrhage, aneurisms of the internal carotid or vertebral arteries, or of the large trunks of the base, or even of the more or less considerable branches of the meninges. These aneurisms, seen by Vieussens, and referred to by Morgagni as capable of producing apoplexy, have been well described by M. Serres,* by Gull,† and quite recently by Gougenheim,‡ and by Lebert.§ It is evident, from the numerous cases related by these authors, that rupture of these tumours may produce death by meningeal hæmorrhage, and secondarily by cerebral hæmorrhage. In two cases which I saw along with M. Charcot, hæmorrhage did not occur. In cases in which they have produced apoplexy, the hæmorrhage has generally been into the meninges, but it might implicate the surface of the brain, and thence tear a way into the interior, and even into the ventricles.

I at once pass to the examination of the aneurismal dilatations of these vessels, which form an integral part of the tissue of the brain.

I shall first speak of the dissecting aneurisms to which I have already had frequent occasion to refer.

Pointed out for the first time by Pestalozzi,‖ under the direction of Kölliker, afterwards described by Kölliker¶ himself under the name of false aneurisms, they have been studied anew by Virchow,** who has included them under the head of dissecting aneurism (*ectasie disséquante*). Pestalozzi†† says, that in five cases of apoplexy which he had observed, in every one these aneurisms were found in the walls of the cavity. He says "that the internal and middle coats were

* Nouvelle division des Apoplexies (Annuaire medico-chirurgical des hôpitaux civils de Paris, 1819).

† Guy's Hospital Reports, vol. v. série 3.

‡ Des tumeurs anévrysmales des artères du cerveau (Thèse de Paris, 1866).

§ Ueber die Anevrysmen der Hirnarterien, Lettres à Frerichs (Berliner Klinische Wochenschrift, 14 Mai, 28 Mai, 11 Juin 1866).

‖ Ueber Anevrysmata spuria der kleinen Hirnarterien. Wurzburg, 1849.

¶ Zeitschrift für Wissenchaftliche Zoologie, band i. p. 204.

** Ueber die Erwesterung kleinerer Gefæsse (Arch. für Anat. und Phys., band iii. 1854, p. 427).

†† Op. cit. p. 7.

ruptured, and that the wall of the aneurismal sac was formed only by the most external layer of the adventitious tunic, as is the case in false aneurisms of the large arteries." Without entering into further details, Pestalozzi refers to the presence of these aneurisms in a man of 82, in a woman of 63, in a man of 69, and lastly, in a man of 70. All these patients presented at the autopsy a recent and considerable effusion of blood into the brain; the last had, besides, some patches of red softening.

It is only in connection with a fifth case that he enters more fully into the consideration of the subject of the anatomical constitution of these aneurisms: I refer to the case of Ursule Hammer, which I have already analysed above. I showed then that these aneurisms were nothing more than hæmorrhage into the lymphatic sheath, that is to say, into a pre-existing cavity. They are, therefore, very improperly called dissecting aneurisms. They are not then, as supposed by Pestalozzi and all those who have since pointed out this condition, a cause of hæmorrhage, but only the first stage of a hæmorrhage already effected. Indeed, there is nothing specific in their nature, since they are found just as frequently in red softening as in hæmorrhage properly so called. Moreover, in admitting that the aneurisms of Pestalozzi are really a cause of hæmorrhage, we only shelve the difficulty; it remains still for us to inquire why the internal and middle tunics are ruptured, or rather why the vessel is ruptured. I have already said that in hæmorrhage this rupture appears to me to be altogether mechanical: it proceeds from tearing of the nervous tissue by the irruption of a sanguineous effusion which is to be ascribed to another cause. The small vessels of the torn portion are also stretched, afterwards ruptured, and then the blood which traverses them is directed into the lymphatic sheath, if it still remains entire, and forms the punctated capillary apoplexy which is so frequently found around hæmorrhagic cavities. If, on the contrary, the lymphatic sheath is primarily ruptured, or if it gives way secondarily, the blood of the vessels torn by the first effusion is added to the primary collection.

In softening the rupture of the vessel is much more difficult

of explanation; yet it does occur, and there are produced effusions into the lymphatic sheath, dissecting aneurisms. Pestalozzi has not taken into consideration, this difficulty; but Hasse * has referred to fatty degeneration, causing rupture of the internal and middle coats, as one of the causes of the aneurisms of Pestalozzi; and Meissner † adds to this fatty change the influence of the collateral flow which produced the rupture. I have remarked above that the extreme rapidity with which capillary apoplexy is produced in certain cases of softening appears to me at variance with this interpretation.

Apart from the so-called dissecting aneurism, there remains little to be said regarding what has hitherto been described as aneurisms of the small vessels of the cerebral pulp. Hasse and Kölliker saw in red softening vessels gorged with blood, and presenting ampullar dilatations, but they did not see in that the possible cause of the hæmorrhage. Moreover, in the case to which I have alluded there is no mention made of extravasations. This dilatation of the capillaries is the lesion of those passive hyperemias which often accompany obliteration of the arteries, and without doubt precedes the rupture whence results the capillary apoplexy. It is this condition which, as it occurs in cerebral softening, has been well described by M. Laborde ‡ under the name of moniliform dilatation.

Hasse § says that he saw along with Kölliker two cases of atheromatous dilatation of the small arteries of the brain continued into the capillaries; but there, however, it was not a question of hæmorrhage. This widening of the small vessels and of the capillaries is, on the other hand, distinctly referred to by Rokitansky ‖ as a possible cause of sanguineous apoplexy.

Eulenburg ¶ cites among the causes of cerebral hæmorrhage the ampullar dilatation of Virchow; but this aneurismal change

* Op. cit. p. 382.

† Zur Lehre von der Thrombose und Embolie, loc. cit.

‡ Recherches sur le Ramolissement, 1866.

§ Ueber der Verschliessung der Hirnarterien als nachste Ursache einer Form der Hirnerweichung (Zeitschrift für rationell Medicin, 1847. Band iv. p. 91).

‖ Lehrbuch der pathologischen Anatomie, 1856, t. ii. p. 450.

¶ Op. cit. 341.

of the small arteries was seen by Virchow only in the arterioles of the pia mater, and this last author* took care to add that the patients had not presented any cerebral symptoms; he only regards this disease as a possible cause of meningeal hæmorrhage. We shall have to return to this subject, however, in the following chapter.

We come now to consider the influence which should be attributed to incrustations of the large arteries of the base of the brain and their principal branches, which have so long been regarded as most important factors in the production of cerebral hæmorrhage.

Abercrombie, who first called attention to the importance of arterial incrustations in the pathology of diseases of the brain, and who not unreasonably attributed to them a large share in the production of softening, likewise thought that they might not be without influence in cerebral hæmorrhage. He admitted† "that the rupture was the result of disease of the artery itself, without being foreign to that state of congestion or hæmorrhage which appears to constitute simple apoplexy. That condition of disease of the artery generally exists indeed in cases of this kind. It sometimes consists in ossification of the vessel at different points, and sometimes in that peculiar state of chalky infiltration which renders the vessel friable." These incrustations, on which so much stress has since been laid, are of different natures. Besides simple fatty change commencing in the middle coat, there is passive calcareous infiltration, or petrifaction, then fatty degeneration or ossification resulting from the arterio-sclerosis of Lobstein, or the chronic or deforming endo-arteritis of more recent authors. I would also indicate chronic peri-arteritis with secondary fatty degeneration of the mascular coat which Rokitansky regarded as of frequent occurrence in the cerebral arteries, and which, according to him, might produce hæmorrhage. This opinion enunciated in his "Manuel d'Anatomie Pathologique" in 1844, was not reproduced in his "Traité," at least in the 1856

* Ueber Erweiterung kleinerer Gefoesse (Arch. f. path. Anat. und Phys. Band iii. 1851, p. 442).

† Recherches pathol. et prat. sur les maladies de l'encèph. ; trad. de Gendrin, 1832, p. 836.

edition. All these conditions, so different in their nature, have certain external analogies which have long caused them to be confounded under the common denomination, atheroma; and it may be said that practically the distinction is not always made out. In considering the pathological import which has been attributed to atheroma of the cerebral arteries, I shall be under the necessity of maintaining this confusion.

The opinion of Abercrombie, taken up by M. Bouilland, admitted without reserve by Rokitansky, who explains by the symmetry of atheroma that remarkable symmetry between old clots and recent hæmorrhage which is often observed, has likewise been adopted by M. Grissole, by Valleix, by Eulenburg, and by Niemeyer. Yet from the first doubts had been expressed regarding this so-called cause of sanguineous apoplexy. M. Gendrin* after remarking that he had four times found hæmorrhage take place into a cerberal hemisphere whose principal arterial trunks were diseased, whilst no effusion took place into the other hemisphere whose vascular apparatus was healthy, ends by saying that he has often seen cerebral hæmorrhage result from rupture of the arteries when there was no disease of the arteries, of the brain, or of the heart. It is this absence of all arterial lesion in a certain number of cases of hæmorrhage which has induced the authors of the "Compendium de Médecine Pratique" to deny altogether the influence of atheroma.

The authors who regard this change in the vessels as playing an important part in the pathology of sanguineous apoplexy have differed, however, regarding the interpretation of the facts. The old opinion which attributed the effusion to rupture taking place at the point of ossification, might be supported by a few facts; but then similar ruptures ought to be found chiefly in the large arteries of the base, and in the meningeal branches; and, as MM. Behier and Hardy† have said, this hypothesis could be invoked only with reference to the pathology of meningeal hæmorrhage.

But in the production of hæmorrhage atheroma may act

* Traité philosophique de médecine pratique, p. 484.

† Op. cit. t. iii. p. 415.

indirectly, and not by rupture of the incrusted wall of the vessel. Without recalling the opinion which attributes hyperatrophy of the heart to arterial atheroma, and hæmorrhage to the combined action of cardiac hypertrophy and atheroma, it may at least be said that through the loss of their elasticity, the vessels, by allowing the blood to arrive in a jerking manner, fail to deaden the shock of the impulse of the heart, which at each beat gives rise to greater tension of the vascular extremities.

We have already considered this influence which does not belong to the class of causes whose examination is the especial object of this chapter. I would only remark that M. Gendrin has ventured on a new explanation,* and that after having sought to establish, as I have said above, the slight influence exercised by atheroma, he subsequently endeavours to explain how it acts. He admits that the cardiac effort clashes with the resistance offered by the contraction, rigidity, and roughness of the incrusted arteries, that the force of the blood current is thereby relaxed, and that a sort of stasis is produced beyond the obstacle. From this stasis may result congestion and the hæmorrhage which is its consequence. This is to anticipate by a gratuitous hypothesis the difficulties which are now experienced in interpreting the hyperæmia which frequently accompanies arterial obstruction.

M. Durand-Fardel,† who has resumed the study of this question, has pertinently remarked that the influence of atheroma has been theoretically admitted without a proper examination of the facts; he says, indeed, that in the works published on this subject he has found only eleven cases of cerebral hæmorrhage in which the condition of the arteries was noted. To these he has added twenty-one others, and has thus been enabled to show that out of thirty-two cases of cerebral hæmorrhage the arteries were healthy in four, indurated in nineteen, and ossified in nine. He then endeavoured to ascertain the proportion in which atheroma occurred in individuals presenting no cerebral disorder, and who were of the same age as those who were the subjects of the first

* Leçons sur les maladies de cœur, p. 234.

† Traité clin. des mal. des vieill, p. 228.

statistics. He found as the result of his inquiry that in thirty-two individuals above sixty years of age, in the Bicêtre and Salpêtrière, the arteries were healthy in nine, thickened in twenty-one, and ossified in two.

I have made similar researches on a larger number of patients similarly situated in all respects. M. Charcot has kindly communicated to me twenty-four cases of cerebral hæmorrhage in which an autopsy was made, collected by him at the Salpêtrière. M. Vulpian has also favoured me with thirty-one analogous cases likewise collected at the Salpêtrière, and during the same years. In these fifty-five cases the state of the arteries of the brain was noted only thirty-nine times. In these thirty-nine cases the arteries were not at all atheromatous seven times in patients from 62 to 84 years of age; they were very slightly, or scarcely at all so eleven times in patients from 53 to 81 years of age; they were noted as being only atheromatous thirteen times in patients from 53 to 84; and they were very atheromatous eight times in patients from 66 to 88 years of age.

Thus, even leaving out of account the eleven cases in which the arteries were very slightly or scarcely at all atheromatous, we have, in thirty-nine cases of hæmorrhage, seven in which the cerebral arteries were perfectly healthy. That is a proportion of about 18 per cent. In the statistics of M. Durand-Fardel the proportion was about 12 per cent. According to the same author the arteries are healthy in 28 per cent. of people above 60 years of age. It appears that atheroma is more frequent in individuals who die of cerebral hæmorrhage than in those who present no disease of the brain; but the difference is not very considerable, and the perfect integrity of the arterial system observed in 18 per cent. of the cases of hæmorrhage occurring in old people sufficiently proves that atheroma is not the essential cause of sanguineous apoplexy, and that it acts only as an aiding or predisposing cause.

It may be added that cerebral hæmorrhage is not observed in the mammifera, which are equally subject to atheroma of the cerebral arteries.*

* Altération graisseuse sénile des vaisseaux de l'encéphale chez certain mammifères (Vulpian Comptes-Rendus de la Société de biologie, 1864).

After having gone over this long series of causes we have only arrived at negative results; we have been able to determine the proximate cause of some exceptional cases of cerebral hæmorrhage, and we have indicated causes which may often intervene, but in a secondary manner. We have not found the real cause of the hæmorrhage either in exaggerated blood tension, in the morbid changes of the surrounding tissue, or even in the vascular changes hitherto described.

CHAPTER IV.

ON A HITHERTO UNDESCRIBED CHANGE OF THE SMALL ARTERIES OF THE BRAIN, AS THE MOST FREQUENT CAUSE OF CEREBRAL HÆMORRHAGE.

BEFORE pointing out the cause which appears to me to produce the most common forms of cerebral hæmorrhage,—those which occur in old age,—I would remark that this is a line of research which has perhaps been too much neglected, and which might, no doubt, long ago have solved this important question in pathology. Before discussing the vascular changes which might produce the hæmorrhage, it was well to determine the vessel whose rupture had produced effusion. An examination of its walls at the point of rupture, greatly facilitated the study of the changes which had preceded the apoplexy. This investigation, it must be owned, is not very difficult, but demands much time and minute attention. Abercrombie, who understood its importance, says* that "researches to determine specially which are the torn vessels are, in general, useless." Yet M. Gendrin affirms that he has been more fortunate; and, were he not careful to say† that the arterial ruptures which always exist are often with difficulty verified, it might be supposed from the precision of his details that this minute investigation was to him only an easy and pleasant task. "The arterioles which supply the blood which is extravasated," says M. Gendrin,‡ "are generally branches of the fourth or fifth degree, rarely more than one-third of a line in diameter. In a great many cases the rupture can be de-

* Researches on diseases of the brain, &c.

† Op. cit. p. 477. ‡ Loc. cit.

tected by very carefully washing away the coagulated blood, by means of a very fine stream of water, from the walls of the cavity to which it adheres. The arterioles may thus be traced from their origin onwards into the clot, by the detritus of which their extremities are surrounded. Their sudden termination in the middle of these pieces of extravasated blood, and the presence of fibrinous striæ which seem to prolong the extremities of the vessels in the clot, leave no doubt as to the connection between the clot resulting from the extravasation and the blood which traversed these arterioles. This arrangement is rarely found on only one arteriole: it is generally common to all the ramifications of many of these small vessels springing from the same vascular branch." I have made, along with M. Charcot, many researches of this kind, and am convinced that the action of a stream of water is not favourable to the isolation of the vascular filaments. I have always obtained much more satisfactory results from freely opening the cavity, and then leaving the brain to macerate in water frequently renewed, without shaking, but simply by inclining the vessel from time to time so as to detach piecemeal the clot which still adheres to the walls. The vessels which I have thus been able to isolate had not the dimensions of one-third of a line, as M. Gendrin says, probably, because vessels of that size are not usually met with in the substance of the cerebral pulp.

Neither have I ever met with these fibrinous striæ prolonged from the orifice of the torn vessel into the depth of the effused blood. Moreover, the results arrived at by M. Gendrin have not been attained by subsequent observers,* and M. Grisolle declares,† that he could never find the ruptured vessel. Yet this investigation had already been undertaken, and with some success, even before the labours of M. Gendrin. Cheyne‡ said, in 1812, that by gently washing away the clot, by means of a brush, one could, after a few hours, "demonstrate the

* M. Cruveilhier, in his "traité d'Anatomie pathologique générale," says (p. 217), "I do not know of any case of an apoplectic cavity situate in the depth of the brain-substance in which the arterial origin of the hæmorrhage has been demonstrated in a positive manner."

† Op. cit. p. 720.

‡ Cases of Apoplexy and Lethargy, p. 39. London, 1812.

presence along the walls of the cavity of a number of vessels terminating in small clots."

Too much importance, however, must not be attached to the presence of these ruptured vessels. It has already been said that many of them are torn secondarily, at the same time as the brain tissue, by the irruption of the blood proceeding from the vessel which first gave way. This has already been remarked by Rokitansky.* It is this first ruptured vessel whose discovery is of most importance, and it can only be recognised by the existence of certain changes, peculiar to itself, which offer a ready explanation of its rupture.

The following case afforded me the opportunity of determining, for the first time, the vessel which was the source of the hæmorrhage, and the special change which had long preceded the rupture.

CASE III.—*Cerebral Hæmorrhage; Aneurisms in the walls of the cavity; Rupture of one of these Aneurisms.*

A female, aged 61, who was said to have been paralysed on the left side for a length of time, was seized on the 11th March 1866, with an attack characterised by loss of consciousness. When admitted to the wards of M. Charcot, a few hours after the seizure she was comatose. She never regained consciousness, but gradually sank and died on the 13th.

Autopsy, 15th March.—The posterior part of the *left* parietal lobe was the seat of a considerable tumour. The convolutions were obliterated. This tumour was furrowed by two very large veins gorged with blood; on a level with one of these were seen some pseudo-membranous-looking granulations in the arachnoid. Nothing in the superior longitudinal sinus. The basilar artery was tortuous on the left side of the pons Varolii, and presented a dilatation as large as a haricot bean, which had left an impression on the pons which was thus driven to the right.

The left sylvian artery was also dilated at its origin. The right sylvian was normal in appearance. At the seat of the tumour the membranes could be raised without injury to the brain substance: there was a little suberachnoid hæmorrhage.

* Lehrb. der path. Anat., bd. ii. p. 449.

The blood, forming a clot twice the size of an egg, was surrounded by the extraventricular substance: there was nothing in the ventricle, and nothing in the corpus striatum or optic thalamus.

Right lobes.—Outside the lateral ventricle, above the optic thalamus and corpus striatum were seen two lacunæ—old cavities of a yellowish colour and very small. The convolutions near these lacunæ were neither flattened nor red.

The white substance forming the walls of the hæmorrhagic cavity was softened, reduced to a diffluent pulp, and reddened by blood in the parts nearest the clot; beyond this it had the colour of white softening, but was dotted over with little hæmorrhagic points. A few fatty granules were found in this tissue. The nuclei of the vessels were increased in number. Between the nerve-tubes were found a great many nuclei of the interstitial connective tissue of the brain.

The hemisphere which was the seat of softening was placed in water, and the vessel inclined from time to time to different sides; the whole of the softened portion was by this means removed in a few hours, and it was found that the convolutions were exposed at their base. Some still preserved villous shreds of the white substance which appeared normal under the microscope. From this base of the convolution were seen to issue vessels which proceeded to the patch of softening. Two of these were the seat of aneurisms. One of these aneurisms, of the size of a small hempseed, was split longitudinally and obliterated by a clot. The patches of cellular infiltration in the right hemisphere presented resisting filaments formed almost exclusively of empty vessels. These vessels, which were of different sizes, presented an enormous multiplication of their nuclei, a remarkable thickening of the cellular coat, and a still greater thickening of the wall proper, so that the canal was much narrowed, and in many of the capillaries was even altogether obliterated: in those in which it existed it was unequal, varicose, and filled with a granular substance readily coloured by carmine. A certain number of the capillaries were incrusted with atheroma, and some had grains of hæmatoidin on their surface. Between the capillaries was observed a finely granular amorphous substance with

nuclei (*myélocytes et embryoplastiques*), granular bodies and fatty granules, and a small quantity of the debris of non-crystallised hæmatoidin; finally, towards the surface were found drops of myeline and nerve tubes.

In this case the hæmorrhage resulted from the rupture of an aneurism developed on a small intracerebral artery. The linear splitting of the sac, and the clot which it enclosed, left no doubt as to this being the mode of origin, and the existence of another aneurism, still intact, in the immediate neighbourhood of the first, showed that the vascular change was not a limited one, but that a morbid condition of the arterial system of the brain had paved the way for this apoplexy long before it occurred.

My attention was then fixed on these slowly forming aneurisms of the small arteries, whose existence M. Charcot had already demonstrated in two cases, though the cause of death did not permit of his connecting them with the pathological history of hæmorrhage.

One of these cases was observed by him in 1863. It was that of a woman 80 years of age, the subject of chronic articular rheumatism, who had been hemiplegic for six months. At the autopsy small red points, some as large as a grain of hempseed, were found in the pons Varolii. Some of these were seen to be small vessels dilated in the form of an ampulla. Nothing is said regarding the nature of the lesion which produced the hemiplegia.

Another case observed in 1865 in a woman 71 years of age served no better to establish the possible connection between these aneurisms and apoplexy. At the post-mortem, ampullar aneurisms were observed on the small arterioles of the pia mater, and besides, a certain number of these remained adherent to the convolutions after the detachment of that membrane.

The discovery of similar aneurisms ruptured in the wall of a hæmorrhagic cavity led to a special investigation of this subject.

On the 24th May an autopsy was made on a woman 62 years of age, the subject of numerous attacks of cerebral hæmorrhage. On removing the pia mater a number of particles were seen

on the surface of the convolutions, some of a bluish black or violet colour, others ochrey; they were readily separated from the surrounding tissue, and were connected with the arterioles which dipped into the cerebral substance. Examined under the microscope they were seen to be aneurisms; in some the blood presented its normal characters; in others there were found only fatty granules and grains of hæmatoidin. The verification of these aneurisms in the superficial parts was trusted to alone; no examination of the cavity was made to see whether the rupture of similar aneurisms had determined the hæmorrhage, as in Case III.

Many similar facts have since been collected. Moreover, aneurisms of the convolutions have frequently been met with in patients who did not die of apoplexy, but who presented the ochrey cicatrices of old hæmorrhage; they have also been found in some individuals whose brains presented no trace of extravasation.

But if the aneurisms were the cause of the hæmorrhage, it would be necessary to demonstrate their presence in the central gray portions of the brain, as hæmorrhage is more frequent there than in the convolutions. Attention was, therefore, directed more especially to the examination of the corpora striata, optic thalami, and pons Varolii, and it was found that the aneurisms were as common in them as in the convolutions. In some cases of recent hæmorrhage there were found in the walls of the cavity ruptured aneurisms whose contained clot was blended with the clot of the effusion. The following cases, all collected with M. Charcot and under his direction, illustrate these different statements.

[The author here gives very full details of four cases in which aneurisms were found along with recent hæmorrhage, of one in which they co-existed with the remains of old hæmorrhage, and of one in which they were unaccompanied with hæmorrhagic effusion. I shall give only a summary of the two latter and of one of the first four.—T. J. M.]

CASE IV.—*Old Hæmorrhage; Intra-cerebral Aneurisms.*

A female, aged 76, had been under observation at the Salpêtrière for four years prior to death. She was paralysed

during all that time, and was said to have been so for six years before admission. There was hemiplegia of the right side.

Autopsy.—Brain: There was nothing noteworthy in the membranes. The arteries of the base, and those of the pia mater, were free from atheroma and aneurismal dilatation.

On examining the surface of the convolutions there were found a certain number of *small spots of a violaceous, blackish, or ochrey colour, globular in form, and in size varying from that of a millet seed to that of a pin's head.* On cutting into the hemispheres there were found in the substance of the *gray matter* of the convolutions, and on their deep aspect, similar miliary productions of the same colour as those on the surface.

There were observed also some small-sized old hæmorrhagic cavities, characterised by *a hard and ochrey cicatrix, and in the optic thalamus of the left side was a large cicatrised hæmorrhagic cavity.*

The miliary products indicated above were simply aneurisms developed on very small arteries, as was ascertained by dissection and microscopic examination. They were formed by an enveloping membrane of connective tissue rich in nuclei, and continuous with the adventitious coat of the artery. In the substance of this membrane, and external to it, were found numerous granules of hæmatoidin and some fatty granules. The arteries on which these aneurisms were developed showed no trace of circular fibres, yet their walls were thickened, and presented a good many nuclei, and often granules of hæmatoidin and of fat.

Examined at points at which neither aneurisms nor the cicatrices of hæmorrhagic cavities were found, the small vessels of the brain appeared more dilated than normal, and formed red tracks on the cut surface. Examined under the microscope they were seen to be charged with nuclei, with granules of hæmatoidin, and fatty granules. In most of them no *muscular fibres* could be detected.

The nerves of the paralysed side were hypertrophied, hyperæmic, and slightly grayish.

The muscles of the same side were seen under the microscope to have lost to a great extent their striated appearance, and to be undergoing fatty degeneration.

CASE V. (VI. of Author.)—*Cerebral Aneurisms without Hæmorrhage.*

A woman was admitted under the care of M. Charcot on the 13th July 1866. For a week she presented only symptoms of gastric derangement and bronchitis. On the 21st she suddenly became aphasic; the aphasia continued, but there was no hemiplegia or other sign of paralysis. Her general condition gradually improved, and she went out on the 5th September.

On the 16th of the same month she was re-admitted in a state of complete dementia. She gradually fell into a state of coma; the respiration became embarrassed, and she died on the 23d September without ever showing the least appearance of hemiplegia.

Autopsy.—Heart considerably enlarged, especially the left side. Two of the aortic valves were adherent, and the seat of vegetations. Mitral valve healthy. Extensive emphysema of both lungs.

There was general atheromatous induration of the thoracic aorta. Liver rather large. Right kidney hard, and very small, weighing 42 grammes; the left was larger, and weighed 88 grammes.*

There was no infarctus of the spleen.

Brain: The arteries of the base were slightly atheromatous, indurated in patches, but free from apparent narrowing of their calibre.

The meningeal arteries were not atheromatous. On the lateral part of the left anterior lobe was a patch of yellow softening, consisting of a milky part situated beneath the membranes, and of a deeper felt-like (feutrée) part, presenting the appearance of a *plaque-jaune.* The whole did not extend beyond the depth of the convolutions. The exact situation of this change was the *posterior half of the third frontal convolution, stretching towards the insula*, and encroaching a little by its posterior part on the anterior portion of the third parietal convolution. The sylvian artery was free, but one of its branches (that which goes to the posterior part of the third convolution) was filled by an old colourless clot which dis-

* One gramme is equal to 15·434 grains.

tended the vessel, and was prolonged into both its branches, which were afterwards free on the side of the softening. The softened patch was filled with granular bodies. In the corpora striata and optic thalami of both sides were lacunæ and some small ochrey patches, perhaps the remains of old hæmorrhage.

This fact may serve to explain what was observed in the medulla oblongata, *a small yellow track towards the middle and internal part of the left anterior pyramid, a track of secondary degeneration,* which under the microscope was seen to consist of an inconsiderable quantity of amorphous matter rich in nuclei, and of vessels presenting an increase of their nuclei, and some of them having fatty granules. On raising the membranes many *small aneurisms* were seen on the convolutions; they were found also in great numbers in the optic thalami and corpora striata; in one of the corpora striata there was a *small, intensely ochrey lacuna,* apparently the remains of an old hæmorrhage.

The small size of the right kidney was the result of the obliteration by a clot of one of the branches of the renal artery. The part of the kidney which suffered in consequence was undergoing fatty degeneration.

CASE VI. (IX. of Author.)—*Cerebral Hæmorrhage. Intra-cerebral Aneurisms; some Ruptured in the Cavity.*

A female, aged 79, fell down unconscious on the 12th September 1866. On the following day she had so far recovered as to understand what was said to her, and to be able to speak a little. It was found that the left arm was completely, and the left leg incompletely paralysed. She died six days after the seizure.

Autopsy.—The friends gave permission to open only the skull. There were no false membranes or adhesions in the dura mater. No opacity, thickening, or adhesions of the arachnoid and pia mater. Moderate congestion of the meninges; cerebro-spinal fluid pretty abundant. The arteries of the base were atheromatous in patches, but free from appreciable narrowing. The meningeal branches were generally normal, yet here and there opaque yellow patches were found in some of them.

In the trunk of the right internal carotid was found a small flattened granular, non-adherent clot, occupying only a part of the calibre of the vessel. A red clot was continued from the peripheral side of the preceding. The hemispheres, stripped of their membranes, were normal both in consistence and colour, saving a rather fine dotting which was dispelled by friction. There were no superficial aneurisms. On cutting into the brain, a clot of the size of a hazel nut was found on the upper and internal part of the right optic thalamus, involving the superior layer of the prolongation of the right cerebral peduncle. The walls of the cavity which contained this clot were a little ragged, but not softened. There was a rent at its upper and internal part, and the blood had made its way into the right lateral ventricle; the ventricle contained some small-sized clots; a small one was also found in the third ventricle. The ventricular fluid on both sides was tinged with blood. There was nothing abnormal in the fourth ventricle.

The brain, cut into thin slices of about a centimetre, presented no aneurism either in the depth of the convolutions or in the *centrum ovale*. Neither was anything found in the central gray portions of the side on which the hæmorrhage was. In the optic thalamus of the opposite side was a small ochrey and blackish mass of the form and size of a pea, which on microscopic examination was seen to be formed of granules and crystals of hæmatoidin, mingled with fatty granules, the whole contained in the meshes of a tissue rich in nuclei and in capillaries. No aneurism was found in it.

After the hæmorrhagic cavity was cleared out by three days maceration in water frequently renewed, we succeeded in isolating a certain number of vessels which dipped into the clot. On one of these vessels were two aneurisms, both ruptured, and the coagulated blood which escaped from them was continuous with the clot of the cavity. After the aneurism was isolated, it was seen to be ruptured on the side opposite to that by which the artery passed into its interior; it was seen, moreover, that the extravasated blood had detached the lymphatic sheath, which was also ruptured.

Another aneurism, also ruptured, was found on another vessel in the cavity.

In the substance of the pons Varolii was found another aneurism not ruptured.

An artery, with its divisions, taken from the substance of the affected optic thalamus outside the cavity, presented a thickening of its walls with longitudinal folding of the internal tunic. The muscular coat was preserved in the large trunks, but was completely awanting, or represented only by a few rings, on the finer sub-divisions. The lymphatic sheath was studded with nuclei in considerable numbers, and here and there in its interior were grains of hæmatoidin and granular bodies.

On the walls of the hæmorrhagic cavity were a number of molecular fatty granules and some granular bodies. There was no appearance of atheroma of the capillaries.

Let us now consider by themselves these aneurisms which appear to be constant in old people having either old or recent hæmorrhagic cavities in their brains, and which have also been found in some cases in which hæmorrhage, though imminent, had not yet taken place. I would first remark that they appear to me to be only an incidental part of a much more general vascular change, a sort of arterial sclerosis which seems to me to be in many respects analogous to what Rokitansky has described under the name of chronic periarteritis.

This change, which is common to the whole arterial system of the brain, manifests itself chiefly in the small arteries, but to a certain degree it may also sometimes be observed in the large vessels of the base, and in those of the meninges. It essentially consists in an exaggerated and often enormous multiplication of nuclei in the substance of the arterial coats, and on the lymphatic sheath, and in atrophy of the muscular coat, most marked in the small arteries, and even in the capillaries of the second and third order of M. Robin. This atrophy of the muscular coat manifests itself in the following manner in the small arteries. The almost contiguous transverse striæ which mark the course of the muscular fibres around the vessel are generally obscured by the nuclear multiplication indicated above, and also by the thickening of the adventitious coat which frequently complicates this condition. They are separated a certain distance from each other, and become more

and more scattered the further the examination is extended towards the parts which are most altered. At last they may be altogether awanting over a more or less considerable extent of the vessel.

Along with this disappearance of the muscular elements there is frequently observed a general dilatation of the vessel, which is constricted here and there at the points at which the circular fibres persist; there results from this a slightly moniliform appearance of the artery.* Most frequently, however, the calibre of the vessel is not increased, apparently in consequence of thickening of its walls, the result of the increased production of nuclei as much as of hypertrophy of the adventitious coat, which presents an appearance of longitudinal striation like a bundle of fibrous tissue. Lastly, and this is the most important change, that on which I would particularly insist, there are sometimes observed on a certain number of the arteries thus changed sudden dilatations, true aneurisms, the presence of which I have always succeeded in demonstrating in the brains of those who have died of sanguineous apoplexy,—the hæmorrhage appearing to me generally to result from the rupture of these aneurisms.

Before entering on the study of the characters of these intra-cerebral aneurisms, I would remark that this general sclerous condition of the arterial system of the brain, of which I have just given a brief description, has only distant analogies with what the Germans call deforming *endo-arteritis*, and that it differs essentially from atheromatous change and from simple fatty degeneration. In more than a third of the cases which have come under my own observation, it is noted that the arteries of the base and of the meninges presented no trace of atheromatous change; and that was in old people from 74 to 85 years of age. In some cases the arteries of the base presented only a few opaque, soft, non-projecting patches; so that arterial atheroma coincided in scarcely half the cases with

* M. Ordõnes has seen and shown to me arterioles from the convolutions of paralytic lunatics, in which fusiform dilatations were developed at points at which the muscular fibres had quite disappeared. These dilatations, which might be a new, but rather rare lesion of general paralysis, were much smaller than the aneurisms which I have described, and did not appear to M. Ordõnes to have any connection with hæmorrhage.

the sclerous condition of the small arteries and the existence of intra-cerebral aneurisms. On the other hand, this coincidence proves that the change described above is not incompatible with atheroma of the arteries of the brain, and that the two affections may advance *pari passu* in the same subject. Moreover, it is not only in the large arteries that this coexistence of atheroma with the sclerous and aneurismal condition of the arterioles of the brain may be observed; incrustations are also sometimes found in the small vessels which are distributed in the cerebral substance, and I have twice seen, along with hæmorrhage and aneurisms, complete calcification of some arterial branches in the substance of the optic thalami. And once I have seen in a brain crammed, so to speak, with aneurisms, a still rarer change than the preceding, and one which MM. E. Boudet and Lélut* appear to have been the only persons to observe up to this time. I refer to calcareous incrustations of the very small arteries, chiefly of the white substance of the brain and cerebellum, an incrustation which imparts to them such rigidity, that when a section of the organ is made they project one or two millimetres above the cut surface; a condition which has led to their being compared to the hairs of a brush. Though these two changes may coexist, their separate occurrence, which I have frequently seen, suffices to show that they are independent of each other, and result from distinct, though not antagonistic processes.

The sclerous change of the small intra-cerebral arteries, I have said, extends in some cases to the large trunks of the base, or the more or less voluminous branches of the meninges. In these more important arteries the change sometimes consists in simple thinning of the walls, more rarely in aneurismal dilatations. I have once seen this sclerous condition, with aneurisms of the arterioles of the cerebral substance, in a woman, who presented at the same time a fusiform and varix-like (cirsoide) dilatation of the basilar artery, and a similar dilatation of the left sylvian near its origin. In another case the same change in the small arteries of the brain was complicated with numerous aneurisms, from the size of a pea to that of a cherry-stone, attached to most of the large branches

* Quoted by Durand-Fardel. Traité des maladies des vieillards, page 53.

of the pia mater. Finally, in two cases I have seen the smallest arteries of that membrane present very numerous ampullar dilatations, similar in form and structure to the aneurisms which existed at the same time in the convolutions and central parts of the brain. There is therefore good reason for regarding the sclerous condition of the small arteries of the brain and the intra-cerebral aneurisms which frequently complicate it, and which are only a more advanced state of the same condition, as a general change in the arterial system of the brain. I am not at present in a position to look upon it as a more general affection, my patients having never presented any analogous change in the vessels of the other parts of the body.*

Let us proceed now to the consideration of the intra-cerebral aneurisms; but before pointing out their structure and their situation, with reference to the arteries which bear them, I would first indicate the appearance which they present, and the points at which they occur. All these aneurisms are visible to the naked eye; they look like little globular particles, varying in diameter from two-tenths of a millimetre to one millimetre, and even more, attached to a vessel which is likewise visible to the naked eye,—a simple lens, at least, suffices to make them quite distinct. The diameter of the vessel may vary from a third of a tenth of a millimetre to one-fourth of a millimetre, and even more. The colour of these aneurisms varies according to the state of the blood which they contain, and the condition of the walls. When the wall is thin, as it usually is, the aneurism is of a purplish colour, more or less dark if the blood which it contains is liquid. If, on the other hand, the blood, long coagulated, has already been transformed into hæmatoidin, the aneurism is reddish-brown or ochrey, or even blackish, and in that case the surrounding cerebral tissue generally presents in a less degree a similar coloration. If the walls are thickened by increase of the connective elements of the adventitious tunic, as is sometimes the case, more especially in the optic thalami, the aneurism is of a grayish

* It will be seen in the Appendix that this statement is slightly modified, and that M. Liouville has demonstrated the presence of miliary aneurisms on the small vessels of the œsophagus and heart. T. J. M.

or slightly bluish or brownish gray colour, according to the state of the contents. These various conditions of the envelope and the blood which it contains modify the consistence of the aneurism. Sometimes it is soft and fragile, giving way under the least pressure, and allowing the blood to escape in an almost normal state; at others it is hard like a grain of sand; and again it is firm and elastic.

I have met with these aneurisms in almost all parts of the brain; but they are not uniformly distributed throughout; they have their organs of predilection. They are most frequently found in the convolutions; either on the surface or in the substance of the gray matter, or at its junction with the white substance. They do not, however, really occur more frequently in the convolutions; but they are more easily discovered there. Since I made a point of looking for them in the central parts of the brain, I have invariably found that when they occur in the convolutions, they are also to be met with in the optic thalami, for example, or in the pons Varolii, and I have often met with them in these last mentioned parts, when I could not detect them in the convolutions. The parts of the brain in which I have observed these aneurisms are, in the order of decreasing frequency, the optic thalami, the pons Varolii, the convolutions, the corpora striata, the cerebellum, the bulb, the middle cerebral peduncles, and the centrum ovale.

The number of aneurisms is very variable, but cannot be estimated with accuracy, as it is not possible to cut the brain into such small pieces as to make sure that a considerable number may not have been passed over. After very minute examination I have sometimes been unable to discover more than two or three; generally a much larger number is found; sometimes even more than a hundred may be counted in one brain.

I have never met with them in patients under 61 years of age; but since my investigations were directed to this point, I have rarely had the opportunity to examine the bodies of individuals of a less advanced age.* I am not, then, in a

* Further observations have shown that these aneurisms may occur at all periods of adult life. See Appendix, in which reference is made to the case of a young man of 20, in whose brain they were found. T. J. M.

position to affirm that aneurisms, along with the sclerous condition of the arteries of the brain, constitute a senile change, though the much more frequent occurrence in old people of the cerebral hæmorrhage which I believe to be due to rupture of these aneurisms, permits at least of the supposition that they are much more frequently developed in old age than at any other period of life.

On stripping the membranes off the brain of one of these patients, there is very frequently seen, either on the surface of the convolutions, or in the fissures between them, small spots scattered about in variable numbers, sometimes very numerous, and varying in colour from a bright red to a reddish or blackish brown. On closer examination each spot is seen to consist of a particle of varying resistance, and more or less regularly spherical. They are readily isolated by means of needles from the surrounding nervous tissue, which forms, as it were, a little cup for their reception. They are continuous by one of their poles with a vessel which extends into the substance of the convolution.

When death has resulted from a recent attack, some of these particles are often found of a larger size and of a violet colour—the blood which distends them being covered only by a very thin membranous pellicle. On expelling this blood by squeezing the little mass between two plates of glass, there may be seen in the centre an aneurism similar to the others, but ruptured, and having blood effused between it and the lymphatic sheath, which alone limits the effusion. In one case such distension of the lymphatic sheath was seen around a ruptured aneurism; the sheath itself ultimately gave way, and the clot was continued from the aneurism into the dilated sheath, and from this latter into the hæmorrhagic cavity.

If the sheath resist the effusion which occurs around one of these ruptured aneurisms, the blood is partly absorbed and transformed into fatty granules, and grains and crystals of hæmatoidin, situated external to the aneurism, which forms secondary adhesions with the lymphatic sheath. Nuclear increase may even take place outside the sheath in the sur-

rounding nervous tissue; and sometimes even capillaries are developed in considerable numbers on the surface of the shrivelled particle, and seem to form vasa vasorum.

Even when there has been no effusion these aneurisms may undergo changes which render hæmorrhage impossible, and form a sort of spontaneous cure. In such cases, the blood being retarded in its course, allows numerous white corpuscles to accumulate on the internal surface of the aneurisms, whence they may be expelled by crushing the aneurism between two plates of glass; they then have a peculiar granulated aspect, and are accompanied by a finely granular, slightly fluid amorphous matter, and rather numerous red globules. At the same time the wall of the aneurism thickens and becomes opaque, and with a high magnifying power there may be seen in it beautiful fusiform connective cellules. At length the granulo-fatty change may involve the walls and contents of the aneurism which becomes obliterated, as well as the vessel which passes to and issues from it, and crystals of hæmatoidin are found in the interior for a certain distance. Coincidently with this the calibre of the vessel is irregularly diminished, the internal coat folds longitudinally, the adventitious coat is thickened, and in it are seen fusiform cells arranged in the direction of the axis of the vessel, or sometimes only numerous nuclei, presenting no regular arrangement. The transverse striæ quite disappear; nuclei appear in considerable numbers on the adventitious coat; and finally, all these elements may be infiltrated with fatty granules.

It seems to me that this description renders it unnecessary for me to show that we have here to deal with a new kind of aneurism of the small arteries of the brain. We have evidently to do neither with the moniliform dilatation of Hasse, the dissecting aneurism (*ectasie disséquante*) of Pestalozzi, Kölliker, and Virchow, the atheromatous enlargement of Rokitansky, those dilatations described by M. Vallin,* nor

* Gazette hebdomadaire, 3 Mars 1865. The case was that of a young man who had died of some pulmonary affection, and in whom the capillaries of the brain presented masses of a yellowish matter interrupting the muscular nuclei here and there. There resulted from this a dented appearance of the vessel. M. Vallin says that he has found a similar change in apoplectics.

those of M. Ordônes. There is only one kind of aneurism which at all resembles those which I have described, according to what I have seen with M. Charcot; I refer to the ampullar dilatation of Virchow. But that author demonstrated the existence of this condition only in the pia mater and not in the brain. Moreover, he insists on the fact that the circular fibres persisted at the seat of the dilatations, which is opposed to what I have observed. Yet I believe that it is impossible not to compare my aneurisms with those which Virchow has described in the meninges; they have the same slow development, the same chronic course, the same tendency to obliteration by retrograde transformation of the blood which they contain. Indeed, in one case I have seen intra-cerebral aneurisms coexisting with ampullar dilatation of the vessels of the pia mater. At all events, they differ in a prognostic point of view; and if Virchow suspected that these ampullar aneurisms of the pia mater might be a cause of hæmorrhage, the fact has never been observed, and must be very rare; for, apart from false membranes, few examples of meningeal hæmorrhage are cited. On the other hand, my aneurisms have never been awanting in twelve cases of cerebral hæmorrhage, which I have collected since I became aware of their existence. Be that as it may, I think that the description given by Virchow of ampullar dilatation may be compared with interest with that which I have given of intra-cerebral aneurisms, and I shall reproduce *in extenso* the article of the learned Berlin professor.*

"*Ampullar Dilatation.*—I take this expression of M. Cruveilhier, and use it as a general designation for the different forms of partial local vascular dilatations observed on vessels not altered, at least in calibre. It ought, therefore, to include true aneurisms and local varices; the last are very rare. The most interesting form of aneurism of the small arteries is one which I have freqently observed during the course of the past winter in the pia mater of old people, especially in the prolongations which it sends in between the convolutions. There were no morbid phenomena presented during life, and the

* Uber die Erweiterung kleinerer Gefässe (Archiv für path. Anat. und Phys. iii. p. 442).

most that could be seen was a predisposition to sanguineous apoplexy.

"They were generally fusiform dilatations involving the whole circumference of the artery. Lateral dilatations were more rare. They were sometimes isolated, at others moniliform, and separated by constrictions situated at one time on the trunk of the vessel, at another at the point of division. They varied in volume from a size almost microscopic to that of a grain of millet seed, and occurred on arteries of all sizes, from the most attenuated to those half a line in thickness. Such of these aneurisms as were recent were filled with normal red globules, and neither on the afferent nor efferent vessel, nor on the aneurism itself, was any change of structure seen. In some the white globules were even more numerous than the red. The dilated part had the same coats as the vessel, and the nuclei of the middle coat with the transverse fibres were, after the action of acetic acid, as distinct as possible. They were therefore true aneurisms.

"Starting from this condition there could be seen all degrees of atrophy of the muscular coat. In many of the sacs the walls presented points in which the muscular coat was awanting, whilst the external and internal coats still existed. The action of acetic acid rendered apparent the spaces between the transverse fibre cells, then these narrow cells were scattered over vacant spaces; in other cases the atrophy of the middle coat was circular. At first there seemed to be only one tunic, but on more careful examination it was seen to be compound. By the side of these recent permeable aneurisms, forming red particles visible to the naked eye, were constantly found older ones forming white, non-transparent particles. By the aid of the microscope there was seen in them a highly refracting granular substance, sometimes in the interior of the canal, at others deposited in the substance of the walls. This consisted of a collection of fatty granules, a sort of emulsion produced by the metamorphosis of the coats themselves and perhaps of other elements deposited between the vessel and the adventitious coat.

"This fatty mass often occupied just the half of the dilatation, and the blood only penetrated into the half of it. I

could not determine whether there was a true stagnation of the contents with fatty degeneration of the white globules."

Notwithstanding the notable differences indicated above, it is evident that there are real analogies between the ampullar aneurisms of M. Virchow and those which I have described. They are quite distinct only with reference to the point which now specially occupies our attention, the pathology of cerebral hæmorrhage.

The only fact of which I am cognizant, which has any analogy with mine, is an observation of Gull,* in which, in referring to aneurisms of the large arteries of the base, he makes mention of an aneurismal tumour in the substance of the pons Varolii, having no direct connection with the basilar artery. Death was caused by hæmorrhage consequent on the rupture of this aneurism.

A case reported to the Société de Biologie by M. Hayem,† subsequently to the communications made to the same Society by M. Charcot and myself, has appeared to that observer to be allied to the cases which we have described. The aneurism was as big as a large pea, and was situated on one of the branches of the sylvian artery. It was, however, imbedded in the cerebral substance, and its rupture had produced hæmorrhage both into the meninges and into the ventricles of the brain.

Another case of hæmorrhage resulting from the rupture of aneurisms, in all respects similar to those which I have described, has recently been reported to the same Society by M. Hayem.

Though the existence of this aneurismal diathesis in the brain does not seem to have been suspected hitherto, it must be said that the principal characters by which it is recognised in the dead body were pointed out with scrupulous accuracy by M. Cruveilhier, who has described aneurisms of the convolutions under the name of *capillary apoplexy with miliary cavities.* He has figured them with perfect accuracy in his atlas of pathological anatomy, and his description conforms in all points to what I have observed. He thus describes capil-

* Guy's Hospital Reports, vol. v. sect. 3.

† Gaz. Méd. de Paris, 1866.

liary apoplexy with miliary cavities :*—"These little cavities are generally situated in the gray matter, some in its superficial, and others in its deep layer; the former are exposed on removal of the pia mater; the latter are covered by a thin layer of gray matter, and at the points at which they occur the surface of the brain is of a violaceous colour." As an instance of that form of capillary apoplexy, he reports the following case :†—A female, aged 63, was seized with vomiting on the 24th November 1836; on the following day she was hemiplegic on the left side; she died on the night of the 26th –27th. Post mortem examination showed pneumonia of the left side. As to the brain, "I believed," says M. Cruveilhier, "that it was softened, and there were two apoplectic cavities, one of which was of considerable size, and was situated in the substance of the lobe of the corpus striatum. This cavity was formed at the expense of the external part of the corpus striatum, of the radiations which separate the external from the internal part, and of a portion of this internal part. A spheroidal hæmorrhagic cavity of the size of a small nut occupied the substance of one of the posterior convolutions. The walls of the cavity were tinged of the colour of wine lees by blood, and this tinge had penetrated for a certain depth. Moreover the brain presented an enormous quantity of petechial spots of scorbutic aspect, or rather of small miliary effusions, most of them projecting on the surface of the brain, but some concealed in the substance of the gray matter. But independently of these dark red spots there was seen on the surface and in the depth of the gray matter a number of very small brown or brownish yellow, very dense granules, like grains of sand differently coloured, scattered over the surface of the brain. Does not the coincidence of small miliary sanguineous cavities, and of brown and brownish-yellow granules, show that these granules and these sanguineous cavities belong to the same form of lesion, and that the granules are only the cicatrices of small hæmorrhagic cavities."

The author adds that he has often met with these granules

* Livr. xxxiii. planche ii. fig. 3.

† Ibid. p. 5.

in the brain, and frequently in the medulla. "One would say that coloured sand had been deposited on the surface of the brain, and had sunk into its substance." A little farther on he again returns to the same subject:—"Diffuse apoplexy manifests itself sometimes as petechial spots of ecchymosis; at others, as miliary cavities or globules of blood, the one superficial and at once appearing when the pia mater is removed, the other concealed by a thin layer of cerebral substance. These small miliary cavities must not be confounded with cerebral varices, which are quite different in their nature; in the latter the blood is contained in dilated veins, whilst in the former it is effused outside its reservoirs.

"In whatever form it shows itself, diffuse capillary apoplexy invades a very large number of points at once, and the intervening cerebral substance is always in a state of perfect integrity.

"I have almost invariably met with these scattered miliary cavities in women belonging to the class of 'gâteuses,' in whom the intelligence is enfeebled almost to dementia, and who, in consequence of this feebleness, no longer retain their urine or fæces." It is clear from all that precedes, that M. Cruveilhier has seen the cerebral aneurisms and has given a very exact description of them. But it is also evident that he did not recognise their true nature, and that he regarded as actual hæmorrhage a change which served only to prepare the way for such an event. Indeed, the case which I have just briefly reproduced, tends to support my argument, by showing the relation which exists between cerebral aneurisms and sanguineous apoplexy.

A new thing deserves a new name. The analogy in form and size which exists between a millet seed and the aneurisms which M. Charcot and I have pointed out, and the name of miliary cavities, given to them by M. Cruveilhier, induce me to propose for them the name of *miliary aneurisms.*

I think that I have shown that the cerebral hæmorrhage of old people, much the most frequent form of hæmorrhage in the brain, results from the rupture of aneurisms developed on small diseased arterial branches. But I would not contend that other forms of hæmorrhage may not occur in old

people; those especially which are connected with changes in the blood appear to me to form an exception; and yet, in the following case, for which I am indebted to the kindness of M. Vulpian, cerebral hæmorrhage, connected with an attack of febrile jaundice, consequent on cancer of the bile ducts, was brought about through the medium of miliary aneurisms, which had no doubt existed for a long time.

CASE VII. (X. of Author).—*Jaundice from Biliary Retention, produced by Cancer of the Bile Ducts; Cerebral Hæmorrhage; Aneurisms.*

A female, aged 62, suffered from jaundice, accompanied with pains in the region of the liver, for some time before death. On the morning of the day on which the fatal event occurred, she was drowsy, spoke with some difficulty, and could not raise herself in bed. The drowsiness increased; she sank into a state of coma, and died during the night.

Autopsy.—There was a cancerous tumour on the lower surface of the liver.

Cranial cavity.—There was no lesion of the cranium or cerebral envelopes.

The arteries of the base were slightly atheromatous, and, like all the tissues, the atheromatous patches were coloured yellow. The pia mater was of a yellowish colour; there was no hæmorrhage into the membranes. The cerebral substance was normal to the touch.

On separating the brain from the cerebellum, a yellowish colour was observed in the centre of the left peduncle, an appearance which did not exist in the right; liquid blood was then seen to flow in considerable quantity from the ventricles. It was contained in the two lateral ventricles. In the right ventricle was found a large, irregular, mamillated recent clot. The optic thalamus was destroyed, and replaced by a large clot, around which there was no more than a thin layer of brain substance, as was proved by microscopic examination; it was still adherent, however, to the rest of the brain.

In the corpus striatum of the same side was a cavity whose walls were tinged of a markedly yellow colour to the depth of one or two millimetres; it contained no clot. In the left

lateral ventricle was a clot the size of a nut, soft, but not breaking down under the finger. On cutting into it there was found a number of rounded cavities, capable of holding a large pea.

The left optic thalamus contained a small hæmorrhagic cavity, about the size of a large hazel-nut.

The walls of the ventricles and of all the hæmorrhagic cavities were impregnated with the colouring matter of the bile to the depth of one or two millimetres. This yellow coloration was observed in all the parts of the brain which were in contact with effused blood.

In the gray matter were found some black points of the size of a lentil, consisting of small hæmorrhagic effusions, and in their vicinity some small aneurisms.

In the centrum ovale the tissue appeared sound. The bulb was healthy. The pons Varolii contained four or five vessels, which were the seat of aneurisms. There was no hæmorrhage in it.

On the surface of the right lobe of the cerebellum was seen a bluish coloration, the sign of a hæmorrhagic effusion, which had not destroyed the nervous tissue; the hæmorrhagic cavity was entirely limited to the surface of the lobe, and did not communicate either with the interior or with the fourth ventricle. This latter contained a clot, of small size, which sent a prolongation into the aqueduct of Sylvius; it proceeded from the lateral ventricles.

The medulla was not examined.

It is evident that though hæmorrhage, connected with change in the blood, may be developed without any appreciable change of structure in the vessels, it may also, as in senile hæmorrhage, result from the rupture of miliary aneurisms.

CONCLUSIONS.

Hæmorrhage occurring in the substance of the brain may be produced under various pathological conditions.

The numerous causes capable of producing increased tension of the blood in the vessels of the brain appear, in some exceptional cases, to have a determining action.

Altered consistence of the cerebral tissue seems to be only an accessory, and even doubtful, cause of hæmorrhage.

The vascular changes hitherto regarded as capable of producing hæmorrhage are much oftener the result of the cerebral lesion than the cause of the apoplexy.

These various forms of hæmorrhage, resulting from different causes, are not connected by any one pathological bond, and can only be regarded as accidents or complications of different diseases.

Senile hæmorrhage, on the other hand, results from a pre-existing change in the vessels, a change which is always the same, and which consists in a sclerous condition of the small arteries, with atrophy of the middle coat, and the secondary production of aneurisms, whose rupture is the proximate cause of the effusion. Senile hæmorrhage is, therefore, a disease *per se*.

These aneurisms which do not appear to have been described or even recognised, were, nevertheless, seen by M. Cruveilhier, and regarded as a variety of capillary apoplexy.

They are slowly developed, and generally exist in considerable numbers. They thus prepare the way long beforehand for the rupture which various accidental causes may determine.

They are readily recognised on the surface of the convolutions ; they are more abundant, but not so readily recognised, in the central gray parts of the brain.

They are never altogether awanting in the brains of those who die of recent hæmorrhage, and are then found ruptured in the walls of the cavity. They are also constantly found in the brains of those who present the cicatrices of old hæmorrhage.

Atheroma of the cerebral vessels is not then the cause of hæmorrhage.

Cerebral hæmorrhage and softening of the brain are two essentially different diseases.

Aneurisms bear to the cerebral hæmorrhage of old people the same relation that atheroma does to senile softening.

APPENDIX.

NEW RESEARCHES INTO THE PATHOLOGY OF CEREBRAL HÆMORRHAGE.

By MM. J. M. Charcot and Ch. Bouchard.

In again approaching the question of the pathology of cerebral hæmorrhage, we propose to limit ourselves to the study of those hæmorrhages which occur primarily in the depth of the brain substance, thus leaving out of account a good many cases of intra-cranial hæmorrhage. We shall, therefore, not enter on the consideration of those which may occur outside the dura mater, those which are produced in the cavity of the arachnoid, or those which are developed in the substance of the pia mater. Neither shall we direct attention to meningeal or sub-meningeal hæmorrhage; we shall thus be spared the necessity of referring to the subject of false membranes of the dura mater, as well as to that of rupture of the sinuses and large arterial trunks of the base. Lastly, we shall not have to consider the more or less extensive aneurismal dilatations which may occur on the internal carotid or vertebral artery, or more frequently on the different-sized branches which anastomose at the base of the brain, or are distributed in the substance of the meninges. This more special subject has been treated in considerable detail of late by Gouguenheim* and by Lebert,† and has been completed by the researches of M. Lépine.‡

* Gouguenheim. Des tumeurs anévrysmales des artères de cerveau. Paris, 1866.

† Lebert. Ueber die Aneurysmen der Hirnarterien (Berliner klinische Wochenschrift, 1866).

‡ R. Lépine. Note sur deux cas d'hémorrhagie sous-méningée (Gazette Médicale de Paris, Novembre et Decembre 1867).

It is to the study of cerebral hæmorrhage, properly so called, that we wish to confine our attention. The pathology of this subject may be said to be in a state of confusion. Causes, the most numerous and most varied, have been invoked in explanation of the vascular rupture which produces the effusion of blood into the brain. These supposed causes may be grouped under three chief heads :—

1. Exaggerated tension of the blood in the vessels of the brain.

2. Diminished consistence of the previously altered brain tissue, which does not afford sufficient support to the vessels.

3. Diminished resistance of the vessels consequent on change in their walls.

In these three groups may be included all the causes which have been considered capable of explaining the production of cerebral hæmorrhage, and to them also may be referred the opinions most generally entertained. We would specially refer to the influence of hypertrophy of the left ventricle of the heart; to increased blood tension consequent on disease of the kidneys, or diffuse sclerosis of the arterial system; to pre-hæmorrhagic softenings; to weakness of the small vessels, the result of fatty degeneration, or weakness of the larger branches produced by atheromatous incrustations.

We have already, on several occasions, endeavoured to show that many of these so-called causes have no real existence; that others have only a doubtful effect; that others, again, intervene only in a secondary manner and as aiding causes; and that not one presents itself with sufficient constancy to entitle it to be regarded as the true pathological process, we shall not say of all cases of cerebral hæmorrhage, but even of a majority of them.

I. In contradistinction to this at least questionable etiology, we have pointed out a new pathological lesion which has constantly been met with in the already numerous cases of cerebral hæmorrhage which have come under our notice. This lesion consists in an altered condition of the arterial system of the brain, with the production of aneurisms on the intra-cerebral arterioles. These aneurisms, which generally exist in large numbers in the brains of those who die of sanguineous apoplexy, which are developed slowly and not all at once, precede the seizure by a variable and often very long time; and finally, giving way under the influence of some accidental cause, they determine the effusion which forms the apoplectic cavity. To these aneurisms we have given the name of miliary aneurisms.

Already noticed by M. Cruveilhier,* then by M. Calmeil,† but without their true nature being recognised, these miliary aneurisms have been seen by other observers. To them, we think, should be referred a case observed by Gull ;‡ they appear to us to have a very close analogy with the dilatations seen by Virchow§ in the vessels of the pia mater, and ranked by that author under the head of ampullar dilatation. Finally, it is certainly them that Meynert‖ and Heschl¶ have studied. But what has not been pointed out is the pathological part which they play in the production of cerebral hæmorrhage. Gull, indeed, ascribes to the rupture of such an aneurism, the hæmorrhage which existed in the only case of this kind which he had observed, but he saw in it only an exceptional fact which does not appear to have fixed his attention on the pathological consequences which might be deduced from it.

The numerous facts which have come under our observation have gradually led us to the opinion which we would now maintain, an opinion which is little different from that which we have formerly upheld. One of us already had occasion in two cases, in 1863 and 1865, to observe these miliary aneurisms, but the facts did not permit of our seeing the relation which they might have to cerebral hæmorrhage. It was on the 15th March 1866, when examining the cavity of a recent sanguineous apoplexy, after having cleaned it with care, we were enabled to demonstrate on its walls the existence of two small globular, spherical masses, each suspended from a vascular filament. These were two small aneurisms, one of which was ruptured ; through the rupture could be seen the bloody contents, which, before being artificially separated by us, must have been continuous with the mass of the apoplectic clot.

This might, indeed, have been an exceptional case ; but our attention was directed to this point, and we were enabled to compare these aneurisms with those which we had formerly observed, without discerning their relation to hæmorrhage, and of which we had preserved

* Anatomie pathologique du corps humain, livrais xxxiii. pl. ii. fig. 3.

† Traité des maladies inflammatoires du cerveau, 1859. T. ii. p. 522.

‡ Cases of aneurism of the cerebral vessels (Guy's Hospital Report, ser. iii., vol. v. London 1859).

§ Ueber die Erweiterung kleinerer Gefässe (Archiv für path., Anat., und Physiologie, b. iii. p. 442).

‖ Ueber Gefässentartungen in der Varolsbrücke und den Gehirnschenkeln (Allgemeine Wiener Wochenschrift, 1864, No. 28).

¶ Die Capillar-aneurysmen im Pons Varolii (Wiener medicinische Wochenschrift, 1865, 6 & 9 Septr.)

the description and the drawings. We then examined at the autopsies to see whether similar aneurisms could be found, and we frequently met with them in those who had died of other than brain disease. Fresh cases of cerebral hæmorrhage presented themselves, and the brains of these always contained miliary aneurisms. Since then we have been able, in numerous communications to the Société de Biologie, to which the first case was reported, to show that in a certain number of cases of cerebral hæmorrhage, the effusion results from the rupture of a miliary aneurism. As case after case occurred without our having once failed to detect these aneurisms, we were enabled to state our opinions in a more categorical manner. Our observations being made in an hospital devoted to the reception of old people, we had met with hæmorrhage only in those advanced in life, since our attention was directed to the search for aneurisms. This circumstance imposed on us a certain reserve. We were obliged to confine ourselves to the statement that *senile* hæmorrhage results from the rupture of miliary aneurisms. It was thus that our opinion was expressed in the communications which we made in common to the Société de Biologie, and more recently in separate publications. But since then numerous facts have been collected; many observers have criticised and confirmed our observations, and these aneurisms have constantly been met with, not only in senile hæmorrhage, but in the cerebral hæmorrhage of adults, and even of young people. We believe, therefore, that we are now in a position to affirm in a general manner that cerebral hæmorrhage results from the rupture of miliary aneurisms; that the true prehæmorrhagic lesion (*lesion hæmorrhagipare*) consists in the production of these aneurisms. This article will be devoted to the demonstration of that proposition.

II. We do not for a moment mean to say that all cases of cerebral hæmorrhage, without exception, should be attributed to the rupture of an aneurism. Fracture with depression, which causes effusion into the substance of the brain, assuredly requires no such intervening agency. Neither is such an explanation required for the hæmorrhage which accompanies cerebral softening in cases in which thrombosis of the sinuses impedes the return of the blood. Lastly, the cerebral hæmorrhage which has been observed in certain fevers and some infectious diseases, and that which occurs in the course of certain depraved states of the system (*dyscrasies*) are, no doubt, produced in another manner. But, besides these exceptional cases, in

which the hæmorrhage supervenes as a symptom or complication of another disease, there are other forms of hæmorrhage, much the most frequent in their occurrence, which have their own independent character and special lesions, and which all result from the rupture of miliary aneurisms. They constitute almost the terminal event, the extreme limit, of a separate disease which invades the arterial system of the brain, a disease which leads to the formation of aneurisms, and finally to their rupture. This is cerebral hæmorrhage *par excellence*,—hæmorrhage which, of very frequent occurrence in old age, is not exceptional in middle life, and may be met with in youth. It might be called *the disease, cerebral hæmorrhage.*

The view which we take is now supported by a considerable collection of facts; our statistics include not less than sixty cases of cerebral hæmorrhage; of this number forty-two have been collected in the Salpêtrière. This is a number which may occasion surprise, especially if it is considered that in so large an hospital as the Charité of Berlin, only twenty-eight cases of cerebral hæmorrhage appear in the post mortem books during a period of three years.* But it must be borne in mind that our observations were made in an hospital set apart for old people, and that it is senile hæmorrhage which has furnished most of the cases which we relate.

The constant presence of miliary aneurisms in so many cases of cerebral hæmorrhage might give rise to a supposition against which we must first of all guard ourselves. One might think that these aneurisms form, if not a constant, at least a very frequent, lesion in old age; that they result from a senile change in the vessels of the brain, in the same way as atheroma, so that they might exist as a pure coincidence in the brains of those who die of cerebral hæmorrhage. This hypothesis is weakened by the fact, that when one takes the trouble to look for miliary aneurisms in the walls of a recent cavity, they are always found there, and are seen to be ruptured and filled with a clot which is continuous with that of the effusion. But we may add that we have met with them at all periods of adult age, from the twentieth year upwards, and only in individuals who had died of cerebral hæmorrhage; whereas, in old age, though constant in brains which contain recent or old hæmorrhagic cavities, they are very rare in those who, on post mortem examination, present no lesion of the nervous centres, or rather, in

* Eulenburg. Ueber den Einfluss von Herzhypertrophie und Erkrangungen der Hirnarterien auf das Zustandekommen von hæmorrhagia cerebri (Arch. f. path. Anat. und Phys., 1862. P. 356).

those in whom no traces of hæmorrhage are found. Nevertheless, we have met with them in such cases; they prepared the way for a hæmorrhage which, though imminent, had not yet taken place.

We cannot tell the exact proportion in which they occur at each age in those who present no trace of cerebral hæmorrhage; but, though their frequency may increase with the increasing age of the patients, we can at least affirm that they are always exceptional. Heschl,* who has pursued this statistical inquiry with more care than we have been able to give it, and who has met with miliary aneurisms sixteen times,—without, however, suspecting the relation which they might have to cerebral hæmorrhage,—arrived at the following results:—In 140 autopsies, made on persons from 40 to 50 years of age, he found them five times, or once in 28; in 133 examinations of individuals from 50 to 60 years of age, he found them five times, or once in 26; in 83 examinations of individuals from 60 to 70, he found them three times, or once in 27; in 38 examinations of patients from 70 to 80 years of age, he found them twice, or once in 19. It is seen that, after 40, the rate of development of the miliary aneurisms is almost a gradually increasing one. Before 40, these aneurisms are much more rare. In more than 800 autopsies, made on individuals under that age, Heschl met with these aneurisms only once; it was in a young man whose age could not be precisely ascertained, but who was between 20 and 30. M. Mayneut had already met with them in a young man of 24. We shall relate further on the case of a young man of 20, in whom they were also found.†

It is noteworthy, that this frequency of the occurrence of miliary aneurisms according to age, bears a close relation to what we know of the frequency of cerebral hæmorrhage at different periods of life.

* Loc. cit.

† The case here referred to was that of a young man of intemperate habits, who, after a copious repast, suddenly fell down insensible, and died in 20 minutes. At the autopsy, the optic thalamus and corpus striatum of the left side were found to be ploughed up by an effusion of blood, which had also torn its way into the ventricles: the clot extended into the fourth ventricle. The arteries of the base presented some soft whitish patches. The heart was the seat of an enormous concentric hypertrophy. Sigmoid valves slightly sclerous, but not constricted or incompetent. The other organs were not examined.

The parts relating to this case were presented to the Société Anatomique, by M. Bremond, interne de l'hospice de Charenton. A part of the brain was given to MM. Charcot and Bouchard for examination, and they found in it, besides a very marked sclerous peri-arteritis, many miliary aneurisms, some of which were very large.

To be sure we cannot place side by side with this table of the frequency of aneurisms a comparative statistical statement regarding the distribution of cerebral hæmorrhage at different periods of life; we do not know, even approximately, to what extent cerebral hæmorrhage occurs at each age in a given number of deaths. But a glance at the statements of Rochoux and M. Durand-Fardel* is sufficient to convince us that cerebral hæmorrhage, almost *nil* before 20, is very rare up to 40; that after that age it becomes frequent, and that the proportion in which the ages are not known is yet a gradually increasing one. Taking into account only the cases of hæmorrhage observed before 80, and dividing them according to their ages, it is seen that only $\frac{1}{160}$th occurs in those under 20; $\frac{1}{7}$th in those under 40; and $\frac{6}{7}$ths in those from 40 to 80 years of age.

But it may also be remarked that miliary aneurisms, which rarely occur before 40, and which then gradually increase in frequency with advancing age, only conform to the law of development of arterial atheroma; and, perhaps, one might be tempted to regard these aneurisms as the result of the extension to the small vessels of the brain of that arterio-sclerosis which produces fatty or calcareous incrustations, or even ossification of the larger vessels. Such an opinion would not be correct. Miliary aneurisms may, and often do, exist, sometimes in considerable number, independently of all atheromatous lesion of the arteries of the base or of the branches which are distributed to the membranes, just as the most marked degree of atheroma may very often be met with, without a single aneurism being found in the brain. But these two different changes are not mutually exclusive, they are not antagonistic, but may, and frequently do coexist. Of this one may be convinced by an examination of our cases, and the statistical statement which sums them up. We shall return to this subject, however, when considering the influence of atheroma in the production of cerebral hæmorrhage.

Yet these two changes, essentially distinct, are not devoid of certain analogies: both result from arteritis; but whilst atheroma is the result of an endo-arteritis, miliary aneurisms are produced as a complication of a peri-arteritis, diffuse peri-arteritis, the study of which, it seems to us, ought to precede that of the miliary aneurisms themselves.

* Durand-Fardel. Traité clinique et pratique des maladies des vieillards. Paris, 1854. P. 286.

III. This arteritis which leads to the formation of aneurisms is diffuse; it is met with, not only on the arteriole which is the seat of miliary dilatation, but extends to the entire system of small intra-cerebral vessels, and is sometimes accompanied by a sort of atrophy of the walls of the large arteries of the base, and of those of the meninges, giving them a sort of onion-peel aspect. Studied only in the small vessels of the brain substance, this sclerous arteritis, which is not without certain analogies with what Rokitansky described under the name of chronic peri-arteritis, is characterised by changes in that peri-vascular sheath described by M. Robin, and which since the labours of His has generally been called the lymphatic sheath. It is characterised, besides, by lesions of the adventitious tunic, and also by changes in the muscular and internal coats. The principal changes take place in the most external parts, they progress from without inwards, thus justifying the name peri-arteritis. This change is observed in the arterioles of every calibre, which are situated in the substance of the brain tissue, and which consequently include both varieties of the capillaries of M. Robin, and which are now generally regarded as part of the arterial system.

The lymphatic sheath which envelopes the vessels of both varieties may present only a striated, wavy appearance, like a bundle of subcutaneous cellular tissue; but this appearance is not modified by the action of acetic acid. At the same time, and generally apart from all appearance of waviness, the sheath presents an exaggerated formation of nuclei analogous to those of the neuroglia, spherical or ovoid, slightly irregular in outline, retracting and becoming more distinct under the action of acetic acid, strongly coloured by carmine and fuchsine, much more so than other larger and more distinctly ovoid nuclei which are sometimes found along with them, and which appear to belong to the epithelium of the sheath. The former, which, on the other hand, appear to be of the nature of connective tissue, may be developed in prodigious numbers, and interfere with the proper observation of the subjacent vascular coats. These nuclei are frequently seen to be constricted, a circumstance which would indicate that they multiply by segmentation.

The cavity of the sheath generally presents nothing abnormal, though a few cells and sometimes fatty granules or grains of hæmatoidin may be seen floating in it.

On the principal arterioles, corresponding to the capillaries of

the third order of M. Robin, the adventitious coat may present two different conditions. Sometimes there is simple thickening, imparting to that membrane a thickness which is occasionally equal to the calibre of the vessel. Its substance is striated longitudinally like a bundle of fibrous tissue, and encloses fusiform corpuscles of connective tissue, arranged in the direction of the axis of the vessel. It is then more difficult to study the subjacent tissues (Pl. II. fig. 1, B). At other times, and this is most frequently the case, the change in the adventitious coat may consist exclusively in a multiplication of the connective nuclei, without thickening, and without a fasciculated appearance of the membrane. The rapidity with which these nuclei are produced may deprive them of all regularity in their distribution. A similar activity of development may be observed on the surface of the finer arterioles, corresponding to the capillaries of the second variety of M. Robin.

In connection with this change in the most superficial parts of the artery, is observed a change in the muscular coat, sometimes general, at others limited to certain points. This lesion of the muscular coat consists in a change in the transverse fibres, without fatty degeneration, the result of which is thinning and separation, and at some points total disappearance of these fibres. This simple atrophy of the muscular elements does not appear to be primary, but seems, on the contrary, to depend on the more superficial change formerly described (Pl. II. fig. 1, A, and Pl. III. fig. 1). Indeed, the peri-arteritis is often seen to be limited to the sheath and adventitious coat, the muscular showing no appreciable change; it is also seen that the superficial arteritis is most marked at those points at which the muscular elements are most defective, or even altogether awanting.

If the adventitious coat is not thickened and fibrous, the vessel is dilated in the form of ampullæ, at the points of which the muscular coat is atrophied. This is the origin of the miliary aneurisms.

The only change in the internal tunic (*tunique propre* of M. Robin) consists in a multiplication of the large ovoid longitudinal nuclei of that membrane. This multiplication, which may be recognised even in the true capillaries (capillaries of the first order of M. Robin), seems to be much less marked than it is in the adventitious coat or lymphatic sheath; it may even be altogether awanting. It seems also to be produced by segmentation, the newly formed nuclei having their long axis in various directions, and not

arranged in a regular longitudinal manner as is the case in the normal condition.

Such is the sclerous peri-arteritis which precedes and accompanies intra-cerebral miliary aneurisms. We have always found it occur in different degrees of intensity, and at the same time as the aneurisms, in those whose brains presented either old or recent hæmorrhagic cavities; it always existed in brains, which, without presenting any trace of hæmorrhage, yet contained aneurisms: finally, we have demonstrated its existence in some cases in which there were no hæmorrhagic cavities, and in which no aneurisms could be discovered. Was it that some of these dilatations had escaped our notice? or had we not rather to do with brains in which hæmorrhage was likely to occur in time, but in which that hæmorrhage, far from being imminent, was not even threatened by the development of aneurisms, which, without doubt, would ultimately be formed?

In the absence of other arguments, the description which we have just given would prove that the formation of the miliary aneurisms is not to be attributed to the ordinary process of atheroma, to sclerous endo-arteritis. We have not followed the histological changes in the large trunks of the base or meninges, but, as has been said above, these vessels appear to participate in the disease, which is not wholly intra-cerebral. Independently of the thinning and of the onion-peel aspect already referred to, we would also cite, in support of this view, what has several times been demonstrated, the coexistence in the small arteries of the pia mater of that ampullar dilatation described by Virchow,—a condition which has such close analogies with our aneurisms, that it seems to differ from them only in the situation and pathological signification of the change. In some cases, too, we have seen miliary aneurisms complicated with much larger aneurisms scattered over the important branches of the meningeal arteries. We once saw, in the same circumstances, a fusiform and cirsoid dilatation of the basilar artery, with a similar change of the left sylvian at its origin.* The coexistence of these different lesions has led us to think that the sclerous peri-arteritis, which we have just described, is not exclusively confined to the intra-cerebral vessels; we regard it as a general change of the encephalic arterial system, but nothing warrants us in looking on it as a more general affection. We have never met with analogous vascular changes in other parts of the body.

* See case III.

IV. We must now enter on the study of the miliary aneurisms. The numerous fresh cases which we have observed fully confirm the description which we formerly gave; we shall not, therefore, have to modify it much. Before pointing out the structure of these aneurisms, we shall first describe the appearance which they present, and the parts at which they occur.

All these aneurisms are visible to the naked eye; they look like small globular particles, varying in diameter from $\frac{2}{10}$ths of a millimetre to one millimetre, and sometimes even a little more, attached to a vessel which is also visible to the naked eye; a simple lens at least suffices to distinguish it clearly; the diameter of the vessel may vary from a third of a tenth of a millimetre, to a fourth of a millimetre. The colour of these aneurisms varies according to the state of the blood which they contain, and the condition of their walls. When the wall is thin, the aneurism is of a more or less deep violet red colour, if the contained blood is liquid; if, on the contrary, the blood has been long coagulated, and is already transformed into fatty granules and hæmatoidin, the aneurism is reddish brown, ochrey, or even blackish; and generally the layer of cerebral tissue immediately surrounding it presents in a less degree a similar coloration. When the walls are thickened by increase in the adventitious coat, as is sometimes the case, especially in the central portions, the aneurism is bluish, if the contained blood is liquid; grayish, if the white corpuscles, which always remain to some extent, are numerous and confined in a granular stroma (*gangue*) which leaves only a limited space for the normal blood; brownish if the contents have already undergone the retrograde transformations pointed out above; and yellowish if there is a predominance of fatty granules or calcareous particles. These differences in the structure of the envelope and in the nature of the contents may cause considerable variety in the consistence of the aneurism, which is sometimes soft and fragile, giving way under the least pressure, and permitting the escape of blood whose external characters are little changed; at others, hard like a grain of sand; and at others firm and elastic.

We have met with these aneurisms in almost all parts of the brain; but they are not uniformly spread throughout; they have their organs of predilection. The different parts of the brain in which we have met with them are, in the order of decreasing frequency, the optic thalami, the corpora striata, the convolutions, the pons Varolii, the cerebellum, the centrum ovale, the middle peduncles of the cerebellum, the peduncles of the brain, and the bulb. Is not

this distribution yet another argument in support of the pathological influence which we attribute to them? In eighty-six cases of hæmorrhage observed by M. Durand-Fardel, the effusion was primarily produced fifty times in the corpora striata or optic thalami; twenty-seven times in the centrum ovale, or in the neighbourhood of the convolutions, or in their substance; five times in the pons; four times in the cerebellum.

It was in the convolutions that the miliary aneurisms were first observed by us, and for a length of time it was almost exclusively in that situation that they were met with; they were situated either on the surface or in the substance of the gray matter, or at the junction of the white substance with the cortical layer. This more superficial position of the aneurisms of the convolutions, by bringing them into notice as soon as the pia mater was detached, led us at first into an error regarding their relative frequence; but, by-and-by, when we got into the habit of looking carefully for them in the central portions of the brain, we found that they were of more frequent occurrence in those parts.

The number of aneurisms in a given brain is very variable, and cannot be accurately estimated, as it is impossible to cut the organ into pieces so small that a good many may not escape notice. Notwithstanding the most minute care, we have sometimes discovered no more than two or three; generally a much larger number is found; sometimes even hundreds may be counted in one brain.

It has already been said that they are chiefly met with in old people, but we have also pointed out that they are not peculiar to old age, as we have seen them occur, along with the sclerous arteritis which paves the way for them, in a young man of 20; and there are on record three cases in which they were found in individuals from 20 to 30 years of age.

When the membranes are stripped off a brain which contains these aneurisms, it frequently happens that they are seen at first on the surface of the convolutions, or in the fissures between them. They then have the appearance of small round points, slightly elevated, and of the size of a millet seed, scattered about in variable numbers, sometimes very numerous; they are variously coloured, from a bright red or violet to reddish or blackish brown; some are even gray or yellowish (Pl. II. fig. 2, A B). Examined more closely, each of these spots is seen to be formed of a particle of varying resistance, and more or less regularly spherical. By means of needles they may be separated

from the surrounding nervous tissue in which they are embedded as in a cup. Sometimes it is more difficult to isolate them in consequence either of the very great delicacy of the membrane of the aneurism, or of its adherence to the surrounding tissue. When the dissection is complete, the granule is seen to be continuous by one of its poles with a vessel which dips into the substance of the convolution.

When the patient has succumbed to a recent attack, larger particles are frequently found, often, indeed, bigger than a grain of hempseed; they are of a violet colour, and the blood which distends them is covered only by a very thin membranous pellicle. If the blood is then expelled by pressing the globule between two plates of glass, there is seen in the centre an aneurism similar to the rest, but ruptured, and surrounded by the blood which distended the enveloping membrane, and which is no other than the lymphatic sheath; so that where it was believed that we had to do with a large aneurism, there was really only an effusion of blood into the dilated sheath, with a true aneurism in the centre. In cases in which this sheath is ruptured, the appearance which we have just described no longer exists, and there is a true hæmorrhagic cavity.

When the aneurisms are completely buried in the gray substance of the convolutions, they may often be dimly seen through the overlying brain tissue like bluish ill-defined spots, giving an appearance of marbling to the free surface of the convolution.

The same appearance is produced on the surface of a section of the brain by aneurisms of the deeper parts; but, generally, their investigation demands more attention than does that of aneurisms of the convolutions (Pl. II., fig. 3, B C D).

So far this investigation presents very little difficulty, and does not require very much time. But it is not so when we come to look for the aneurisms which ought to be found on the walls of recent cavities. The cavity must first be cleaned, and a large part of the clot removed; but in order to do this properly, the blood should neither be removed with the back of a scalpel, nor washed away by a stream of water, nor by means of a brush. These various means, which have all been employed in searching for the ruptured vessel where it was expected to find it, are all defective. The process which has appeared to us to furnish the best results is the following:—After having freely opened the cavity, and allowed to escape so much of the clot as does so of its own accord, the brain should be placed in water, which should be frequently renewed, without shaking, simply by inclining the vessel from time to time,

so as to detach piecemeal the clot which is still adherent to the walls. After a time, which varies in different cases, there are isolated a considerable number of small bloody masses, which float on the surface of the cavity, but remain attached to it by vascular filaments. It is on these little masses that the microscope should be brought to bear. A good many of them may be examined before an aneurism is found. Most of them are only fragments of the clot, covering as with a hood the end of the ruptured vessels (Pl. IV. fig. 2); but at length some are found in which the vessel, on being followed through the fibrinous mass, is seen all at once to be dilated in the form of a spherical ampulla still containing some blood in its interior, but split on one side; and it is then found that the sheath dissected, or rather injected, by the effusion, is likewise ruptured, so that the clot of the cavity, the clot which distends the sheath, and the clot which is still within the aneurism, are continuous with each other (Pl. IV. fig. 1). It is evident that microscopic examination, which should only be made with a low power, and which is not requisite for the determination of aneurisms away from the clot, is indispensable in the investigation of those which may have caused the effusion.

When an aneurism is isolated in a state of integrity, and examined with a higher magnifying power, its walls are seen to be continuous with, and separated by no line of demarcation from, the coats of the vessel which runs into and emerges from it; but these tunics are no longer distinct from each other. The muscular coat, which is deficient to a greater or less extent on the arteriole on either side of the aneurism, is absolutely awanting on the aneurism itself, so that the adventitious coat is in contact with the internal. Indeed, these two coats are so thoroughly blended together that they cannot be separately distinguished; only one membrane can be detected, on the surface of which may be seen numerous nuclei, or, more often, fusiform corpuscles of connective tissue. The sheath, which may remain distinct, is sometimes also blended with the wall of the aneurism.

In the interior may be seen the blood, the normal elements of which appear to be mingled with granular masses. On pressing the plate of glass which covers the preparation, this blood flows away through both ends of the artery, or escapes suddenly by rupture of the aneurism. It may then be studied more thoroughly. Along with a more liquid portion resembling normal blood is seen another portion less fluid, granular, transparent, and containing very numer-

ous white corpuscles, some of which are swollen and granular, like the bodies of Gluge ; mingled with these may also be found globules and granules of fat. The accumulation of white globules and the amorphous granular matter may, by their increase, produce stagnation of the blood in the aneurism ; this blood then undergoes more fully the retrograde changes by which it is transformed into fatty granules, and into granules or crystals of hæmatoidin. The aneurism is obliterated along with the vessel which bears it, and, for a certain distance in the interior of the latter may be seen fatty particles and crystals of hæmatoidin. Whilst these changes in its contents are taking place, the walls of the aneurism, and of the vessel in the neighbourhood of the aneurism, may be thickened by increase in the adventitious coat, and infiltrated with fatty granules. In cases in which a previously ruptured aneurism has allowed the blood to distend the sheath without rupturing it, and, consequently, without forming a true cavity, fat and grains or crystals of hæmatoidin are found, not only in the interior of the aneurism, but also diffused around it, and the products of the metamorphosis of the blood infiltrate a tissue of new formation, resulting from the blending of the sheath with the aneurism, and the nuclear increase in the surrounding nervous tissue. Sometimes, indeed, capillaries are developed in considerable numbers on the surface of the shrivelled particle, and seem to form *vasa vasorum*.

Such is the constitution of miliary aneurisms. These ampullar, or sometimes only fusiform, dilatations are generally produced at the expense of the whole circumference of the vessel ; at other times they are lateral, and do not completely interrupt the calibre of the vessel. These lateral expansions may be pedunculated. Generally an arteriole bears only one aneurism, but sometimes many groups of them may be seen forming a sort of cluster suspended from one vascular filament ; at others, the vessel presents many successive dilatations, and assumes a moniliform aspect.

Having described the aneurisms themselves, we must now study them in their relation to cerebral hæmorrhage. We shall first give the cases of cerebral hæmorrhage which we have collected since our attention was directed to the investigation of these aneurisms. These cases may be divided into three groups : those in which the hæmorrhage caused death a short time after the attack, and in which the brain showed only recent cavities ; those in which recent extravasations coexisted with ochrey traces of old sanguineous apoplexy ;

and finally, those in which there was found at the autopsy only the distinctly recognisable cicatrix of a former hæmorrhage.

V. [The authors then proceed to give, in an abridged form, the notes of a large number of cases observed by themselves, and communicated to them by other observers, chiefly MM. Vulpian, Barth, and Behier, of the Salpêtrière, Hotel-Dieu, and Pitié hospitals. They state that the examination of the lesion was always most carefully made, and that in all their own cases, and in most of those communicated to them, the existence of the aneurisms was verified, not only by the naked eye, but also by the microscope.

It is unnecessary for me to reproduce these cases. Suffice it to say that there are recorded seventy-seven cases in which these miliary aneurisms were found in various parts of the brain : in twenty-nine of these there were only recent cavities ; in seventeen there were both recent extravasations and traces of old cavities ; and in thirty-one there were only the traces of old hæmorrhage.

The authors give in addition seven other cases recorded by Cruveilhier, Gull, Heschl, Hayem, and Bastian, whose details warrant the inference that they were cases in which cerebral hæmorrhage co-existed with miliary aneurisms, although the true nature of these aneurisms was not recognised or even suspected.

The authors further state that they refrained from giving cases which were in the least degree doubtful, or in which the nature of the lesion was not perfectly evident. As examples of the care and accuracy of their statistics they give three cases, one observed by M. Charcot, and two by M. Vulpian, in which old hæmorrhagic cavities were believed to co-exist with miliary aneurisms, but which were excluded from their statistics because it was not quite certain whether the cavities resulted from hæmorrhage or softening.]

In the fact that they resulted from one or the other of these conditions, the authors proceed to say, there is nothing very exceptional. Aneurisms pretty often co-exist with arterial atheroma, and hence they may also exist along with cerebral softening. It is therefore not surprising that they should be met with in brains which present no trace of hæmorrhage, but in which there is found a patch of softening. We have seen many examples of this, and others have been communicated to us by M. Vulpian ; and it was doubtless such a coincidence that existed in a case published by M. Dieulafay.* The lesion, at first regarded as cerebral hæmorrhage, was, after a dis-

* Gaz. des Hôpit., 31 Mars 1868. Hémorrhagie au niveau de la scissure de Rolando.

cussion to which it gave rise at the Anatomical Society, looked upon as only a case of red softening.

Miliary aneurisms have been seen to occur along with other lesions of the nervous centres. Thus, in a case observed by M. Liouville, in the wards of M. Vulpian, they were found in considerable numbers in the brain of a subject who had some pretty large aneurisms on the meningeal arteries, and thick very vascular false membranes in the dura mater. In a case communicated to us by M. Vulpian, a woman aged 41, affected with progressive locomotor ataxy, presented at the autopsy, along with sclerosis of the posterior pyramids of the medulla, a considerable number of intra-cerebral aneurisms.

We have said in the first part of this memoir that we were not warranted in regarding these aneurisms and the peri-arteritis which gives rise to them as part of a change extending to the whole vascular system. Some facts observed by M. Liouville would tend to modify that opinion. He has seen, along with intra-cerebral aneurisms, similar aneurisms in the œsophagus and heart. He has kindly shown us his preparations, and we have been able to satisfy ourselves as to the perfect identity of these last aneurisms with those which are developed in the brain.

We would also remark that many cases which have come under our own observation, or have been communicated to us by other observers, lead us to believe that the larger aneurisms of the viscera may co-exist with miliary aneurisms.

On recurring to the eighty-four cases of cerebral hæmorrhage which we have collected (and which, as already explained, have been omitted by the translator), it will be seen that miliary aneurisms were found in every one of them, or in 100 per cent.

Other anatomical conditions regarded as capable of playing a part in the production of cerebral hæmorrhage may be met with in them, but much less frequently. Thus, atheroma of the arteries of the brain, to which a preponderating influence has been attributed, no longer occupies the first place in our statistics.

The condition of the arteries of the base was noted sixty-nine times. Out of this number the arteries were not at all atheromatous in fifteen, or in 21·74 per cent. more than one-fifth of the cases; they were very slightly atheromatous in twenty-five, or in 36·37 per cent., more than one-third of the cases; they were simply noted atheromatous in twelve, or in 17·39 per cent.; lastly, they were very atheromatous in seventeen, or in 24·63 per cent., scarcely one-fourth of the cases. It may, therefore, be said that the arteries of the brain

are not atheromatous in more than one-fourth, or scarcely 22 per cent. of the cases which die of cerebral hæmorrhage, and that in almost three-fourths of these the atheroma exists in different degrees. This frequent occurrence of apparent integrity of the arteries of the brain in those who present recent or old sanguineous cavities is not much in keeping with the opinions usually held, and this result differs considerably from the figures given in former statistics. M. Durand-Fardel,* who collected thirty-two cases of cerebral hæmorrhage in which the condition of the arteries of the base was noted, found them intact in twelve per cent. In a statistical inquiry (referred to at page 42), based on thirty-nine cases of hæmorrhage, we found that the arteries appeared healthy in eighteen per cent. The proportion of cases of cerebral hæmorrhage in which the arteries of the brain are not atheromatous, is almost the same as the proportion of cases in which the arteries are found to be healthy in old people not affected with brain disease. According to the statistics of M. Durand-Fardel, the arteries are free from atheroma in twenty-eight per cent. of those over sixty years of age. In those who die of sanguineous apoplexy the cerebral arteries are free from atheroma in twenty-two per cent. The difference, it will be seen, is very slight, and so insignificant that the arterial incrustations seem to play only a very secondary part in the production of the extravasation.

Let us pass to the consideration of another pathological condition, hypertrophy of the heart, referred to by Legallois, Corvisart, Bricheteau, Bouillaud, Ménière, Rokitansky, Leubuscher, &c. In our cases the state of the heart was noted fifty-five times ; hypertrophy was found only twenty-two times, that is, in forty per cent.; but in two of the cases this hypertrophy was compensatory of valvular lesions, which brings to 36·36 per cent. the proportion of cases of simple hypertrophy of the heart, the only form which could increase the tension of the blood in the arterial system. It is, therefore, only a very secondary cause in the production of cerebral hæmorrhage.†

We would say the same of atrophy of the kidneys, which in its turn has been referred to as one of the causes of cerebral hæmorrhage, that it may or may not be accompanied by secondary hypertrophy of the heart. In our cases the state of the kidneys was noted forty-nine times, and in only sixteen, or 32·65 per cent., did these organs present any lesion capable of embarrassing the circulation and increasing the tension of the arterial system ; there was simple atrophy

* Durand-Fardel. Traité clinique des maladies des vieillards, p. 228.

† See foot notes at pages 7 and 28.

thirteen, or in 26·53 per cent. of the cases, and interstitial or parenchymatous disease in three, or 6·12 per cent.

Of all the organic conditions capable of playing a part in the pathology of cerebral hæmorrhage, one only, by reason of its constant presence, appears to us worthy to be regarded as the real cause of sanguineous extravasation; that one is the existence of miliary aneurisms.

This conclusion, to which we have been directly led by the observation of numerous facts, might indirectly be deduced from a consideration of the inadequacy of the hypotheses hitherto advanced in explanation of the mechanism of cerebral hæmorrhage.

EXPLANATION OF PLATE I.

Fig. 1.—Sclerous condition of the small arteries of the brain.

a, Arteriole whose muscular fibres have disappeared, and on which numerous nuclei have been produced ; *b*, lymphatic sheath, likewise studded with nuclei.

Fig. 2.—Aneurisms of the convolutions.

a, An arteriole whose lymphatic sheath is blended with the aneurism ; *b*, thickened wall of the aneurism ; *c*, contents of the aneurism undergoing retrograde metamorphosis.

Fig. 3.—Aneurism from a corpus striatum.

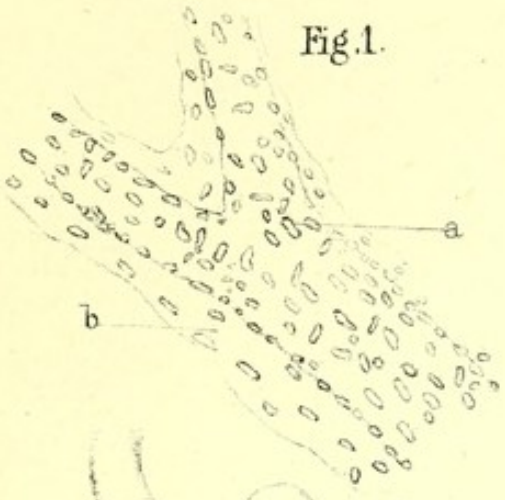
Fig. 4.—Arteriole from an optic thalamus having on it three aneurisms, one of which has given way under pressure, and permits its coagulated contents to be seen.

Fig. 5.—*b*, Aneurism ruptured in a hæmorrhagic cavity ; *c*, the clot, after having distended and torn the lymphatic sheath (*a*), is blended with the mass of blood enclosed in the cavity.

Fig. 6.—Vessel secondarily ruptured in a recent hæmorrhagic cavity and capped by a clot, which at first was mistaken for an aneurism.

[Plate I. belongs to M. Bouchard's work ; plates II., III., and IV. to the appendix. They were drawn from nature by M. Charcot.]

Fig.1.



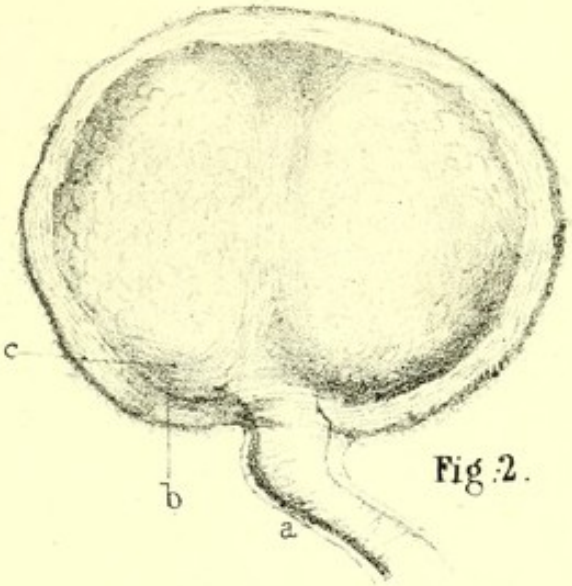


Fig.2.

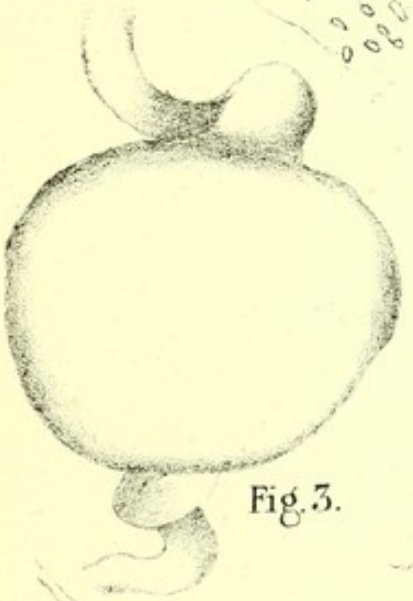


Fig.3.

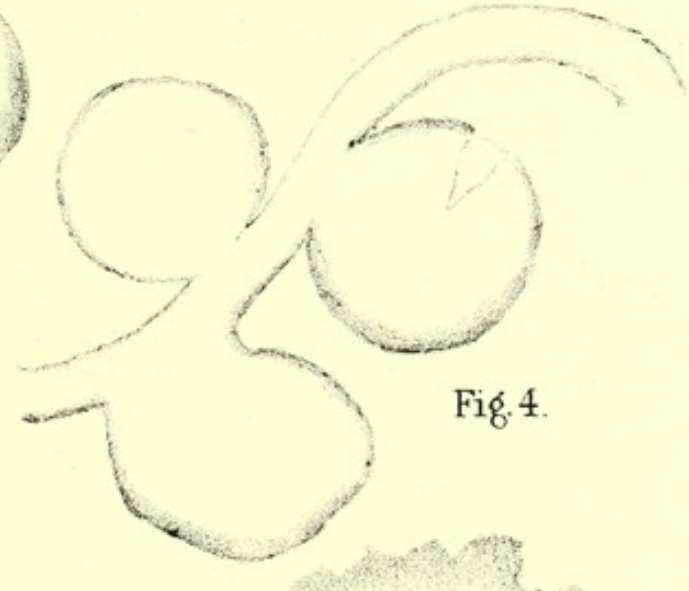


Fig.4.

Fig.5.

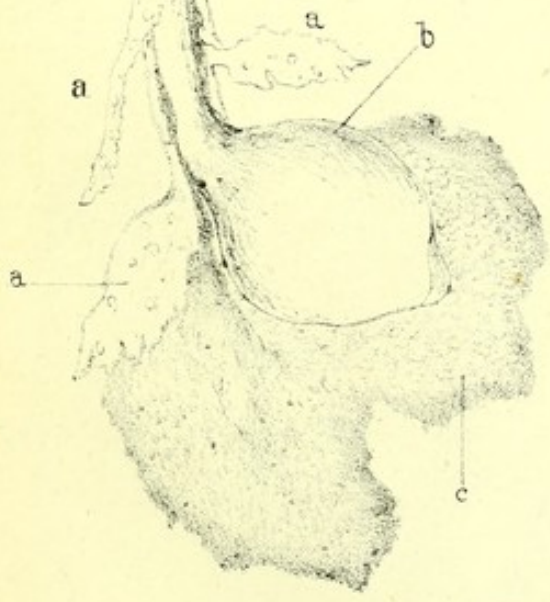


Fig.6.

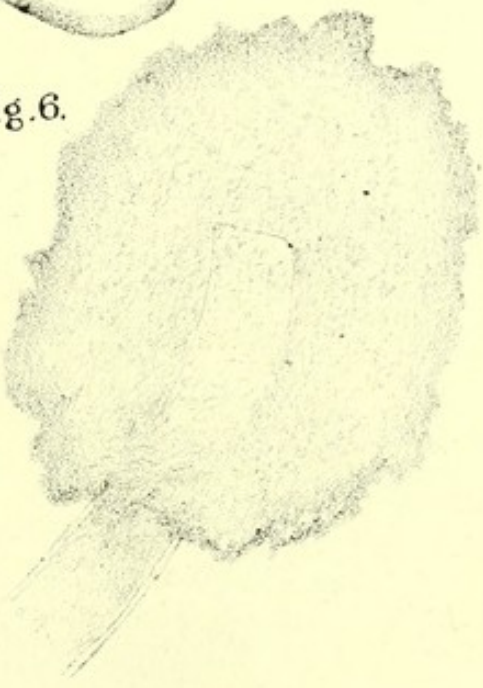


Fig.1.

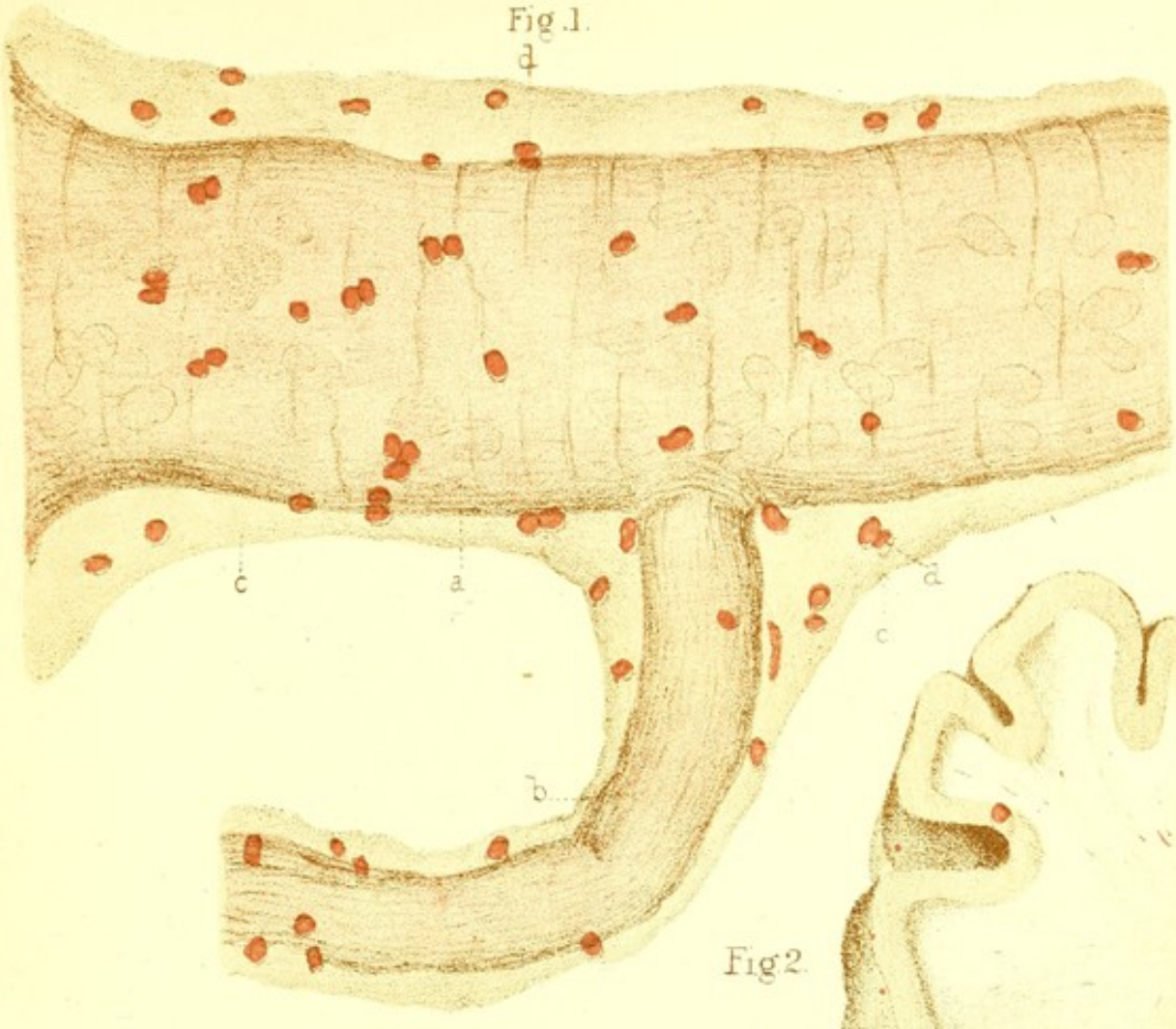


Fig 2.

Fig.3.

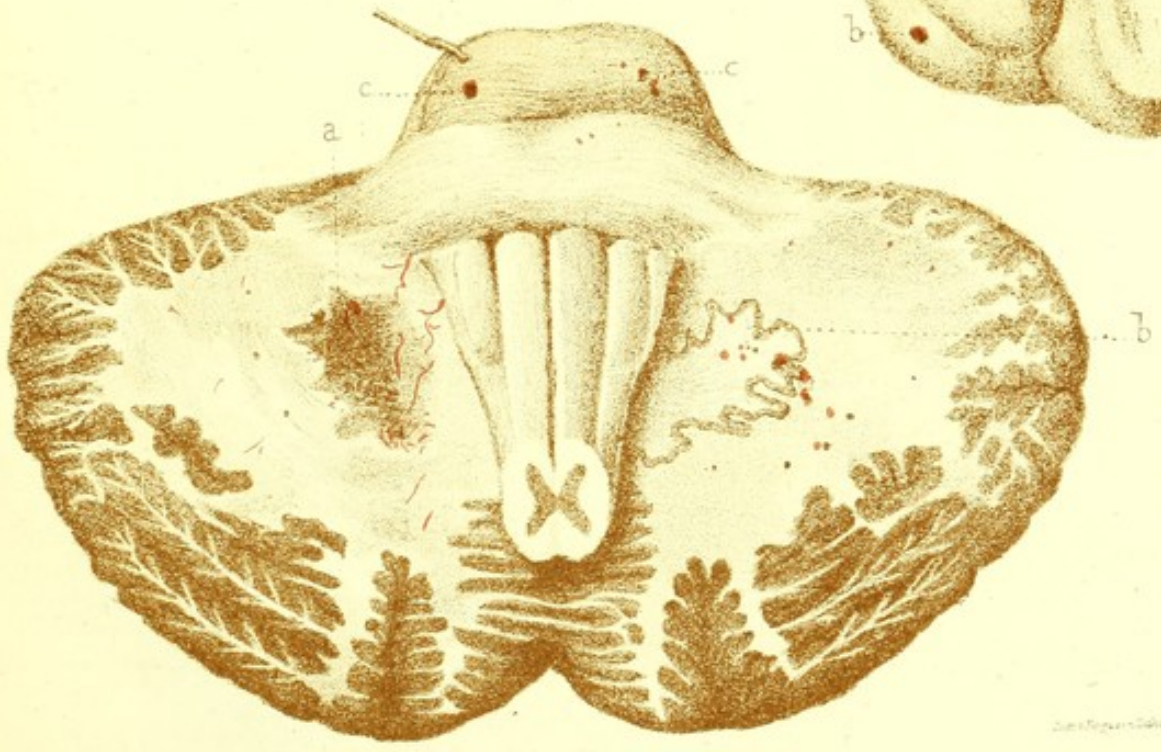


Fig 1

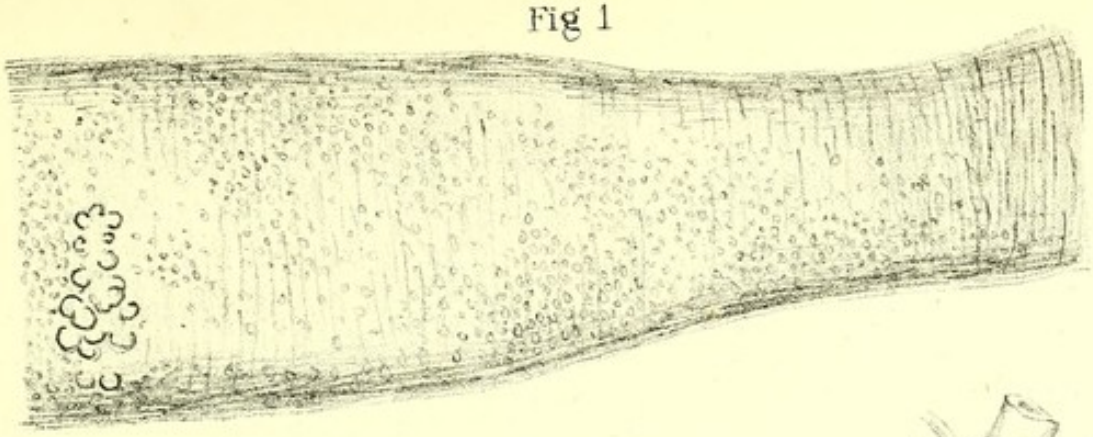


Fig.2.

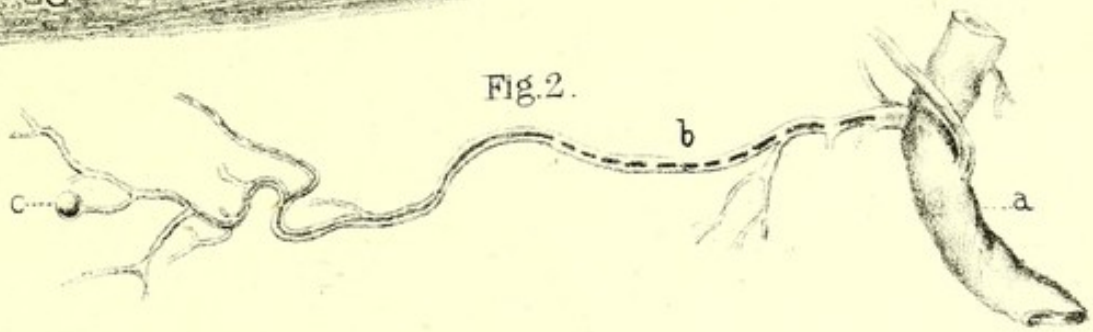
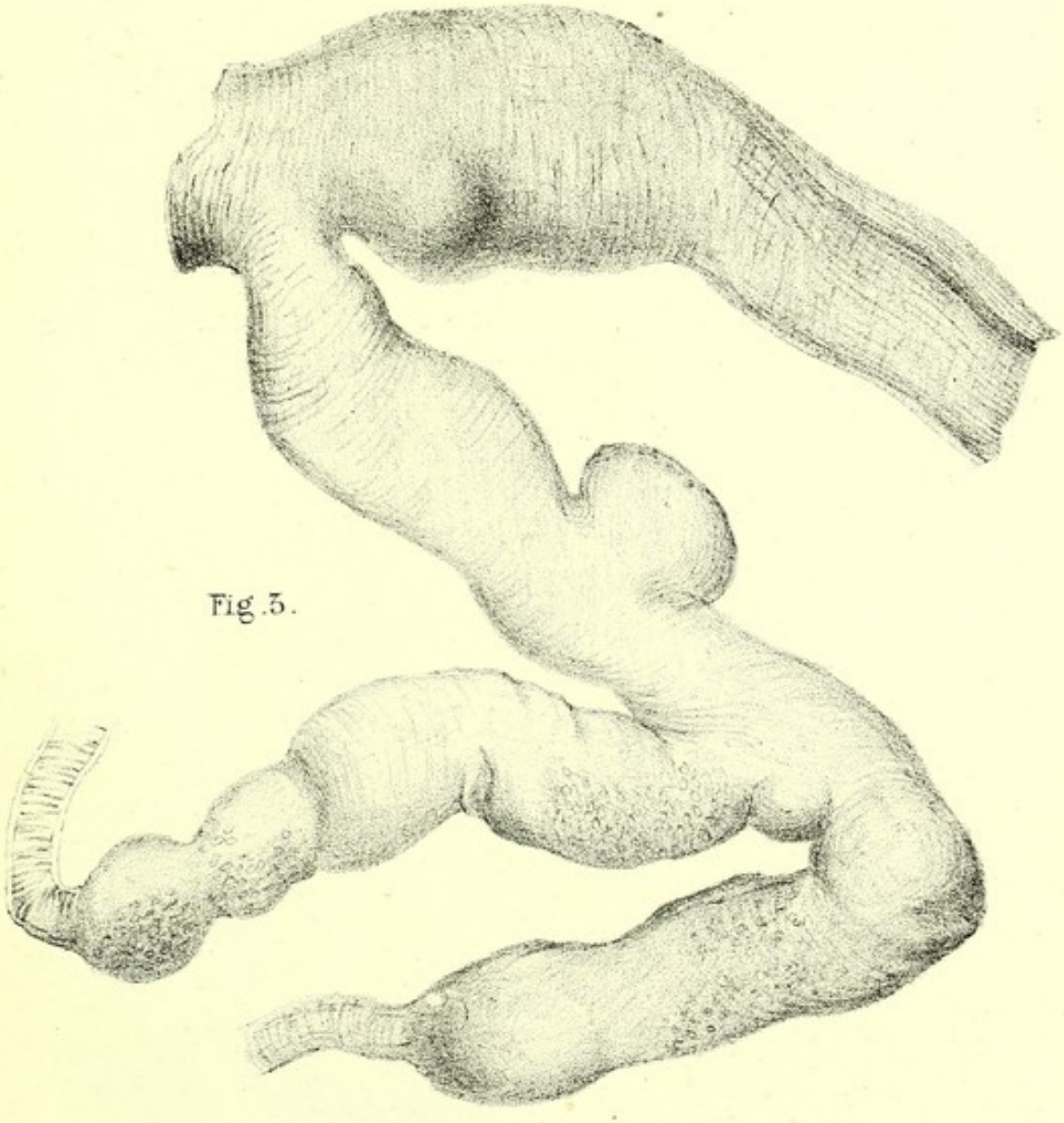


Fig.3.



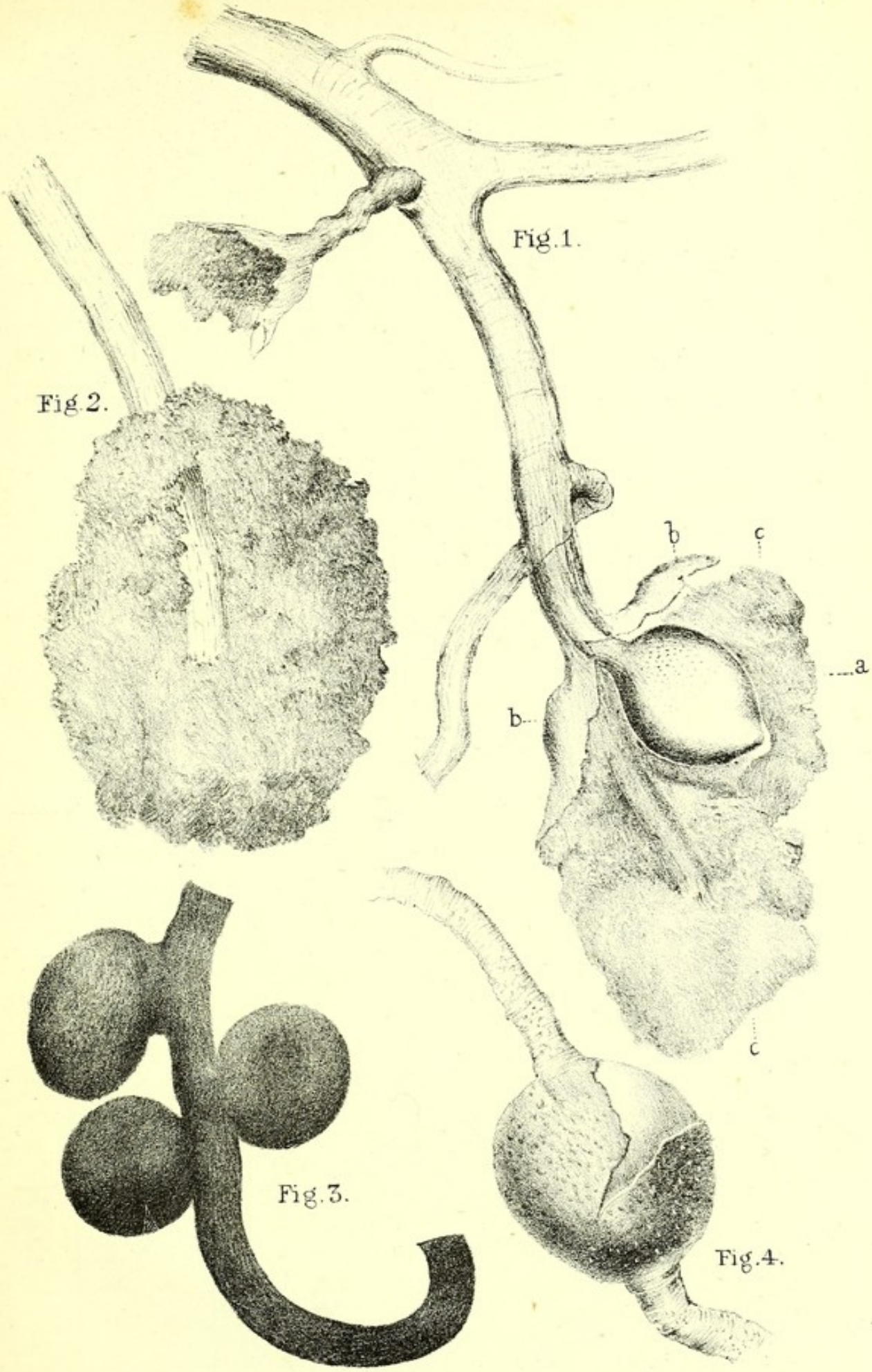


Fig. 1.
Fig. 2.
b
c
a
b
c
Fig. 3.
Fig. 4.