A practical treatise on apoplexy (cerebral hemorrhage) : its pathology, diagnosis, therapeutics, and prophylaxis ; with an essay on (so-called) nervous apoplexy, on congestion of the brain and serous effusion / by William Boyd Mushet.

A PRACTICAL

TREATISE ON APOPLEXY

(CEREBRAL HEMORRHAGE);

ITS PATHOLOGY, DIAGNOSIS, THERAPEUTICS,

AND PROPHYLAXIS:

WITH AN ESSAY ON

(SO-CALLED) NERVOUS APOPLEXY, ON CONGESTION

OF THE BRAIN AND SEROUS EFFUSION.

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Δύο γὰρ ἐπιστήμη τε καὶ δόξα, ὧν τὸ μὲν ἐπίστασθαι ποιέει, τὸ δὲ ἀγνοεῖν.

ΙΠΠΟΚΡΑΤΟΥΣ ΝΟΜΟΣ.

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PREFACE.

I HAVE attempted to extricate Apoplexy as a substantive disease from an assemblage of symptoms, *i.e.*, from the multiform phases of coma. I am strongly impressed that the main obstacle to a proper and simple understanding of the affection has been its confusion with every malady attended by unconsciousness, irrespective of pathological conditions; coma (the order) and apoplexy (the genus) having been almost invariably regarded as metonyms, loosely expressing a deeper or more pronounced degree of cerebral torpitude than their obsolete and less definite congeners—carus, cataphora, and lethargus.

It may be my doctrines are extreme, my efforts too decided in the direction of simplicity and unity. My argument also, I fear, is short of convincing. My views are based on experience, on practice; and I entered on the subject free from bias or preconception, the constancy of cardiac lesions first suggesting, and, indeed, instigating the inquiry.

From the nature of the task, the references are necessarily considerable, yet I have repressed their

bulk as much as possible, and from this deterrent action have, in some instances, waded through voluminous works, without appropriating a single passage.

I launch my conclusions on the troublous sea of medical controversy, keenly conscious that they are but imperfect and tentative, yet not without expectation that they will, in some degree, be acknowledged by succeeding physicians, and tend to emancipated and more enlightened opinions on physio-pathology and therapeutics.

W. B. M.

85, St. George's Road, Warwick Square, S.W.

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PART I.

APOPLEXY.

(CEREBRAL HEMORRHAGE.)

CHAPTER I.

DEFINITION.

According to Drs. Copland* and Hughes Bennett,† whose language almost coincides, apoplexy is "a loss of consciousness, feeling, and voluntary motion, or, in other words, a suspension of the functions of the brain—respiration and circulation being more or less disturbed." This definition closely accords with that of Cullen, "Motus voluntarii fere omnes imminuti, cum sopore plus minusve profundo, superstite motu cordis et arteriarum."‡ The former is the more comprehensive, yet neither will alone enable us to distinguish apoplexy from numerous affections of the brain and nervous system; for example, from epilepsy, uræmia, severe cerebral concussion, narcotism, and other organic and dynamic disturbances. In fact, they are little in advance of the description of the old Cappadocian, who tersely informs us that ἀποπληξίη μὲν ὅλου τοῦ σκήνεος, καὶ τῆς αἰσθήσιός, τε καὶ γνώμης καὶ κινήσιός ἐστι παράλυσις.§

It may be urged that it is frequently difficult or impossible to diagnose the disorders I have mentioned

* Dictionary of Medicine, art. Apoplexy.

† Library of Medicine, art. Apoplexy.

‡ First Lines of the Practice of Physic.

§ Aretæi De Causis et Signis Morb. Diuturn. lib. I. cap. VII.

from a true apoplectic seizure, and that the foregoing formulæ are, therefore, sufficiently exact; but it will be conceded that almost any known disease may be confounded with others, differing in their pathology and essential characters. A precise definition is, in consequence, exceedingly important, as *apoplexy* should be regarded as a disease, not a term; thereby differing from mere comatose or apoplectiform symptoms. Dr. G. B. Wood, of Philadelphia, is of the same opinion, and his definition much resembles the following, which is, I think, not open to the above objections.

Apoplexy may be defined as a *more or less sudden impairment of the functions of the brain and nervous system—of consciousness, motion, and sensation—from extravasation of blood into the substance, or upon, or between the membranes, of the brain, arising from internal causes.* This, of course, excludes simple congestion, serous effusion, traumatic apoplexy, and the (so-called) nervous apoplexy of certain authors; but as I contend that these are not *idiopathic* states, or, strictly speaking, not apoplectic, I shall at present confine myself to the subject of primary, sanguineous, or hemorrhagic apoplexy, which is in conformity with the views of Cruveilhier, Rochoux, and Littré, who restrict the meaning of apoplexy to *spontaneous* hemorrhage in the brain (épanchemens de sang spontanées).

CLASSIFICATIONS.

After such limitation, it may be deemed supererogant, or even irrelevant, to advert to the forms or species of apoplexy which have been described by nosologists. Nevertheless, as a matter of justification, I am induced to recite a few of the principal classifica-

tions appointed only within the last two centuries, in order to display the uncertainty and laxity with which the word has been employed.

Ettmüller, professor of medicine at Leipsic, in the middle of the seventeenth century, divided apoplexies into *privative* and *positive*. The former he supposed to depend upon obstruction to the excursion of the animal spirits without alteration in their quality; the latter, to changes in the animal spirits themselves, rendering them unfit to perform their office.*

Baglivi (MDCCIV.) adopted a simpler classification. Thus, "apoplexiam distinguere oportet in sanguineam et pituitosam."†

Sauvages enumerates *eleven* species of apoplexy, but some of these he holds doubtful:—A. sanguinea, A. traumatica, A. temulenta (ab ebrietate), A. hysterica, A. arthritica, A. exanthematica seu scorbutica, A. pituitosa, A. epileptica, A. febrilis, A. suspiriosa, and A. polyposa. He previously describes several varieties of soporose affections.‡

Cullen gives *nine* forms:—A. sanguinea, serosa, hydrocephalica, atrabilaria, traumatica, venenata, mentalis, cataleptica, suffocata.§

Monsieur Portal devotes *twenty* articles to the varie-

* Cessat spirituum animalium motus, vel ob poros et meatus cerebri vitiatos, ita ut spiritûs denegetur transitus, vel ob motum sanguinis in et ad cerebrum sublatum, quo sanguinis circulo sublato, subito omnis animalium sensus et motus, et subito omnis spirituum animalium motus, ex sanguine in cerebro scaturientium cessat et tollitur.—*Michaelis Ettmulleri Opera omnia* (1688), vol. I. cap. X.

† De Praxi Medicâ, lib. I.

‡ Morborum Genera et Species, class VI. (1764).

§ First Lines, part II. chap. I.

ties of apoplexy, each treating of one or more species, and having reference to the circumstances connected with the attack, rather than to the pathological condition of the brain. According to him,* the proximate cause is only to be known on autopsy, though he divides apoplexies into essential and symptomatic, according as the brain is primarily or secondarily affected.

Abercrombie† and Clutterbuck‡ divide the disease into *three* forms, — sanguineous, serous, and simple apoplexy, and describe three groups of symptoms.

This arrangement, I need hardly say, is yet commonly recognized.

Dr. Mason Good classifies apoplexies into *entonic* and *atonic*.§

Dr. Hughes Bennett ‖ recounts *four* forms founded on symptoms, not on pathological conditions; viz., transient or fugitive apoplexy, sudden or primary, ingravescent apoplexy and paraplexy, or apoplexy with paralysis.

Dr. Copland¶ also divides the disease into simple or primary, ingravescent, apoplexy complicated with paralysis, and paralysis terminating in apoplexy.

PATHOLOGY.

The opinions of the older writers with regard to

* Observations sur la Nature et le Traitement de l'Apoplexie (1811).

† Researches on Diseases of the Brain and Spinal Cord

‡ Cyclop. of Pract. Med., art. Apoplexy.

§ Study of Medicine, vol. IV. p. 636 (1825).

‖ Library of Medicine, art. Apoplexy.

¶ Dictionary of Medicine, art. Apoplexy.

apoplexy were eminently vague and indefinite. They ascribed it to a phlegmatic or watery humour contained in the brain, contrary to nature; to failure of motion of the animal spirits in the cerebrum or cerebellum; to a violent ferment engendered in the stomach; to polypous concretions; to inflammatory spissitude of the cruor; to contraction of the pia mater or arachnoid constringing the encephalon; to intumescence of the cortical substance; to arrest of circulation within the cranium; although Cullen more correctly attributed the attack to some compression of the origin of the nerves, or to something destroying the mobility of the nervous power, and Hoffman employed apoplexy and hemorrhagia cerebri as convertible expressions.

Amongst later and contemporary writers more enlightened views have succeeded, yet considerable difference of opinion has prevailed, and, I believe, continues to prevail, in the minds of very able physicians as to the peculiarities of the intracranial circulation, the histological alterations in the brain and its vessels, the presence and connection of cardiac changes, with their special nature, the existence of renal degeneration and the constitution of the blood in cases of sanguineous extravasation.

In adducing authorities of repute, whose investigations or experience entitles them to reference and consideration, I shall attempt to demonstrate that the most prominent and constant lesion which exists prior to the advent of the apoplectic seizure, and is discoverable on autopsy, has been far from invariably and fundamentally insisted on. If, however, my conclusions appear invalid or unwarrantable, I shall be

amply gratified if this contribution be the means of reopening and determining an inquiry which undoubtedly possesses deep practical and scientific interest.

It may be well to assert, at the commencement, my conviction of the necessity of inspecting the viscera of the thorax and abdomen, as well as the contents of the skull, in fatal cases, otherwise observations must be limited and valueless. As I regard most, if not all, diseases to be more or less constitutional or systemic, a comprehensive knowledge of the structural deviations of all internal organs from the normal standard is essential, in order to be cognizant of the morbid complications which are occasionally, and which are uniformly associated, and to establish their import. Strenuously opposed to the narrowness of specialism in therapeutics, I equally repudiate a specialistic pathology. I shall, in the first place, briefly discuss the debatable question of the ENCEPHALIC CIRCULATION, concerning which diverse views are entertained by eminent physiologists. At the outset, it may be remarked, that it is not so desirable to determine if the amount of blood in the cerebral vessels varies *after death*, as to ascertain the constancy or variability of the intracranial circulation *during life*—a distinction which seems to have been greatly overlooked during researches on the subject. Dr. Burrows observes,* that "experiments and physiological considerations lead to the conclusion that the quantity of blood within the cranium is extremely variable at different times and under different circumstances." Regarding the serum as an important element, he admits that the whole contents

* Disorders of the Cerebral Circulation.

of the skull, that is, the brain, the blood, and the serum together, must be, at all times, nearly a constant quantity. In support of his opinions certain experiments on rabbits are detailed, which are recorded as *indisputable proofs* of the accuracy of his positions. In the first experiment two well-grown rabbits were taken. *A* was killed by opening the jugular vein and carotid artery on one side. *B* was strangled. A ligature was drawn tightly round the throat of the rabbit A, immediately it expired, to prevent any further escape of blood from the vessels of the head. *They were then allowed to remain twenty-four hours* on a table, resting on their sides.

On examination, the brain and vessels in A were pallid and exsanguined. In B the vessels, membranes, and brain-substance highly vascular, and as if stained with extravasated blood (pp. 13, 14).

Now this experiment appears to me to be open to objection, as twenty-four hours were allowed to elapse before examination of the heads.

It will be admitted that the blood, after death, obeys the laws of gravitation rather than the forces which operate during life, and *assuming*, in the animal bled to death, that the blood within the cranium *was not diminished at the time of death*, it might yet tend to become distributed, *after death*, over the body (through the vertebral veins, &c.), to replenish the emptied state of the venous system, notwithstanding the precaution of a ligature round the neck. If so, the anæmic appearance of the brain would be a *secondary* result. On the contrary, the ligature in B would tend to produce effusion of blood from forcible compression.

In the above experiments (A and B) not any men-

tion is made of the presence of serum, and if this was absent, we must conceive that a *plenum* nevertheless existed. The *exsanguined appearance of the brain in* A *is perfectly consistent with a full state of the vessels*, the pallor (from hemorrhage) depending, not on *deficiency*, but on *dilution and poverty* of the blood, from absorption and increase of its watery constituents.

In the next experiment Dr. Burrows killed two other full-grown rabbits (C, D) with hydrocyanic acid, and suspended C by the ears, D by the hind legs, for twenty-four hours. These, again, exhibited differences in the appearance of the brain and its vessels, referable to difference of position; but a similar objection will apply to that offered in the preceding experiment; for if the same conditions prevail during life as after death, the brain of man and of animals which maintain an upright posture, are always deficiently supplied with blood. Not to evade the opposite condition, *i. e.* inversion of the human body, it is much more probable that the sensations and effects, under the altered circumstances, are not due to simple congestion, but to increase in the pressure of the column of blood on the brain and derangement of the heart and other viscera, which may or may not accelerate, retard, or disturb the cerebral circulation, as schoolboys, professional tumblers, and others, as was pointed out by Dr. Kellie, sustain no injury or inconvenience therefrom.

Dr. Burrows adverts to those who have (he says) asserted that the quantity of blood within the cranium is invariable. Dr. Alexander Monro was of this opinion, as he found, on examination of several executed criminals (by hanging), that there was no internal congestion of the brain, though much blood

was accumulated in the vessels of the integuments. Drs. Kellie and Abercrombie are also held to have thought that no material change in the ordinary state of the parts ever took place in the vessels of the brain, and they were led to conclude so, because the brain often presented its usual appearance in animals bled to death. Dr. Clutterbuck entertained even more decided opinions.* It is, nevertheless, certain, that the conclusions of Dr. Kellie, so often introduced, have been grossly misrepresented, as he does not absolutely maintain that the amount of blood in the brain never deviates, but endeavours to show† that there are natural obstacles to the free depletion of the brain, which have no existence in any other part of the system; that we cannot, in fact, lessen to any considerable extent the quantity of blood within the cranium by arteriotomy or venesection; and that, when by profuse hemorrhages, destructive of life, we do succeed in draining the vessels within the head of any sensible portion of red blood, *there is commonly found an equivalent to this spoliation in the increased circulation or effusion of serum, serving to maintain the plenitude of the cranium* (Part II.). Dr. Kellie admitted that when a perforation was made in the cranium previous to venesection, the blood was withdrawn from the interior, equally with the remainder of the body.

As Dr. John Reid affirms in a very able review ‡ of the work of Dr. Burrows, Dr. Kellie did not pretend to declare that the relative quantity of blood in the

* Cyclop. of Pract. Med., art. Apoplexy.

† Edinburgh Med. Chir. Trans., vol. I. (1824).

‡ Monthly Journal of Med. Science, Aug. 1846.

vessels of the encephalon, and in those external to it, may, and does, not undergo alteration, but that when hemorrhage takes place from the general system, it does not affect the quantity of *fluids* within the cranium.

In support of my dissent from some of the propositions of Dr. Burrows, I beg to add that Dr. Reid, whilst according much credit and considerable talent to the former physician, differs from him in many important points, considering his conclusions are not warranted by the premises.

Perhaps in the whole domain of physiology, there is not a much more perplexing question than the one before us, or one which more forcibly manifests the unsatisfactory grounds on which men of high standing will at times stake their creed.

Dr. Watson is disposed, on the whole, to agree with the inferences of Dr. Burrows.

Dr. C. J. B. Williams,* as a compromise between the sentiments of Dr. Burrows and Dr. Kellie, believes that the vessels of the brain are not exposed to atmospheric pressure, being situated within bony canals. That they do not shrink as the blood within them is reduced in quantity, and they therefore retain more than their share of the circulating fluid. Although not agreeing with Dr. Kellie, he deems his own views not to be invalidated by the experiments of Dr. Burrows, as it is clear that the circulation within the head, especially in man, is affected by losses of blood differently from the circulation in other parts.

* Principles of Medicine, 3rd edition, p. 219.

Dr. Kirkes * clearly indicates the reasons why the amount of blood sent to the brain should be subject to less variation from external circumstances than it is in other parts. The tortuosity of the large arteries, their wide anastomoses in the circle of Willis, the transit of the large arteries through bone, the arrangement of the vessels in the pia mater, the peculiarity of the intracranial sinuses, formed as to be scarcely capable of change of size, and the brain itself, a mass of nearly incompressible substance, placed in a cavity with unyielding walls, all combine to produce such result.

Dr. Kirkes thinks the supply of blood to the brain, under ordinary states, is both uniform and guarded from the accidental disturbances to which other organs are liable even in health.

Dr. Copland † believes that the atmosphere has no direct or sensible influence on the circulation of the brain, and that the cranium must always contain nearly the same quantity of blood during life, the differences which occur being chiefly in velocity of circulation, and in relative proportion of blood in the orders of vessels, increase in the capillaries causing proportionate diminution in the veins.

Dr. Todd,‡ on the contrary, remarks that the cerebral circulation is not entirely removed from atmospheric pressure, though he thinks it less amenable to its influence than the vascular system of the surface.

Dr. Bennett, of Edinburgh, imagines that the circulation in the brain is removed from atmospheric influence, except through such as is communicated

* Handbook of Physiology, 1848. † Art. Apoplexy.

‡ Cyclop. of Anat. and Physiol., art. Nervous Centres.

through the blood-vessels which enter it. His views generally incline to those of Drs. Abercrombie, Kellie, Monro, and Carson. He thinks want of equilibrium or of just proportion of blood in veins and arteries, is the cause of pressure, as the calibre of some vessels being diminished and of others increased, the latter necessarily induce unaccustomed pressure on the nervous tissue with which they are in contact.*

Dr. Carpenter,† a practical physiologist, in opposition to the surmises that the amount of blood is always the same, but differing under certain circumstances in its relative volume in the veins and arteries, believes that it is *not* constant, as the brain varies in bulk according to the fluid it contains, and the cerebro-spinal serum varies according to the bulk of the other contents of the cranium, and rises and falls according to the state of vascular distension of the nervous centres.

Dr. Neil Arnott,‡ an authority in physics, thinks the atmospheric pressure always keeps the head full, and that there must always be the same quantity of blood within the cranium, however much soever the quantity may vary in the body generally. He observes that the arteries or the veins may be too full, with compensatory reduction, or emptiness, in the opposite vessels, and that this must impede, according to its extent, the general cerebral circulation, as narrowing of one part of the channel must retard the entire current. Simple increase of pressure produced by the

* Library of Medicine, art. Diseases of the Nervous System.

† Manual of Physiology.

‡ Elements of Physics, vol. I. p. 601.

blood on the brain, he holds to have no injurious effect, if the balance in the vessels be sustained, as the strain is borne by the cranial parietes, and not by the coats of the arteries. Hence they need not be, and are not, so strong as those of other parts of the system.

As a summary from the above conflicting, and in certain cases imperfect opinions, which might be readily multiplied, and which are in harmony so far as the general admission of an intracranial *plenum*, it may be concluded that the circulation within the cranium (notwithstanding its less liability to disturbance than the circulation in other situations) varies even under normal physiological processes. That this variation may depend either on differences in the *relative complement of blood* in the arterial, venous, and capillary channels, as during excited action of the heart in running, coughing, and other exertion, and under emotional, intellectual, and psychical activities; or difference in the *absolute amount* in its entire vascular system, as Mr. Durham* has conclusively shown that the brain, during sleep, is in a comparatively bloodless condition.

The cerebral circulation, moreover, as the circulation elsewhere, must be much modified by local (nutritive) forces, so that functional exercise of the brain is doubtless attended with increased *rapidity of flow* of blood in all its series of vessels. Alteration in the amount of blood is also regulated by the rise and fall of the serum, or rather *vice versâ*, which is freely and conveniently introduced on all occasions (though by some entirely overlooked), as able to minister to

* Guy's Hospital Reports, 1860.

deficiency or vacuum in the arterial or venous system, or cerebro-spinal space; but its quantity is very limited—supposed to amount to about two ounces,—it can be but gradually absorbed or effused, and when met with in abundance in the dead, except in age, is to be regarded for the most part as a *post mortem*, or at least *apud mortem* phenomenon.

The intimate anastomoses of the carotid and vertebral arteries assist to ensure the equability of the vascular supply to the brain, which, nevertheless, under conditions of health, is also affected by the muscular force of the heart, state of the circulating fluids, and the performance of the respiratory processes.

The inspiratory act favours the progress of the venous blood from the head, and consequent ingress from the arterial trunks, reversal of which occurs during the period of expiration.

Further, have physiologists sufficiently reflected—they, at least, are almost silent on the anatomical relations of the cranium at the earliest period of infant life, as modifying the vascular system of the encephalon?

I allude, of course, to the fontanellar spaces, the patency of which must permit atmospheric pressure to operate equally, as in other situations. Yet how rare is apoplexy, although changes in bulk of the intracranial contents, in the same child, are frequently manifested by the alternate depression and prominence of the scalp in the fronto-parietal region, under variations of health and disease? The hypothesis of disproportion, or of want of equilibrium in the distribution of the blood in arteries, veins, and capillaries, as a cause of congestive pressure, and as the proximate

origin of many comatose diseases, is not only totally undemonstrable, but unsupported by any fact, probability, or indeed anything but the airiest speculation, and is, in a word, altogether irreconcileable with the preceding considerations.

It is, of course, incontestable that engorgement of the cerebral vessels is commonly described and encountered, but this, like sugillation of the posterior aspect of the trunk and the so-termed *pneumonia morientium*, is much due to position and mode of death. The cadaveric blood-stasis is little criterion of the circulation in life. A congested appearance of the vessels of the brain, which is usually limited to, or most marked in, the veins, may be merely compensatory to an empty condition of the arteries, and certainly is chiefly owing to the nature of the death and state of fulness of the vascular system, independent of cerebral disease.

The compressibility and elasticity of the encephalic mass, though very slight, may aid in enabling it to accommodate itself to the effects of the systolic impulse (and the movement of respiration ?). When, however, from injury, disease, or experiment, a part of the cranium is removed, the corresponding portion of brain substance is protruded through the aperture, being no longer circumscribed by the resistance of the osseous walls. But Dr. John Reid doubts, and with much reason, whether there is any movement of the brain whilst the cranium remains entire.

With regard to the *momentum* of the blood, as Dr. Burrows remarks, physiological and pathological investigations, as well as the records of surgery, satisfactorily prove, that *sudden* diminution, arrest or prolonged exaggeration of the arterial impulse, tends to

produce disastrous consequences, as in syncope, pressure on, or ligature of, the carotids, and excessive action of the left ventricle (?); but I maintain that, in the respective cases, these do not depend on mere diminution or increase of blood within the head, or on variation of pressure, but on circumstances connected with the nutrition of the brain, which I shall more fully consider after completion of the subject of cerebral hemorrhage.

The points most important to be here considered in connection with abnormalities of the encephalic vascular supply are, their practical bearings on the occurrence of apoplexy; and I fully agree with Dr. Kellie (loc. cit.), who believed the agency of the causes which have been presumed to have a powerful and undoubted tendency to force blood into, or to confine it within, the vessels of the brain, so as to produce a dangerous morbid congestion, has been greatly overrated, and that while the structure of the brain remains *healthy* and *unchanged* and its *vessels sound*, these causes are little capable of occasioning plethora, congestions, effusions, or comatose diseases.

When we reflect that those pursuing the most laborious employments and taking the most violent exercise, as artisans, athletes, runners, divers, children, and what is more weighty, that those suffering from obstructive, simple hypertrophous, and other affections of the heart are not, in early life, prone to true apoplectic seizures, we must admit that simple modification in the conditions of supply of blood to the brain does not *singly* exercise any material pressure or other influence in the causation of apoplexy.

Having disposed of the circulation within the cranium,

and endeavoured to argue that irregularity or deficient balance in the venous, arterial, or capillary apparatus is inoperative, *per se*, to induce cerebral hemorrhage, I shall allude to the important changes which are met with in the vessels, and immediately consider how far these appear to contribute to, or avail in, its production.

It need scarcely be premised, that most systematic writers are agreed as to the weighty influence of disease of the cerebral vessels and tissue in the production of apoplexy; yet by some, this is not regarded as a necessary concomitant, and, in published works, examples are incidentally quoted or referred to, whose history *à priori* precludes the probability of cerebral or arterial degeneration. Nevertheless, I am persuaded that if these apparently exceptional instances were minutely detailed and analysed, they would confirm the doctrine, that in all cases of *primary* idiopathic sanguineous cerebral effusion, disease of the coats of the vessels previously subsists.

From these remarks, it must not be imputed that I conceive textural alterations in the walls of the arteries to be the sole and efficient ætiological agents in apoplexy, as their frequent presence in subjects of mature and advanced age, without the supervention of any cerebral symptoms, would disprove such view. On the other hand, apoplexy is almost restricted to the afternoon of life, a period attended by degeneration of the vascular tunics. These facts cannot be overlooked; but, for the sake of convenience, I shall defer the discussion of the relationship between disease of the arteries and apoplexy until I enter on the consideration of the changes observed in the cardiac walls and

orifices. Albeit, lest hesitation arise in accepting the truth of the *coincidence* of morbid alterations in the cerebral vessels of persons cut off by an apoplectic seizure, it is expedient to affirm that this had been particularly noted by the majority of observers.

I now come to the organic deviations which are commonly detected in the heart, to inquire how far to these, in conjunction with a morbid state of the cerebral arteries, the hemorrhagic extravasation can be assigned. "But in this attempt," says Dr. Burrows, "there is much difficulty. It has been so commonly the practice, in the examinations of the bodies of apoplectic patients, to remain content with the lesions discovered in the brain, that *few authors afford the precise information* essential to forming a calculation of the relative frequency of diseases of the heart in cases of apoplexy and hemiplegia." Every practitioner can attest the truth of these observations of Dr. Burrows, and recall instances of (presumed) apoplexy or other cerebral disease, where examination on autopsy was confined to the most obvious source of mischief—the encephalon. On this account, structural changes in the vessels of the brain have been almost universally registered; and if researches had been equally addressed to the thoracic and abdominal cavities, there can be but little doubt that the concurrence of other lesions would have been nearly as generally appreciated. On the contrary, the pathologists who have specially occupied themselves with cardiac diseases, have (with exceptions) incidentally adverted to and recognised their connection with apoplectic seizures, but in a similar limited manner seem to have been oblivious of, or inattentive to, the minute but impor-

tant morbid appearances presented within the cranium. Hence the discrepancy of views in vogue. The condition of the cerebral arteries has been very commonly recorded, the altered state of the heart only occasionally inspected and described; the influence of the former has, in consequence, been unduly exaggerated; of the latter, either ignored or regarded by most as a fortuitous accompaniment.

The dictum of the few has received the sanction of the many; error has been perpetuated; and the current opinion of the omnipotence of artérial degeneration in the production of cerebral hemorrhage, is the result of imperfect investigation, whilst the adequacy of disease of the heart is likewise refuted by the revelations of pathology and the lessons of clinical experience.

In support of these views, I narrate the annexed *unselected* cases, in which the post-mortem appearances were noted at the time, the heart scrutinized, and weighed in all save one, perusal of which must, I think, lead to an almost irresistible persuasion, especially when fortified by certain auxiliary considerations.

Case I.—J. G., aged 63. Clot in right thalamus and corpus striatum, extending to left side and into third and fourth ventricles. Two antecedent attacks—the first five years previously—of softening or limited hemorrhage, inducing permanent hemiplegia (left). Cerebral vessels atheromatous, not calcified. Heart, 14 oz.; hypertrophy of left ventricle; no valvular disease. Kidneys small and granular (co-existent uræmia?). No œdema Consciousness not entirely

abolished. General impairment of muscular power, succeeded by rigidity, coma, and death.

J. G., aged 63, was seized with a fit on January 29th, 1856, about 2 p.m. I found him sitting on a bench, and was informed he was seen to stagger by another man, who prevented him from falling. He was in his usual state in the morning. Epistaxis had occurred about a fortnight before. He had already had two attacks of paralysis, in neither of which, according to his sons, had he been insensible, but ever afterwards he had impairment of the use of the left side, and indistinctness of speech. The first attack was about five years, the second about twelve months previously. He was reported to have been very intemperate, when young, as to beer and spirits, and to have been a very intelligent draughtsman. When I was summoned, he could speak imperfectly, and sit without support. He had not vomited. The tongue was slightly furred, and did not markedly deviate. Bowels had been opened in the morning. Pulse feeble: about 70. His appearance was cachectic; skin sallow, with slight colour in cheeks. There was an expression of hebetude, and a general diminution of muscular power. No stertor. The state of the pupils was not, at this time, recorded.

He was ordered to be placed in bed, and to have two scruples of compound jalap powder.

At 8 p.m. he was insensible, and breathing heavily and slowly, not stertorously. Pulse 90, full, weak, compressible. Pupils contracted and insensible to light. No facial paralysis. Left arm rigid; right also, in less marked degree. Pinching elicited no

complaint, but instinctive movements. The powder administered was rejected. Fluids could be swallowed. Râles in throat. Ordered eight grains of calomel, and a blister on the nucha.

At 12 p.m. the breathing was becoming stertorous. Pulse fuller and less compressible. Surface hot. Not any more sickness. Pupils contracted. Upper extremities still rigid; not convulsed. Face drawn to left side. Pinching does not produce movements, Ten ounces of blood were taken from the arm, without any marked alteration being produced. The clot was small and friable; the serum great.

Death took place at 7 a.m.—seventeen hours after the attack—with previous vomiting of some thick grumous fluid. The powder did not operate, but urine had passed involuntarily.

According to the nurse, death was not preceded by whiffing or convulsion.

P.M. twenty-nine hours after death. Weather cold, fine. Face placid, sallow, with redness in cheeks. Lips lividly pale. Pupils rather contracted. Arcus slightly marked. Rigor mortis marked in upper and lower extremities and jaw. No œdema. Moderate amount of fat superficially and around viscera.

Head. — Scalp congested. Calvaria rather thin. Dura mater partly adherent to skull. Arachnoid natural. Pia mater congested—large and small vessels. No subarachnoid effusion. Two to three ounces of dark red blood at base of brain and dark fluid blood in lateral sinuses. Brain weighed 42 oz. It seemed to start up on removal of the bone, as if too large for the encephalic cavity. The grey matter of the convolutions

was remarkably atrophied, and the white substance rather less vascular than usual. Substance of brain normal. On section of right hemisphere, a *clot* was exposed before reaching the level of the ventricle. It had completely destroyed the right thalamus and posterior part of the corpus striatum. It had also broken up and lacerated the surrounding brain-substance, external to the right ventricle, so as nearly to extend to the surface of the hemisphere—at points encroaching on the *thin* layer of grey matter. The effusion also passed into the left ventricle, but the cerebral tissue was not destroyed on this side. The wall of the hemorrhagic cavity was very soft and irregular, and the vessels of the thalamus on the right side were very conspicuous; whence the effusion appeared to have proceeded. The clot, which was black and chiefly solid, weighed $2\frac{3}{4}$ oz. It extended into the third and fourth ventricles. No other seat of previous or present extravasation could be detected, but there was a slight ecchymotic appearance of the pia mater over the surface of the vermiform process. The basilar artery appeared to be healthy, but the cerebral arteries were very distinctly atheromatous, not calcified. No softening nor hemorrhagic cyst could be discovered.

Chest.—Lungs engorged posteriorly, emphysematous. Bronchi and divisions dusky red in their interior, containing frothy mucus. No tubercle. No adhesions. No fluid in pleura. About an ounce of serum in pericardium. Heart 14 oz., no valvular disease, but fibrous thickening of auriculo-ventricular valves. Mitral orifice easily admitted two fingers; the tricuspid, three. Cavities of normal dimensions, but left ventricle hypertrophied, nearly an inch in thickness. Considerable

amount of dark fluid blood in right chambers, none in left; but a little dark fluid blood escaped on section of aorta. Coronary arteries atheromatous, and some patches of the deposit also in interior of aorta. No calcification. Muscular substance of heart pale, but tolerably firm to the finger. The walls of right ventricle at parts were yellowish on section.

Stomach.—Contained dark grumous matter and semi-digested food. Surface pale. Spleen $3\frac{1}{4}$ oz., firm, pale. Liver small, bloodless, cirrhosed. Gall-bladder moderately distended. Kidneys, left, $2\frac{1}{2}$ oz., small, firm, pale; small cysts on the surface. Capsule not readily separable. Section very pale, and scarcely any distinction between cortical and medullary portions. Finely granular after the removal of the capsule. Right weighs $3\frac{1}{2}$ oz., a little larger than its fellow, but similar in characters. It has a knobby elevation on its surface, with a yellowish section. Intestines natural. Bladder thickened, but prostate not enlarged.

Case II.—S. A., aged 72. Hemorrhagic effusion, limited to *left* lobe of cerebellum. Paralysis (*right*) of fifteen years' standing. Basilar and cerebral vessels atheromatous and brittle, but not calcified to the feel. Heart 11 oz., hypertrophy of left ventricle, no valvular disease. Kidneys small, granular. No œdema.

S. A., aged 72; who, in earlier years, had been a ladies' nurse, and was reported to have been very temperate, was found dead in bed about 2 a.m., on January 30, 1856. She was noticed to be sleeping as usual about an hour previously. She had been infirm for some period, and was deficient in intellect and indistinct in speech, but her general health was

moderately good, and she appeared well nourished. An attack of paralysis of the *right* side had occurred, fifteen years before, in presence of her daughter, suddenly, without loss of consciousness. She appeared to be in her accustomed state on the evening preceding death.

Autopsy eighty hours afterwards. Weather cold, fine. Pupils dim. Arcus distinct. Face calm. Rigor mortis well marked, most on left side. No œdema. Body very fat.

Head.—Scalp congested. Calvaria moderately thick. Dura mater more vascular than usual, and very adherent to inner table, so that it can only be separated in shreds at certain spots. Arachnoid slightly opalescent. Considerable subarachnoid effusion. Pia mater congested, especially the larger veins, and over left lobe of cerebellum. There is extravasation of blood into its meshes about the size of a shilling. About 3 oz. of thick red blood at base of brain, and some dark clots adherent to posterior fossæ. A clot also adherent to the under surface of the tentorium on the right side. Dark fluid blood in sinuses.

Brain 42½ oz. Consistence normal. Ventricles contain serum, slightly reddish in colour, which distends their cavities, and a small black clot, about the size of a bean, is floating in each ventricle. Not anything marked about thalami or corpora striata. On section of cerebellum, the left lobe presents evidence of extensive effusion of blood—a dark clot, weighing exactly half an ounce, existing towards the inferior surface of the lobe, and occupying the central part. The cerebellar substance is much softened, and irregular in outline around the extravasated mass.

There is no effusion on the right side or into the pons or medulla.

The basilar and cerebral arteries are extensively atheromatous and brittle, and break on slight traction, but there is no calcification apparent to the feel.

Chest.—Old adhesions, lungs congested and emphysematous along anterior borders and at bases. Bronchi and divisions injected and containing muco-purulent secretion. On section of lungs, considerable frothy fluid exudes; they crepitate imperfectly. There are one or two cretaceous nodules at apex.

About two drachms of pericardial serum. Heart weighs 11 oz., right ventricle flaccid; left, contracted. Much fluid black blood in right auricle and ventricle, and pulmonary artery. Some dark blood in left auricle; left ventricle empty. No marked valvular disease. Tricuspid orifice easily admits three fingers; the mitral admits two—not easily, appearing somewhat contracted. Walls of left ventricle hypertrophied, nearly an inch in thickness. No dilatation. Consistence and colour of muscular substance good. More fat than common on the surface of the heart, but the amount not great in proportion to its deposit superficially and around the viscera.

Aorta atheromatous, also coronary arteries, but not so extensively as the cerebral vessels. Liver pale. Stomach small, mucous surface congested with uniform injection at spots. It does not contain food. Intestines healthy, containing chyle and fæcal matter. Spleen firm, small, weighs $1\frac{1}{2}$ oz. Uterus has a virgin appearance; os smooth. Bladder contains urine. Kidneys—each weighs $3\frac{1}{2}$ oz. Similar in physical appearance, having a lobulated surface. Capsules not

readily removed, and the renal substance is detached with them. Section is firm and pale. The cortical substance can be distinctly defined, but is narrower than natural, and presents an appearance of granular deposit in its substance. The medullary portion is also pale, but seems less altered from the healthy standard.

Case III.—E. S., aged 72. Laceration of right corpus striatum, and of the thalamus, and extravasation into ventricles and at base of brain. Vessels extensively atheromatous. Heart 13 oz., hypertrophy of left ventricle, valves nearly normal. Kidneys small, granular; urine albuminous. No œdema. Gradual invasion of symptoms, consciousness and speech not suddenly abolished, but slow supervention of insensibility, stertor, and paralysis.

E. S., aged 72, generally in tolerable health, but pale and feeble and subject to winter cough. She has never complained of giddiness or pain in the head, but for three months past has observed her left arm (only) to be colder than usual.

On June 16th, 1855, about 8 p.m., she went to the closet, and was there suddenly taken in a fit. On my arrival I found her in bed. She was conscious, but could not answer questions well. She stated that she had never suffered from headache or giddiness, but then had pain over the right brow and temple. There was loss of power on the left side, but she could raise, with some effort, the left leg from the bed, and also her left arm with more difficulty. There was no convulsive movement nor rigidity. The mouth was slightly drawn to the right. Respiration was easy, natural.

Face pale. Tongue furred, did not seem to deviate. Pulse slow, compressible, radials calcified. First sound of heart weak and feeble; second sound, clear. Impulse very weak. Pupils contracted, very little affected by light. Arcus marked. No œdema. No pain in back. Said to usually pass much water, which, on examination, I found to be albuminous. Bowels had been opened twice in the day.

Slight hemorrhage was diagnosed, and compound jalap powder, with a mixture of acetate of ammonia, and a blister to the nape were ordered.

Insensibility slowly came on, and she died at 1 a.m., stertor and convulsive movements of the paralysed side having previously ensued.

Examination of body sixty hours afterwards. Weather warm, fine. Appearance calm, pupils moderately dilated, lividity of nails. Some emaciation. Rigor mortis well marked in lower extremities, less in upper, and equally on both sides.

Head.—Dura mater very adherent to inner table. Arachnoid opalescent. Pia mater finely injected. Consistence of brain and number of vascular points normal. The right ventricle is completely filled with black coagula, which have broken up the external part of the right corpus striatum and thalamus and surrounding substance of the wall of the ventricle. The effused blood extends into the left ventricle, but does not, on this side, lacerate the brain. The clot also occupies the third and fourth ventricles, and there are coagula at the base of the brain. There is slight hemorrhage into the meshes of the pia mater. The mass of blood weighs about 2 oz. The sinuses are

filled with dark blood. The vessels are extensively invaded by atheroma.

Chest.—Lungs on both sides universally adherent. Some chronic bronchitis, from the appearance of the air-tubes. The lungs in parts are non-crepitant, and have the coriaceous, non-friable consistence often present in the aged. They are not emphysematous. No tubercle. About an ounce of fluid in pericardium. Heart weighs 13 oz. Coronary arteries atheromatous. Much dark fluid blood in right auricle, but little in the other cavities. The right side is flabby, the left contracted. Not much fat on heart. Dimensions of its chambers appear natural. Left ventricle is hypertrophied, nearly an inch in thickness. The mitral valve is beaded with fibro-calcific deposit, and the orifice is perhaps a little contracted. Other valves normal, but the aortic sigmoid valves exhibit some fibrous thickening and calcified deposit along their attached border. Colour and consistence of heart good. Some, but not extensive, fatty degeneration under the microscope.

Stomach small, pale. Spleen small, very friable. Kidneys cystic. The right weighs $3\frac{1}{2}$ oz., is granular and pale, and has a large cyst at its lower extremity. The left weighs $2\frac{1}{2}$ oz., is also pale and granular, and is studded with small cysts. Liver congested. Some small gall-stones in gall-bladder. Intestines natural. Bladder distended. Uterus very small, has several very small fibrous tumours containing osseous deposit, which are easily detachable from its fundus.

Case IV.—J. T., aged 70. Limited effusion into third and fourth ventricles, with but slight laceration of

adjacent cerebral substance. No previous symptoms. Cerebral vessels extensively atheromatous with points of calcification. Heart 15 oz., hypertrophy of left ventricle, no valvular disease. Kidneys apparently healthy; urine albuminous, about one-twelfth. No œdema.

J. T., aged 70, has not been of late under medical treatment, and seemed in his usual state on September 11th, 1856, not making any complaint of illness. About 5 p.m. he was perceived to be leaning on one side as if resting on his seat, and on closer inspection was found to be insensible. When I was summoned he was inclined to one side, with his head against the wall, quite unconscious to external impressions. Pupils were contracted; arcus very marked. There was slight stertor, pulse quick, small and weak; no paralysis, but rigidity and convulsive twitchings of the limbs. Feet were cold. Mouth scarcely, if at all, deviated. Symptoms of, but not actual, vomiting. Urine, which was drawn off, was found to contain albumen, about 1-12 on standing. He was removed to the infirmary. Warmth was applied to the feet, head was raised, and a dose of calomel and croton oil administered. To be seen again shortly. He died, however, at 6 p.m., an hour from the commencement of the attack.

Inspection forty-five hours afterwards. Weather warm, gloomy. Face placid, pupils moderately dilated. Rigor mortis well marked in lower limbs, less in upper. Body not much emaciated. Abdomen moderately distended. No œdema.

Head.—Calvaria tolerably thick. Dura mater very firmly adherent to inner surface of skull. Considerable opacity of arachnoid and some subserous effusion.

Pia mater not readily separable from brain. Consistence of brain-substance natural. Puneta vasculosa rather numerous. Ventricles filled with serum, but no trace of effused blood. Fornix remarkably soft, almost pulpy, and, on raising the velum interpositum, a clot is seen in the third ventricle, which extends backwards beneath the valve of Vieussens into the fourth ventricle. The effusion is circumscribed by the boundaries of these parts, not extending to the lateral ventricles or to the base of the brain. The cerebral tissue in the neighbourhood of the thalamus on each side, and the floor of the third ventricle are, to some extent, broken up, softened and disintegrated. The vessels of the brain are extensively atheromatous, with calcified patches at intervals, especially at their bifurcations. The whole amount of the extravasated blood is about the bulk of a damson. About an ounce and half of sanguinolent fluid at the base of the brain, and the sinuses are filled with dark blood.

Chest.—Lungs very firmly adherent. They crepitate well, are congested and gorged with serum. Bronchial tubes dusky in their interior. No tubercle.

Pericardium contains small amount of fluid. Heart weighs 15 oz.; right side firm, left side flabby. Coronary arteries are atheromatous and calcified, not obliterated at their origin. Much dark semi-coagulated blood escapes on section of all the great vessels. The chambers of the heart do not appear to be enlarged, but the left ventricle is almost an inch in thickness. The semilunar valves on both sides support a column of water well. All the valves are normal. The tricuspid admits three fingers easily, the mitral orifice two.

Colour of heart pale, but consistence good. Traces of atheroma in the aorta.

Stomach distended, containing imperfectly digested beef and potatoes. Mucous surface congested, and veins very prominent near the greater end; towards the pylorus pale. Liver congested; gall-bladder empty. Spleen small, firm. Pancreas apparently natural.

Case V.—D. Q., aged 68. Extravasation into left cerebral hemisphere external to, and not opening into, lateral ventricle of same side. Cerebral vessels extensively atheromatous. Heart 16 oz., hypertrophy of left ventricle, no valvular obstruction. Kidneys apparently healthy, urine slightly albuminous. No œdema. Rigidity and convulsive movements of inferior extremities, arms flaccid.

D. Q., aged 68, labourer, brought to St. Marylebone Infirmary on March 3rd, 1857, about 2 p.m. Nothing known of his mode of life. It was stated that he fell down in a fit about an hour previously.

There was profound coma, the pupils were widely dilated and almost insensible to light, the legs rigid with convulsive twitchings, the arms flaccid. Respiration stertorous, but not deeply so. No yawning. Mouth half open, not deviating. Tongue clean. Surface warm, very dirty. Pulse 76, calcified, full and labouring. Hypogastrium dull. About a pint of limpid urine which was slightly albuminous, was withdrawn by catheter.

He was removed to bed and ordered ♏j of croton oil. At 3 p.m. his state was much the same, but the surface cooler and the pulse intermittent. Death took place at 4 p.m.

Autopsy two days after death. Weather mild, fine. Face calm, pupils less dilated, arcus tolerably marked. No external injuries. Rigor mortis well developed.

Calvaria very adherent to dura mater. Arachnoid partially opalescent. Pia mater natural. Brain substance of good consistence. Puncta not very evident. Lateral ventricles filled with fluid. Cerebral vessels very extensively atheromatous, not calcified.

On section of hemispheres, a clot appears on the left side, weighing $3\frac{1}{2}$ oz., which occupies almost all the interior of the left half of the brain. It is situated external to the ventricle, not extending into it, but encroaching on its outer boundary. The effusion apparently originates from the extra ventricular part of the corpus striatum. The brain substance around is of course lacerated and softened, but no such condition can be discovered elsewhere. About two ounces of sanguinolent fluid at base of skull.

Chest.—Heart weighs 16 oz.; it is firmly contracted. The right cavities contain much dark fluid blood; the left are empty. No valvular disease. Walls of left ventricle are much thickened, not dilated. No fluid in pericardium. Coronary arteries atheromatous. No fluid in pleuræ. Slight adhesions of lungs. No tubercle. Lungs engorged and emphysematous. Bronchi and larynx contain much mucus, and the lining membrane is dusky red.

Stomach empty. It exhibits traces of chronic inflammatory action. Spleen seems healthy. Right kidney about 4 oz.; very firm and rather pale; capsule separates well, not granular. Left kidney nearly 4 oz.; same characters. Liver slightly congested. Intestines natural. Bladder distended. No fluid in peritoneum.

CASE VI.—R. B., aged 68. Extensive hemorrhage into the meshes of the pia mater, cavity of arachnoid, and at base of brain. No laceration of cerebral substance. Atheromatous and cretaceous changes in cerebral vessels (? rupture of posterior cerebral). Heart attenuated; not weighed. Valves healthy. Kidneys congested. Sudden death.

R. B., aged 68, dropped suddenly dead in the street on July 4th, 1853.

Post mortem two days afterwards. The brain was healthy. Blood was effused on the surface of the left hemisphere, but there was no laceration. There was hemorrhage into the meshes of the pia mater, extending almost over the entire brain, and also into the lateral and fourth ventricles; in fact, in most parts invested by this membrane. Great amount of fluid blood at base of brain. Atheromatous and cretaceous changes in the cerebral arteries, and an opening (?) apparently in the left posterior cerebral, which seemed to indicate the source of the extravasation.

Chest. — Lungs congested and emphysematous. Some consolidation and adhesion at upper part of left lung. Heart attenuated and fatty (?). It was not weighed, as the examination was conducted in a close court, under difficulties,—in the presence of a distinguished physician.

Chymous matter in the stomach and intestines. Kidneys congested. Liver healthy. Urine in bladder.

The general surface of the body was pale, and the mouth was slightly drawn to the right.

CASE VII.—C. H., aged 72. Slight effusion of blood between dura mater and calvaria; also very limited

extravasation into left thalamus. Vessels of brain atheromatous. Considerable serous effusion. Heart 10½ oz. No obstructive valvular disease. Kidneys very small and granular. No œdema.

C. H., aged 72, has suffered from chronic bronchitis. She was tolerably well on January 22, 1855, and walking about without complaint. About 2 a.m. on the 23rd she was abusing an old woman in the next bed, and at 3.30 was found dying, and expired in about half an hour. Necropsy forty-eight hours afterwards. Body tolerably fat. No œdema. Countenance placid. Arcus well marked.

Head. — Calvaria natural. Dura mater adherent posteriorly to its inner surface, and a small effusion of blood between the two over the situation of the longitudinal sinus. Arachnoid opaque at numerous spots over the hemispheres. Considerable effusion of sanguinolent fluid in subarachnoid space. Pia mater not congested. Substance of brain of normal consistence and appearance. Slight amount of fluid in ventricles. A clot of blood, about the size of a bean in left optic thalamus, firm, nodular, and circumscribed. No other extravasation could be detected. Basilar and cerebral vessels much invaded by atheromatous deposit.

Thorax.—No fluid in pericardium. Heart 10½ oz. Right auricle and ventricle appear to be dilated, and the tricuspid orifice admits all the fingers and thumb. Pulmonary valves do not perfectly support a column of water. The right cavities contain dark fluid blood, and there is also a little on left side. Lungs are emphysematous, and the lining membrane presents the evidences of chronic bronchitis. There are slight

adhesions and some fluid in right pleura—none on the left side. Liver engorged. Spleen natural. Right kidney weighs 2½ oz.; it is granular on section, and removal of the capsule lacerates the subjacent substance. The left weighs nearly 3 oz., and is very similar to the right.

Other viscera have the ordinary appearances.

Case VIII.—L. W., aged 46. Effusion of blood into cavity of arachnoid, and two apoplectic cysts of old standing in right hemisphere. Vessels of brain atheromatous. Heart 16½ oz., dilated, with contraction of aortic orifice and valvular thickening. Kidneys congested.

L. W., aged 46, has been hemiplegic for about six months, and has kept her bed, except occasionally when she has been dressed with assistance. Her general health has been lately tolerably good, and she has only complained of constipation, which has been relieved by white mixture.

She appeared as usual on December 1st, and was lifted out of bed on the 2nd, about 4.30 a.m., and at 7 o'clock was found hanging over the side of the bed. I was immediately in attendance, but found her dead.

Autopsy three days afterwards. Rigor mortis well marked in lower and right upper limbs, not so marked in left. Sugillation of neck and posterior region. Face somewhat livid; pupils equal, moderately dilated. Body fat.

Head.—Vessels of scalp much congested, and dura mater more than ordinarily vascular. About an ounce of effused blood, dark, but not black in colour, in the cavity of the arachnoid, over the posterior part of

right hemisphere. Surface of brain not unusually injected, and no extra punctiform vascularity on section, but the veins are generally gorged. Some fluid in ventricles, and on slicing the right hemisphere, above the level of the ventricle, two apoplectic cysts are discovered: one in the central part, the other at the circumference of the brain, the central one being yellowish, the marginal one reddish in colour. The surrounding substance does not appear softened. Cerebellum and pons present nothing abnormal. The vessels at the base are extensively atheromatous.

Thorax.—About two ounces of fluid in pericardium. Heart weighs $16\frac{1}{2}$ oz. The cavities are dilated, both auricles and ventricles, and the right auricle has very thin walls. All the valves are natural but the aortic, which are thickened by fibrous deposit, and the orifice is much contracted. Dark fluid blood on both sides of the heart—most on right. Lungs congested and emphysematous. Lining of trachea much injected. Liver congested, also kidneys, especially the right. Spleen natural. Bladder empty. Intestines pale. Stomach small, contracted; inner surface pale; rugosities highly marked.

For the purpose of comparison, I record two cases of cerebral hemorrhage, in which the inspection was confined to the cranium, whereby the bald unsatisfactory information obtained may, with practical advantage, be contrasted with the completeness of those in which the several visceral cavities were examined.

Case IX.—J. S., aged 54. Effusion into ventricles and laceration of left corpus striatum. Cerebral and basi-

lar arteries atheromatous. Convulsive movements of left arm. Chest and abdomen not examined.

J. S., aged 54, has been for some months affected with hemiplegia (right). He is described as being sometimes "lost to himself," and "not appearing quite right." It is not known whether he has ever previously had a fit.

On October 1st, about 6 p.m., he was found insensible. The body was warm, covered with perspiration, the face pale and placid, mouth not drawn, but presenting some amount of foam. No whiffing. Pupils moderately dilated, and acted under stimulus of a lighted candle. There were convulsive movements of left arm; and, when interfered with, he raised this limb instinctively to remove the offending agent. Pulse 90, bounding; peculiarly thrilling, but incompressible. Brachial artery visible in its pulsations.

About a pint of blood was drawn from the right median cephalic, which escaped slowly. He then became sick, and rejected sour-smelling half-digested food. Shortly afterwards, as the pulse remained full, another half-pint was removed from the opposite arm. Mustard poultices were applied to the calves, hot bottles to the feet, and ice to the head, which was kept raised. Eight grains of calomel. He remained in the same state until the next morning, but the pulse became weaker, and he gradually sank about 5 p.m., about twenty-three hours after the commencement of symptoms.

Autopsy 44 hours after death. Head only opened. The body was well formed and moderately fat. Rigor mortis well marked, equally in lower limbs, less in upper.

Dura mater very adherent to calvaria, especially about the middle of the longitudinal sinus, so that part of the fibrous membrane is torn away with the bony covering. Arachnoid opaque. Pia mater natural. Laceration of left corpus striatum from effusion of clots and fluid blood, which extends into the lateral, third, and fourth ventricles. There is also extravasation of blood into the meshes of the pia mater, on the under surface of the cerebellum. Cerebral and basilar arteries, and their branches, atheromatous, not calcified. From 3 to 4 oz. of fluid blood at base of brain. No appearance of a cyst, or antecedent cerebral lesion.

Case X.—S., 65. Extensive effusion into and laceration of left cerebral hemisphere and substance of the pons varolii. Effusion also into lateral, third, and fourth ventricles. Evidence of former extravasation (?) in left anterior lobe. Basilar and cerebral arteries atheromatous. Rigidity of all the extremities, with convulsive movements of arms. Body not opened.

S., aged 65, night nurse, was on duty on Sept. 13th, 1853. She was last seen in her accustomed state about 5 a.m. on the 14th, and about 6.30 a.m. was discovered in the water-closet in an insensible condition. Nothing was known of her previous history.

I found her unconscious, snoring loudly. Her face was pale, to some extent bloated. There was slight foam about the mouth, which did not deviate. Occasional whiffing during expiration, but not marked or constant. The upper and lower limbs were rigid, and there were convulsive twitchings of the arms. The left pupil was contracted, the right also in less

degree, and both irides were motionless to the flame of a lighted match. The heart gave a peculiar, slow, heaving impulse, and the pulse was about 60, jerking and incompressible. I bled her in the left arm, and subsequently in the right; but the blood flowed slowly, and I only obtained about ʒxij. of blood. I ordered 5 grs. of calomel immediately and her head to be raised.

Mr. Squire bled her, about an hour afterwards, in the jugular vein, to about a pint.

The pupils by degrees dilated, the limbs became flaccid, the pulse rose to about 100, clammy perspirations ensued, and she died about 11 a.m.

P. M. 50 hours after death. The head only examined. Calvaria of moderate thickness. Dura mater natural, but firmly adherent, near the median fissure posteriorly to the surface of the hemispheres. It can be, however, detached without lacerating the cerebral substance. There is a deposit interposed between the dura mater and the brain, which is yellow, soft, and has an atheromatous appearance (? syphilitic). Arachnoid not opaque. Pia mater readily separable. Its vessels are engorged, especially posteriorly and on the left side.

On section of the brain, before arriving at the level of the ventricle, the substance of the left hemisphere is found to be lacerated, from extensive extravasation of blood, dark and fluid, mixed with coagula. The effusion extends into the anterior and posterior lobes and distends the ventricle. The right ventricle is also filled with fluid blood, but there is no laceration of the brain on this side. The effusion extends into the third and fourth ventricles, and there is laceration of the pons varolii, and about 2 oz. of fluid blood be-

neath the cerebellum. The consistence of the cerebrum appears to be normal, except in the left anterior lobe, at which site, a little external to the anterior cornu of the lateral ventricle, is a patch, of a straw-yellow colour, about the size of a small walnut. This is much softer than the surrounding tissue, and has a flocculent appearance on being subjected to a very gentle stream of water. There is not any vascularity around the mass. Cerebellum healthy.

The cerebral and basilar arteries are atheromatous, but there are no traces of calcification.

So obvious a relation appears to be established between the morbid state of the heart, of the cerebral vessels and of the kidneys, in the first group of cases, that, to an impartial observer, accustomed to the ordinary processes of reasoning, the evidence of their connexion will almost amount to demonstration. Nevertheless, this important connexion has been so greatly unrecognized or disregarded, that a prejudiced theory or defective pathology has respectively referred the apoplectic attack exclusively to disease of the cerebral vessels or of the heart, whilst the renal alterations, with rare exception, have altogether escaped attention. In proof I shall advert to distinguished writers, past and present, — foremost amongst whom stand Dr. Abercrombie and Dr. Burrows,—and then record the weight of the heart in apoplectics and non-apoplectics of the same mean age, the latter being derived from the tables of Dr. Clendinning.

To elude all fallacy, I shall make a few remarks on the age at which sanguineous effusion is most liable to occur.

Dr. Burrows maintains that the coincidence between

apoplexy and diseases of the heart is something more than accidental. Thus out of 132 cases of apoplexy and *sudden hemiplegia*, collected by him from his own practice and the works of Andral, Clendinning, Hope, and Guillemin, 84, or 63·6 per cent., presented *unequivocal* signs of cardiac disease. If the hemiplegic cases (which often depend on softening or cerebral embolism) are rejected, will not the percentage be higher ?—and if cases of undoubted (sanguineous) apoplexy *only* are included, will not the percentage be higher still ?

Dr. Burrows is of opinion that hypertrophy of the left ventricle must be admitted as a powerful predisposing or even exciting cause of apoplexy. Thus out of 34 cases of apoplexy (sanguineous ?) under his own observation, 23 presented disease of the heart—10 hypertrophy with valvular disease, 6 simple hypertrophy, 6 valvular disease, 1 simple dilatation of the cavities.

Dr. Copland also (without, however, resorting to facts in confirmation) believes that something more than mere coincidence exists between apoplexy and hypertrophy of the left ventricle.

Lacunec* also pointed out that simple hypertrophy of the left ventricle is, of all affections of the heart, that which most frequently gives occasion to apoplexy.

He mentions that several remarkable instances of this result are recorded in Monsieur Bertin's work,†

* Diseases of the Chest, translated by Dr. Forbes, page 590.

† On pourrait avancer, *a priori*, qu'un des résultats immédiats de l'hypertrophie du ventricule gauche sera une prédisposition à l'apoplexie . . . . l'observation ne le confirme que d'une manière trop positive (Maladies du Cœur et des gros vaisseaux, par R. J. Bertin, page 351.) He refers to Malpighi, Cabanis, and Ramazzini, who died of cerebral hemorrhage, being affected by marked cardiac hypertrophy.

and that the attention of practitioners has been more particularly called to it by MM. Legallois and Richerand.*

Dr. Todd, on the contrary, was opposed to the notion that hypertrophy of the heart gives rise to apoplexy, by sending blood with undue impulse to the head. He considered the hypertrophy to be merely necessary for the exigencies of the circulation, to preserve the force of the current as near as possible to the normal point, in spite of the existing obstruction —the actual force being most probably less than in health. But, notwithstanding the great authority of Dr. Todd, we are compelled to assert that this obstruction is, in most cases, purely hypothetical, and I believe the opinion of Dr. Law to be much nearer the truth; viz., that hypertrophy of the left ventricle of the heart, in order to produce apoplexy, must depend upon some impediment to the circulation, placed at a greater distance from the heart than the origin of the vessels which convey blood to the brain.†

The contributions of Dr. Hope to medical literature, chiefly derived from his practice at the St. Marylebone Infirmary—the fertile field, I may add, of my own later professional instruction—enunciate sounder doctrines on the subject than those of his predecessors and most of his successors. In the institution named, containing wards for children, in which every variety of malady to which juvenile flesh is heir is exhibited,

* Richerand held similar views. He thought that contraction (retrécissement) of the aortic orifice tends to diminish the influence of the hypertrophy.

† On Disease of the Brain dependent on Disease of the Heart, in Dublin Journal of Medical Science, vol. XVII.

the infrequency of *true apoplectic affections* in the young is remarkable, and the cases of *infantile* apoplexy collected by Dr. Quain, as proof of its non-rarity in early life, are rather to be viewed in the light of curiosities in morbid anatomy. That the greater number of these cases (to which I shall again refer) *were apoplexy*, even in a loose sense, will be shown to be more than problematical. The objection advanced against the statistics of Rochoux by Dr. Quain, that his practice was restricted to old persons, cannot be urged against Dr. Hope as tincturing his conclusions, it being apparent that ample opportunities were afforded the latter of witnessing the disease, if it occurred, at all periods from childhood to extreme age. According to the experience of Dr. Hope, apoplexy is most prevalent between forty and fifty and seventy and eighty years of age, and at these periods disease of the heart coexists in the proportion of nine out of ten, and ten out of eleven respectively. He insisted that the effects of simple hypertrophy and hypertrophy with dilatation of the left ventricle on the brain, are so important, that it is necessary to advert particularly to the subject, for the purpose of bringing it prominently into view. Instances of apoplexy supervening upon hypertrophy have been so frequently noticed, that *the relation of the two as cause and effect* he regarded to be one of the best established doctrines of modern pathology.* Yet the medical mind generally has failed to discern or admit the constancy and importance of alterations in the left heart in cases of sanguineous extravasation. Fully to bear out this assertion, I give a *résumé* of all

* Diseases of the Heart, 3rd edition.

the cases of cerebral hemorrhage which are to be found in the works of Morgagni, Cheyne, Portal, Abercrombie, Bright, Andral, Copeman, and in the Pathological Transactions. These sources are unobjectionable and convenient, as they are most frequently quoted, they contain considerable material for the inquiry, and are, collectively, fair illustrations, for the last hundred years, of the mode in which researches on the disease have been conducted.

On analysis of the cases recorded by Morgagni,* it might be imagined that the heart was usually normal; but there are references to the aorta and to other diseased conditions connected with the heart which justify the conclusion that he, unaware of its signification, if not altogether overlooking this viscus and other organs, merely noted in an incidental manner deposits on the valves and changes in the lining membrane of the vessels, without attention to augmentation in the walls or size of the organ, unless extremely manifested. Thus in Lib. I. Epist. III. art. xxxvi. is the only instance in which the heart is mentioned as enlarged (cor magnum), and he adds, in this particular case, "diligentius extra cadaver cor examinabam." Many of Morgagni's histories are to be met with in Copeman's Collection of Cases of Apoplexy. The following are all Morgagni's cases of intracranial hemorrhage:—

Lib. I. Epist. II.

1. A cardinal. 2. A man, 60. 3. A woman, 70.

In these respectively, with the exception of the extravasation, we read "omnia secundum naturam,"

* De Sedibus et Causis Morborum. Patavii, 1765.

"partes omnes reliquæ sanæ erant," "sana omnia in thorace."

4. An old man (senex). Brain only described.
5. Man, 70. Viscera not mentioned.
6. Man, 62. In ventre sana omnia, in thorace quoque.
7. Man, 22. Depended on laceration of internal carotid. Polypous concretion in right heart.
8. About 60. Viscera not referred to.

Epist. III.

Woman, 55. Valves of aorta very hard.
Man, 40. Heart itself not mentioned.
Woman, 40. Viscera not spoken of (? not examined).
Man, 73. Do. do.
Old man. Head only opened, amputated.
Man, 40. Head only opened, do.
Elderly man. Nihil in ventre aut thorace, aut quæ ad res præter naturam attineant.
Boy, 14. Head only opened. Appears to have resulted from disease of the cerebellum, as "æquo mollior est visa substantia."
Man. Cor magnum (already noticed).

Lib. V. Epist. LX.

Contains two cases of sanguineous apoplexy.

1. Old man. Valves slightly affected. Polypous concretion.
2. Middle-aged man. Heart very lax.

The other four cases in this epistle are not hemorrhagic, but apparently uræmic.

The statistics of Morgagni *over*-support the non-connection of cardiac lesions with hemorrhage into the brain, as in only one case is the heart mentioned as morbid; but as this distinguished anatomist was most sedulous in his description of the appearances within the head, to the exclusion almost of the other viscera, as may be inferred by his decapitations from the trunk,

for demonstration of the brain in the public theatre, little weight can be attached to his limited and negative pathological views.

All the cases are narrated without distinction, and as the history is often very scanty, some of them may have been of traumatic nature. One it is distinctly stated (Epist. II.) arose from a heavy fall, which must be excluded when the pathology of apoplexy is concerned; another in a man servant, aged 22 (Epist. II. art. xx.), depended on laceration of the internal carotid; and a third, in a boy of 14 (Epist. III. art. xxiv.), seems to have been *secondary* to cerebellar disease.

The following is an abstract from the work of Dr. Cheyne* of all the cases of *apoplexy* therein contained:—

1. Appears to have been delirium tremens. Recovery.
2. Vertigo. Recovery.
3. Puerperal convulsions. Recovery.
4. Apoplexy in a puerperal woman. Coagulum in left ventricle.
5. Death some time after submersion. No P. M.
6. Apoplectic attack. Recovery.
7. Death in bed. Appearance of inflammation of the meninges. Heart not examined.
8. Apoplectic attack. Extensive hemorrhage into brain. Viscera not mentioned.
9. Apoplexy. Hemorrhagic laceration of pons. Heart not mentioned.
10. Apoplexy. Hemorrhage into brain. Heart not mentioned.
11. Apoplexy (? traumatic). Coagula between the hemispheres and over surface of brain. Except the stomach and liver, viscera appeared sound.
12. Apoplexy. Surface of brain deluged with blood. Viscera not mentioned.
13. A drowned sailor.
14. Drunkenness in a distiller's man.

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* Cases of Apoplexy and Lethargy, by J. Cheyne, M.D. (1812).

15. Lingering death in a child of three years from administration of whisky.
16. Case of meningitis.
17. Hemiplegia. Recovery.
18. Hemiplegia. Result not stated.
19. Hysteria in girl of fifteen. Death from melæna.
20. Softening of brain ? Result not stated.
21. Softening. Result not stated.
22. Symptoms of dementia. Old disease and softening of brain. P. M.
23. Dotage. Apparently atrophy of brain.

Dr. Cheyne's first twelve cases are to be found in Dr. Copeman's Collection (Nos. 85–96), and also his sixteenth case (No. 97). Dr. Cheyne thought that the liver is often unsound in apoplectics; that it exhibits the kind of disorganization which is the effect of the abuse of ardent spirits.

As far as his recollection went, there is no morbid appearance in any other part of the body which is to be considered as belonging to apoplexy (page 25).

The cases of Portal* are worthless in relation to the presence of cardiac alterations in apoplectics, as the heart is alluded to (enlarged) in only very few cases, and all stertorous affections are described as apoplexy, this pathologist insisting (as did also Boerhaave) that stertor was distinctive of the disease.

After enumeration of engorgement, extravasation, effusion of serosity, hydatids, false membranes, scirrhi, polypous concretions and fungosities in the cranium, he remarks that he has often found changes in the aorta, arteries of the extremities, vena cava,

* Observations sur la Nature et le Traitement de l'Apoplexie. Paris, 1811.

valves of the heart, and *many other alterations in this organ* (plusieurs autres altérations dans cet organe,—page 330).

The following are all the cases of "apoplexy with extravasation" to be met with in the work of Dr. Abercrombie.* It will be seen that his inspections were for the most part limited to the head; hence imperfect. The heart, which was enlarged, is mentioned in only one case of sanguineous apoplexy (CXXXI.).

Case.

CVI.—Hemorrhage into brain. Arteries ossified.

CVII.—Hemorrhage into brain.

CVIII.—Hemorrhage on surface of brain from substance of right hemisphere.

CIX.—Hemorrhage into right anterior lobe.

CX.—Hemorrhagic laceration of left hemisphere.

CXI.—Laceration of right anterior lobe. Kidneys unusually vascular.

CXII.—Recent coagulum in right hemisphere. Old effusion into left hemisphere.

CXIII.—Ventricles of brain full of coagula. Arteries extensively ossified, and, at parts, of soft, pulpy consistence.

CXIV.—Extravasation over surface of brain which was healthy.

CXV.—Extensive coagulum over right hemisphere. Stomach healthy.

CXVI.—Extravasation over nearly the whole surface of the brain. Small coagula in right anterior lobe.

CXVII.—Coagulum in right cerebellar lobe.

CXVIII.—Coagulum beneath cerebellum surrounding the foramen magnum.

CXIX.—Disease of cerebellum and pons. Tubercular?

CXX.—Coagula in lateral third and fourth ventricles (? from extension from spinal canal). Age 9.

CXXI.—Aneurism of the meningeal artery.

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* Pathological and Practical Researches on Diseases of the Brain. 3rd edition, 1836.

Case.

CXXII.—Coagulum in right hemisphere. Remarkably diseased state of the whole arterial system of brain.

CXXIX.—Coagulum in right hemisphere. Cyst in left.

CXXX.—Coagulum in left ventricle. Old cysts in brain.

In the above nineteen hemorrhagic cases, occurring and communicated to Dr. Abercrombie, the history is often faulty, and there is no mention beyond what I have noted, of the state of the heart, cerebral vessels or kidneys; but in a few it is remarked that there was "no other morbid appearance." It is doubtful whether this relates to the brain or the viscera generally.

I next give the cases of cerebral hemorrhage from the works of Dr. Bright,* which are arranged under "Cases illustrating the occurrence of pressure from effusion of blood within the cranium."

Case.

CXXV.—Rupture of an aneurism of middle cerebral artery. Heart considerably enlarged.

CXXVI.—Blood effused over surface of brain. Viscera not mentioned.

CXXVII.—Blow on the head. Large quantity of blood over right hemisphere.

CXXVIII.—Fall. Subsequent symptoms, and much coagulated blood in skull.

CXXIX.—Effusion of blood into arachnoid. Heart remarkably large, but parietes feeble and thin.

CXXX.—Much semifluid blood in ventricles and laceration of brain. Heart appeared to be generally sound, and the muscular walls had the ordinary appearance of healthy muscle.

CXXXI.—Extensive hemorrhage over surface of and into brain. Viscera not mentioned.

CXXXII.—Extensive hemorrhage into brain. Heart rather large, cavities dilated, valves healthy.

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* Reports of Medical Cases, vol. II. (1831).

Case.

CXXXIII.—Clot in brain. Viscera not mentioned.

CXXXIV.—Hemorrhage into brain from disease of the basilar artery. Viscera not mentioned.

CXXXV.—Clot in thalamus. Heart large, left ventricle most unusually strong.

CXXXVI.—Large clot in right hemisphere. Viscera not mentioned.

CXXXVII.—Large clot in left hemisphere. Heart rather large. Valves healthy.

CXXXVIII.—Much dark grumous blood in brain. Coronary arteries of heart ossified, but heart itself not mentioned.

CXXXIX.—Old cysts and grumous blood in brain. Vessels of the body highly diseased. Heart not mentioned.

CXL.—Effusion of blood into right hemisphere. Heart not noticed.

CXLI.—Disease of corpus striatum. Hemorrhage?

CXLII.—Disease of corpus striatum. Apoplectic clot?

CXLIII.—Apoplectic cells. Heart very large. Decided hypertrophy of left ventricle. Kidneys granulated.

CXLIV.—Hemiplegia. Cyst in left thalamus. Heart considerably enlarged, left ventricle much thickened. Kidneys small, granular.

CXLV.—Imperfect hemiplegia (*left*). Yellow opaque deposit over *left* hemisphere. Scar of apoplectic cyst and induration of *left* side of brain. No trace of disease on right side. Heart large. Kidneys healthy.*

CXLVI.—Hemiplegia. Complete cyst in right hemisphere, containing clot of blood. Another cyst on same side. Head only examined.

From Case CXLVII. to CLXI. are hemiplegia, none of which proved fatal, which complete the illustrations of cerebral pressure from effusion of blood contained

* This case is peculiarly interesting, as the paralysis was on the same side as the lesion in the brain, as was confirmed by evidence of relatives, friends, and the medical officers of the hospital.

in the first part of the second volume. In the second part, under additional cases, are—

Case.
CCLXXVII.—Hemiplegia. Recovery.
CCLXXXVIII.—Clot in brain. Aneurism of cerebral artery. Viscera not mentioned.
CCLXXXIX.—Hemiplegia. Clot in right thalamus. Heart not mentioned.
CCXC.—Apoplectic symptoms. Recovery.
CCXCI.—Hemiplegia. Recovery.
CCXCII.—Apoplectic cyst in brain. Very extensive disease of arteries. Heart not mentioned.
CCXCIII.—Hemiplegia. Scrofulous tubercle in left thalamus.
CCXCIV.—Tumour in corpora quadrigemina, preceded by torpor and defective vision.

(The remaining cerebral cases are not hemorrhagic.)

Now, it will be observed, that in all the cases of Dr. Bright the heart in only one instance *appeared to be* generally sound. In most it is not spoken of; but we may presume that this physician habitually noticed changes in the organ, as he states (page 327), "on inquiring into the previous history, we not unfrequently find that strong evidence exists of disease in the heart and large vessels."

I have extracted from Andral* all his cases of intracranial hemorrhage. They testify how purposeless a collection of autopsies may prove, unless methodically directed towards the solution of pathological problems.

Case.
I.—Walls of heart hypertrophied.
II.—Doubtful case. Other disease of brain? Heart not mentioned.

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* Clinique Médicale, translated by Dr. Spillan.

Case.

III.—Hypertrophy of left ventricle.

IV.—Hypertrophy of left ventricle.

V.—Hypertrophy of both ventricles. Some ossifications of aortic valves. Arteries at base of brain presented numerous cartilaginous or bony patches.

VI.—Remarkable hypertrophy of left ventricle.

VII.—A doubtful case. Death from anthrax. Heart not mentioned.

VIII.—Old apoplectic cyst. Hypertrophy of left ventricle and pulmonary apoplexy.

IX.—Hypertrophy of left ventricle.

X.—Heart normal. Patient died of phthisis. There was chocolate-coloured cyst in right corpus callosum (? hemorrhagic).

XI.—Hypertrophy of the parietes of the heart. Cartilaginous incrustation on base of mitral valve. Adherent pericardium.

XII.—Greenish cyst in left cerebral peduncle. Heart not mentioned.

XIII.—A very extensive effusion, causing death in two hours. Heart not mentioned.

XIV.—Heart not mentioned.

XV.—Hypertrophy of left ventricle. Bony incrustation at base of mitral valve.

XVI.—A doubtful case. Death from carcinoma of stomach. Two serous cysts in hemispheres. Heart not mentioned.

Cerebellar Hemorrhage.

I.—Sanguineous effusion occurring during scirrhus of stomach. Heart and appendages natural. Patient aged 21.

II.—Hypertrophy and dilatation of auricles. Ventricles normal. Tricuspid, mitral, and aortic valves hard, thickened, and ossified.

III.—Hypertrophy of left ventricle. Points of ossification on aortic valves.

IV.—Effusion also into cerebrum. Heart not mentioned.

V.—Effusion into cerebrum and old clot in cerebellum. Heart not mentioned.

VI.—Effusion also into cerebrum. Heart not mentioned.

Out of the preceding twenty-two cases, the heart is stated to have been normal in two; but these two are *secondary*, and open to objection, and four others are

also doubtful; leaving sixteen free from cavil. Of these sixteen, eleven presented disease of the heart, seven of which appear to have been hypertrophy, four hypertrophy with slight aortic or mitral obstruction. In the remaining five cases the heart is not noticed; so these are inadmissible. In only one of the cases is reference made to the cerebral vessels.

The impartial reader of Dr. Copeman's collection* will admit that very many of the cases have little or no relation to the head, and are entirely unfitted for comparison or consideration with respect to apoplexy. Such cases tend to invalidate and throw unmerited discredit on statistics generally, by illustrating the laxness of nosological definitions.

Thus out of the 250 cases, of which I have made a careful table, originally intended, but found to be too long for insertion, there are only 156 post mortems, and in only 81 of these was extravasation of blood detected within the cranium. They are, nevertheless, of service by warning us to regard all accounts with suspicion, in which the symptoms and autopsy are not precisely narrated.

With the above author the history is scarcely ever full or complete, and in numerous instances there is absence, or at least no mention, of paralysis.

Of the 250, 76 recovered, leaving 174 for analysis; and of these only 156 were examined after death. Amongst the last (156), 81, or according to my estimate rather fewer, exhibited extravasations of blood within the head. The remainder of those opened seemed to depend on cerebral aneurism, tubercular

* A Collection of Cases of Apoplexy, 1845.

meningitis, renal disease, fever, poisoning (?), ramollissement, inflammation of the brain, cardiac disease, insolation, abscess of brain, delirium tremens, puerperal fever or convulsions, tumour, narcotism, hydatids, or other obscure or evident disease of brain. The morbid appearances, I must add, are often detailed in unsatisfactory language.*

Amongst the (94) recovered and unopened cases, we find examples of apoplectic seizure, apoplexy from fear, drunkenness, drowning, epilepsy, obscure coma, paraplegia, hemiplegia, poisoning (?), insensibility, paralysis without coma, tubercular meningitis (?), delirium tremens, vertigo, febrile attack (?), uræmia (?), puerperal coma and convulsions, embolism (?), cerebral aneurism (?), hysteria (?), lethargy, cardiac symptoms (?), otitis, fever, convulsions, and fright.

It should be recollected that the collection is drawn from Dr. Darwin, Fothergill, foreign journals, *Medical Gazette*, *Medico - Chirurgical Review*, Abercrombie, Cheyne, Pitcairn, Bright, Copland, Cooke, Morgagni, Willis, Portal, Lieutaud, Wepfer, Hall, Richond, etc., and the practice of the author; but some of the cases are originally copied from preceding writers.

On reviewing the entire series with reference to cardiac lesions, in connection with hemorrhage into the brain, I find that the following are the only cases in which the heart is alluded to where extravasation was observed within the skull.

Case
15. Hypertrophy of left ventricle.
34. Left ventricle hypertrophied.

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* As a sample, I may particulerly call attention to Case XXXIII., copied from the *Times* newspaper.

40. Heart enlarged and soft.
42. Heart preternaturally large.
106. Heart sound. Cavities of natural size. Slight deposit on aortic and mitral valves.
108. Heart rather large.
111. Heart large.
113. Heart rather large.
190. Left ventricle greatly hypertrophied.
206. Rheumatic disease of heart.
245. Heart normal.
246. Heart normal.

The two last cases are from the same author, and the heart is simply stated to be *normal*, which I am disposed to consider refers to its valves and orifices, not to bulk or weight. In the first the aorta was of "great size." In both there was disease of the cerebral vessels.

Lastly, I give a table of all the cases of clots in the brain from the Transactions of the Pathological Society of London, according to the recent Index of Mr. Holmes, under the heading of "Brain-clots, or Apoplexy." The following is the account of the heart and cerebral vessels.

Vol. I.

Page 36, No. 5. Heart not mentioned. Arteries of brain healthy.
„ „ 6. Heart loaded with fat. Arteries of brain extensively diseased.
„ 38. Apoplectic cell. Heart and arteries not mentioned.
„ 180. Heart enlarged. Arteries diseased.
„ 224. Heart much enlarged. Arteries diseased.

Vol. III.

Page 82. Heart 14 oz. Arteries atheromatous.

Vol. IV.

Page 15. Left ventricle thickened. Arteries atheromatous.
„ 19. Heart hypertrophied. Arteries diseased.

Page 22. Heart fatty. Vessels diseased.
„ 28. Heart not examined. Vessels calcareous and fatty.

Vol. VI.

Page 30. Heart 13 oz. Vessels atheromatous.
„ 36. Nothing worthy of remark in thoracic and abdominal viscera. Vessels atheromatous.
„ 39. Heart fatty. Vessels atheromatous.
„ 383. Heart hypertrophied. Vessels atheromatous.

Vol. VII.

Page 72. Heart 16½ oz. Vessels not mentioned.
„ 122. Aneurism of basilar artery.

Vol. VIII.

Page 33. Organs healthy. No degeneration of vessels discovered.
„ 140. Heart natural in size. Rupture of posterior cerebral artery.

Vol. IX.

Page 7. Heart 19 oz. Cerebral arteries healthy.
„ 18. Heart not mentioned. Atheroma of arteries.
„ 69. Heart enlarged. Vessels diseased.
„ 124. Heart 24 oz. Cerebral vessels atheromatous.
„ 334. Heart 21 oz. Vessels atheromatous.

Vol. X.

Page 3. Heart *healthy, but weighed* 12 *oz.* Aneurism of anterior communicating artery.
„ 4. Hydatids in a boy. No extravasation.
„ 21. No extravasation. Syphilitic disease of brain and obstruction of arteries.
„ 54. Extensive disease of brain. Old clot. Heart perfectly healthy (doubtful case).

Vol. XI.

Page 3. Clot in brain of a girl aged fourteen. Heart and vessels not mentioned.

Page 72. Heart natural. Vessels not mentioned.
„ 84. Heart enlarged and fatty. Vessels atheromatous.
„ 222. No hemorrhage.
„ 229. Heart and vessels not mentioned.

Vol. XII.

Page 2. Heart not mentioned. Vessels atheromatous.
„ 6. Nothing unusual in thorax. Vessels atheromatous.
„ 15. Thoracic viscera healthy. No disease of cerebral arteries or capillaries.
„ 16. Body not examined. Cerebral vessels not mentioned.

Vol. XIII.

Page 7. Heart and vessels not mentioned.

Vol. XIV.

Page 55. Heart healthy. A few vessels atheromatous.

Vol. XV.

Page 4. Left ventricle greatly thickened. Vessels not mentioned.
„ 8. Heart not mentioned. Arteries atheromatous.
„ 9. Heart healthy. Atheroma of arteries.
„ 13. No extravasation.

Of these 42 cases, granting even that not any were traumatic, 5 must be excluded, as 4 are not hemorrhagic, and one is doubtful.

With respect to the condition of the heart in the remaining 37 cases, in 18 it was markedly diseased; in 11 it is not mentioned or was not examined; in 8 it is recorded as healthy, or as displaying nothing unusual; but as the case in vol. X. page 3, is included amongst the last, though the heart weighed 12 oz., it justifies me in iterating the belief that many, if not all, of the attacks were attended with hypertrophy or other alterations in the parietes or bulk of this

organ; but as the valvular apparatus remained normal, less obvious changes passed unheeded and the weight was not ascertained.

As to the state of the vessels in the 37 cases, 25 (including aneurism) presented disease of the cerebral arteries; in 8 the vessels are not alluded to; in 4 they are said to have been healthy.

It is necessary to make a few remarks concerning the occurrence of cerebral hemorrhage in infancy and early manhood.

In the *London Journal of Medicine* for January, 1849, two cases of apoplexy in childhood were published by Dr. Richard Quain. One occurred in a boy, aged nine years, which terminated fatally in seven hours. There was a large clot in the right hemisphere, external to the ventricle, and passing downwards to the corpus striatum. The heart was enlarged —between 5 and 6 oz.,—and the left ventricle hypertrophied. The valves appeared competent, but those of the aorta were stiff like parchment. The vessels of the brain were visibly ruptured, but are not otherwise described. It is uncertain whether they were healthy, as there was a change in a part of the vascular system (the aorta). The second case was one of asthenic or cachectic meningeal apoplexy in a girl two years and seven months old. There was effusion into the arachnoid and at base of brain, purpuric and passive in origin; therefore intercurrent, and not acceptable for illustration or analysis.*

* Out of 386 cases of effusion of blood within the cranium compared by Andral, there is not one of meningeal hemorrhage.

The cases of hemorrhagic extravasation—cerebral and meningeal—collected with much industry by Dr. Quain, occurring in children, which fell under the observation of other practitioners, are not put forward in detail; but from the scanty account of some of them it may be gleaned that they were dependent on previous cerebral disease, injury, purpura, or other antecedent morbid state, so as to fairly entitle them to the appellation of secondary or intercurrent, and consequent exclusion.

The following are *all* the fatal cases of (so called) *apoplexy* in persons of *twenty years and under* in Dr. Copeman's collection :—

No.		Age.
8.	Clot in brain. Heart not mentioned. Aneurism . .	20
16.	Tubercular meningitis. No hemorrhage	11
19.	Intercurrent tubercular disease of brain. No hemorrhage	18
27.	Clot in brain. No mention of heart or vessels . .	20
71.	Hemorrhage into brain (from Abercrombie) . . .	18
102.	Rupture of a cerebral aneurism	19
110.	Hemorrhage into brain. Disease of basilar. Aneurism ?	20
152.	General dropsy, evidently renal. No hemorrhage . .	20
157.	Found dead. Disease of brain. Not apoplexy . .	20
159.	History and appearances of tubercular meningitis . .	18
172.	Epilepsy. Inflammatory disease of brain. Not fairly apoplectic	12
175.	No hemorrhage. Very doubtful case	20
176.	Case of meningitis ? No hemorrhage	19
183.	Two spoonfuls of black clotted blood in ventricles and substance of cerebellum	14
203.	Extension of otitis to meninges. No hemorrhage . .	20
205.	Puerperal convulsions. No hemorrhage	20

There is another case in a child of seven years (151), which died comatose after measles, and a large

quantity of blood and water (? sanguinolent serum) was found in the brain.

Of the foregoing sixteen cases, only six presented evidence of hemorrhagic extravasation, one of which was due to the rupture of an aneurism. The remaining five are imperfectly recorded and are almost without history.

They are as fairly explicable by the same pathological change, which is most common in young subjects, as by assumption of primary intracranial effusion. There was disease of the basilar in one instance. With the exceptions observed, the condition of the heart and cerebral vessels remains unmentioned.

Abercrombie, with his vast experience, speaks of only one case of true apoplexy as early as the ninth year (Case CXX.), and it is evident from the symptoms and the "effusion of coagulated blood enveloping the whole extent of the cord," that it was extension of hemorrhage from the spine upwards. As usual, the vessels of the brain and state of the heart are not noticed.

Andral* met with a case of cerebral hemorrhage in a boy of twelve; but no particulars are given. Dr. Copland also has seen a case of true hemorrhagic apoplexy at eighteen years, but the case is not related; and in another of Dr. Abercrombie, at the same age, the account (Case CX.) of the *post mortem*, which is limited to the head, is short and unsatisfactory. Not to enlarge unnecessarily on this subject, I may refer to Dr. West† as a decisive authority. He relates two

* Pathological Anatomy, vol. II. p. 723.

† Diseases of Infancy and Childhood, 3rd edition.

instances in infants, of hemorrhage into the sac of the arachnoid; and observes (page 43) that it is most fallacious to associate effusion of blood upon the brain, in the case of children, with rude health and general plethora. It is much more frequent in the weakly than in such as are robust. There seems to be reason for supposing that the hemorrhage is sometimes purely passive and dependent on altered state of the blood (cachexia). He further adds that *hemorrhage into the substance of the brain*, though extremely rare in infancy and childhood, does sometimes occur. He has *only twice* met with distinct extravasation of blood into the substance of the brain in children. Once in a girl aged eleven months, which was due to impediment to the circulation through the brain, produced by an attack of phlebitis and occlusion of the sinuses of the dura mater. There were head symptoms a long time before death, old pleurisy and peritonitis. The other instance was in a girl aged eleven years, subsequent, at some interval, to necrosis and abscesses. On examination, there was distinct softening of the brain around the coagulum.

Now, both these cases may be justly regarded as cachectic, or of secondary kind. They are included in the table of Dr. Quain, as also is the following one, narration of which, I think, renders further allusion to this point superfluous.

It occurred in a female child aged two and a half years, "of scrofulous constitution, always fed upon very poor diet, consisting principally of oatmeal, and for many months subject to great functional disorder of the abdominal viscera."* After death, a large quantity of

* Case of sanguineous apoplexy in *London Medical and Physical*

blood was found poured out upon the surface of the brain; but the substance of the brain itself was without disease?

The liver, spleen, and kidneys were unusually large, as were also the mesenteric glands. No mention is made of the state of the vessels, and we may confidently refer this case of meningeal apoplexy to hemorrhagic or cachectic dyscrasia.

The above facts abundantly prove the rarity of true apoplectic seizures in children, of a primary nature, in spite of Dr. Quain's researches. In addition, although I have examined the brain in a very large number of children, of all ages, I am unable, except in connection with recent birth,* to call to mind one case of idiopathic cerebral hemorrhage, and I fearlessly appeal to the profession generally, in confirmation of my own experience.

For the sake of convenience and clearness, I will in this place speak of the age at which apoplexy is believed to be most common. Dr. Quain holds that the liability to *first attacks* of apoplexy is greatest

Journal (No. XLVII.) for 1822, by T. H. Greenhow, Esq., of Newcastle.

* In the newly-born the hemorrhage is almost always into the arachnoid cavity, according to Cruveilhier, who terms it "apoplexie veineuse." I believe it only occurs where there is congenital defect of nutrition (dyscrasia), as all infants are subjected, though very few are obnoxious to, the pressure incident to the parturient act. Cruveilhier remarks, "les apoplexies des enfans nouveau-nés sont-elles autre chose que le résultat de la stase du sang veineux par suite de la compression des veines jugulaires ou de la veine ombilicale?"—*Dict. de Méd. et Chir. Pratiques*, tome iii., art. Apoplexie, prop. VIII.

between 40 and 50 in males, and between 50 and 60 in females. Dr. Hope, as already remarked, thought the disease to be most prevalent between 40 and 50 and 70 and 80. He does not refer to influence of sex. Dr. Copland thinks it to be most common in the far-advanced periods of life — most usual between 40 and 70. Dr. Watson says it is common after 50; and Dr. Bennett thinks it most rife between 50 and 80. Dr. Bright thought apoplectics were chiefly above 40, and many above 60; and Portal that the disease was most frequent after 60. Dr. Cheyne met with most cases of apoplexy between 50 and 70. Hippocrates deemed persons to be most subject to it between 40 and 60;* and Sauvages, at 60 years of age.†

According to my own observation, which agrees with that of Cullen and Rochoux, the disease is most common between 60 and 70, especially when the diminished number of the population, at this decenniad, is considered. At any rate, it may be affirmed to occur most often after the fiftieth year, and oftener in males than females.

Respecting the influence of season, I may briefly state that Cullen believed persons to be most liable to attack in the spring. The returns of the Registrar-General are so vitiated by the insertion of various forms of coma, under the generic term "apoplexy," that the statistics, in reference to cerebral hemorrhage are rendered inadmissible, few of the cases being

* Ἀπόπληκτοι δὲ μάλιστα γίνονται ἡλικίῃ τῇ ἀπὸ τεσσαράκοντα ἐτέων, ἄχρις ἑξήκοντα.—Sect. VI. Aphor. LVII.

† Apoplexia sanguinea sæpius sexagenarios adoritur (p. 497).

authenticated by necrotomy. Dr. Maclachlan considers apoplectic effusion to be greatly more frequent in winter than in summer, which agrees with the researches of M. Falret, who believed hemorrhages of the brain in Paris to be more common in winter than in summer and spring; but this does not accord with the experience of M. Rochoux, whose cases (69) were almost equally distributed amongst the seasons.

To avoid imputation of error, I give the mean weight of the heart in all my apoplectic cases—viz. 13·7 oz., the average age, including males and females, being 65 years.

According to Bizot, as life advances, the heart enlarges in all its dimensions,—length, breadth, and thickness, especially the left ventricle and septum.

Bouillaud gives the mean weight from 25 to 60, from 8 to 9 oz., being a little more in large and muscular subjects, slightly less in females. (This of French hearts.)

According to Dr. Clendinning, from 50 to 60 years the heart averages $10\frac{1}{6}$ oz.;

$$\text{After 60 to } x \begin{cases} 10\frac{1}{2} \text{ oz. in males.} \\ 8 \text{ oz. in females.} \end{cases}$$

Dr. Reid makes the estimate slightly higher; but it will be seen that the heart in apoplectics is greatly enlarged, to the extent of, at least, 3 oz. on the average, in my cases. The lowest weight in any case was $10\frac{1}{2}$ oz., *in a female* aged 72.

To return to the degenerations in the coats of the cerebral vessels, in cases of sanguineous extravasation.

Steatomatous and other alterations in the vascular tunics are alluded to by Morgagni, Monro, and Haller; and Dr. Baillie* pointed out, in 1793, the frequency of bony or earthy matter in the arteries of the brain, when spontaneous hemorrhage occurs within the cranium.

Mr. Hodgson, in 1815,† affirmed that he had rarely examined a case of apoplexy, not arising from accidental violence, which did not exhibit a morbid condition of the arteries of the brain. He held that the same alteration of structure in the coats of the arteries, which, in other parts of the system, by causing ulceration and rupture, gives rise to aneurism, in the vessels of the brain produces apoplexy. Apoplexy, indeed, when it arises from the rupture of an artery, may be regarded as a species of aneurism.

Now this gentleman, with the exception of Baillie, appears to have been one of the earliest to insist *fully* on the changes in the vessels, as in "Cullen's Practice," edited by Dr. Peter Reid (1810), there is not any allusion to disease in the cerebral arteries.

Dr. Cooke ‡ does not refer to the heart or vessels in connection with sanguineous apoplexy. In fact, the learned work of this physician is rather an elaborate epitome of the opinions of others, than an expression of his own views.

Dr. Hope, however, did not omit to perceive the importance of arterial degeneration. He observes that in the arteries at the base of the brain, calcareous

* Morbid Anatomy, p. 466.

† On Diseases of Arteries and Veins.

‡ Treatise on Nervous Diseases. 1820.

and other alterations are remarkably frequent, and are a principal cause of rupture of the vessels and apoplectic effusion. He imagined it rare indeed to meet with instances of such effusion, exclusive of those from external violence, in which some disease of the arteries may not be detected; and urged it as remarkable that disease of the artery is in general connected with hypertrophy of the left ventricle. Yet he thought it incorrect to suppose that the catastrophe was peculiar to hypertrophy of the heart, as he had seen several instances in which dilatation of the organ was associated with sanguineous apoplexy. Nevertheless, how few pathologists have acknowledged or adopted these facts in all their correctness!

Thus, Dr. Todd evidently entertained the most erroneous opinions concerning the condition and influence of the heart, and believed that apoplexy was alone due to the diseased state of the arteries, and the diseased state of the brain, which imperfectly supported them. Dr. Abercrombie, again, alludes in his work in only one instance to the heart, and maintains that ossification of the arteries, which is a common appearance in elderly people, is a very frequent source of apoplexy, with extravasation of blood in advanced periods of life.

Dr. Clutterbuck scarcely alludes to the pathology of cerebral hemorrhage, but notices that in old persons, the arteries of the brain frequently become diseased from ossification.

M. Rochoux* defined apoplexy as "une hémorragie par rupture, suite d'une altération du tissu

* Dict. de Méd., art. "Apoplexie cérébrale."

propre de l'encéphale." He thought that cardiac affections were not frequent in apoplectics, as he found only three cases of aneurism of the heart (un état aneurismatique du cœur) amongst forty-two patients; but unless we are assured, as Andral adds, that he comprehended hypertrophy of the organ under the term "aneurism," his researches on this subject are not to be depended on.

Dr. Copland remarks, that in a considerable proportion of cases of sanguineous apoplexy, the arteries of the brain are either ossified or otherwise diseased.

Laennec, who insisted on the connection of heart-disease and apoplexy, makes no allusion to the condition of the cerebral vessels.

Andral, in like manner, in his necropsies, in the "Clinique Médicale," refers, in only one case, to the arteries of the brain; and in his "Pathological Anatomy,"* he remarks that, in a great many cases of hemorrhage in the brain or spinal cord, there is no trace discoverable of alteration in the vessels of the part. But he believes the truth of the assertion, that hypertrophy of the heart is frequently observed in apoplectic patients, and is one of the causes of the hemorrhage.

Dr. Bennett thinks the most important alterations are the various changes which occur in the heart and lungs, as the obstruction they occasion to the circulation may produce a state of the brain highly favourable to apoplexy. He mentions thickening of the pericardium, collections of serous fluid in its cavity, and adhesion of its layers, thickening and ossification of

* Vol. II. p. 114.

the aortic valves; and quotes Dr. Watson, Legallois, and Lallemand as to the not infrequent complication of hypertrophy of the heart. In a like indiscriminate manner, he speaks of the lungs, as occasionally condensed, congested, hepatized, or disorganized by tubercles or calcareous concretions; and observes that an exciting cause in old persons may be rigidity, or earthy deposits in the vessels.

Rokitauski * believes the general proximate causes of apoplexy to be *congestions*, *excessive action of the heart*, and *disease of the blood-vessels*. He states that hemorrhage very commonly occurs in the brain in persons labouring under simple hypertrophy, or hypertrophy with moderate dilatation of the left ventricle. The coincidence of apoplexy with this disease of the heart is so constant as to afford grounds for stating it as a rule (constitutio apoplectica cordis), (vol. III. p. 398).

Dr. Burrows, an advocate for the dependence of apoplexy on cardiac disease, adverts, it must be confessed, to the alterations in the vascular system of the brain, but lays no great stress on their importance.

Dr. Walshe, in his oral lectures, affirms that the chief agent in the production of apoplexy is atheromatous alteration in the arteries; and he adds the reminder, that young subjects may exhibit such deposit in rare cases.

Dr. Markham † expresses his belief that a consideration of the history of the cases of apoplexy, and of fatty degeneration of the heart, independent of its

* Pathological Anatomy, translated by C. H. Moore, F.R.C.S.

† Diseases of the Heart, 2nd edition, Appendix Y.

rupture—recorded in the "Pathological Transactions" —quite justifies us, at least, in greatly doubting whether mere obstruction to the cerebral circulation, situated in the heart, or any violence in the contractions of the left ventricle, is sufficient to produce sanguineous effusion into or upon the brain, unless the cerebral vessels be more or less impaired by disease—be softened or rendered brittle through the presence within their coats of oily, earthy, or fatty matters; and whether, in fact, apoplexy ever occurs unless disease of the vessels pre-exists.

Mr. Paget * draws attention to a fact of some weight; namely, that the cases in which *atheromatous* changes were first observed in the minute vessels, were cerebral apoplexies, in which the hemorrhage appeared certainly due to rupture of the wasted and degenerate blood-vessels. As a surgeon, he makes no allusion to the throax and abdomen.

Dr. Maclachlan,† a very recent and high authority, states that ossification, or cretification, and fatty degeneration of the cerebral and smaller vessels are very common in advanced life. In 94 persons over 60, 42 of whom were free from any vestige of disease of the brain, Durand Fardel found the vessels *apparently* healthy 22 times; they were thickened or ossified in the remaining 72 cases. Ossification existed in 16 instances only (p. 80). He adds further, that so constantly are the cerebral arteries in a calcareous or fatty state in persons above 55 or 60, dying of this disease, that we are irre-

* Lectures on Surgical Pathology, p. 108.

† On the Diseases and Infirmities of Advanced Life.

sistibly led to the conclusion, that this is, of all others, the most important predisposing cause, the one to which we may unquestionably ascribe the chief frequency of apoplexy in advanced life (p. 134). He elsewhere repeats, that in persons above 50, the most frequently associated lesion is a diseased state of the cerebral arteries, *generally fatty degeneration.*

Next to disease of the vessels, Dr. Maclachlan thinks organic alterations in the heart may be ranked in importance, as causing and predisposing to apoplexy, though he views simple hypertrophy as the most constant change, but the least injurious. I cannot agree with this conclusion; yet I venture to express my admiration at the philosophic reflections, and at the felicitous truthfulness of the descriptions, especially concerning the modifying influence of age in masking and peculiarizing most disorders. Few who have practised much amongst old persons will fail to appreciate the observant acuteness and lucidity with which this learned physician portrays "the diseases and infirmities of advanced life."

In contradiction to those who discredit any correspondence between the condition of the heart and sanguineous apoplexy, I advance the observations of pathologists who have specially committed themselves to this branch of inquiry, such as Dr. Hope, Dr. Burrows, Laennec, Rokitauski, and Dr. Maclachlan, together with the testimony inadvertently rendered by published reports of cases and the valuable proof afforded by comparison of the weight of the heart in my own cases of apoplexy, with its weight in the non-apoplectic, derived from the careful statistics of

Dr. Clendinning. Most of the authors quoted, who are in opposition to the foregoing, speak with but little definiteness or precision, not having particularly and personally devoted themselves to the subject, and being obviously biassed, from deference to precedent or traditional authority.

On the other hand, with regard to the changes in the coats of the cerebral vessels, may I not almost say that they are *universal* (?) after a certain age, varying, of course, in date according to the mode of life and original constitution of the individual. These degenerations most frequently partake of an *atheromatous* character, consisting of a deposition or substitution of fatty molecules and cholesterine, in lieu of the ordinary textural elements of the vessels. Or the changes may be rather of earthy or calcific nature, though rarely uncombined with the preceding alterations. Such condition necessarily impairs the tenacity and elasticity of the arteries of the brain, and predisposes to rupture of their walls. It is a matter of some dispute what is the primary cause of production of the changes in the vascular tunics. Dr. Hope was inclined to believe they arose from over-distension of the arterial walls by the force of the circulation, as they are most liable to occur in those situations exposed to over-distension, as the arch of the aorta and the arteries of the brain, and are especially incident to the aged, in whom the vessels sustain a diminution of elasticity and cohesiveness. But the more enlightened pathological knowledge of to-day ascribes them to degenerative alterations, or impairment of the nutritive operations, for the most part, if not entirely, unconnected with inflammation. In fact, their approach indicates a dis-

position to physical debility, and a proneness to perishability and decay; and in advanced life is to be viewed rather as an almost unavoidable senile degradation than a feature of essential disease. But these degenerations may occur in much earlier years, in consequence of original dyscrasia or cachexy; or ensue as a sequel of excesses, syphilis (?), abuse of alcohol and mercury, bad diet, mental anxiety, and other heteræmic influences, by which the nutritive forces are lowered or perverted. It is beyond question that these causes exert powerful agency in accelerating the access of degenerations, which, in the more vigorous, better nourished, cheerful, and abstemious, are postponed to the evening of life. That it is possible for changes in the blood-vessels of an atheromatous nature to take place in early life, is upheld by Dr. Walshe and Mr. Hodgson; and Andral also witnesses that vascular ossifications are not confined to old persons, as he very often met them from the age of forty to sixty; several times in persons from twenty-five to forty, and once he ascertained the existence of a very remarkable state of ossification in the heart of an individual who was not eighteen years old when he died (Clin. Méd.), and in his Pathol. Anat. (vol. II. p. 392) he refers to Bichat, Baillie, Young, and Dr. Wilson, the latter of whom saw the aorta ossified at the age of three years. The period of invasion of atheromatous and earthy deposits in the coats of the arteries of the brain must be therefore regarded as fluctuating and indeterminable in any given instance, being governed by the state of health, vigour, previous habits and method of life of the particular subject; though *normally* (?) occurring in all (sufficiently) old persons.

Yet the exact relationship to each other and the proximate causes of fibrous, atheromatous, and cretaceous deposits, are not thoroughly and definitely understood. For example, is atheroma a normal (involutional) product, and morbid only when occurring prematurely? If so, what is the ordinary epoch of its appearance, and is its earlier approach dependent chiefly on original or acquired cachexia? Is it always necessarily antecedent to calcareous precipitation, or usually, rarely or never convertible into the latter; and may the latter be primary and independent of previous alterations, or is it mostly a sequence of fibrous thickening of the vascular tunics? Or, when atheromatous and cretaceous depositions are coincident, are they invariably, and to what extent, connected, or are they merely accidentally concomitant?

Dr. Handfield Jones thinks that calcareous degeneration is rarely primary but mostly consequent on atheroma, but he believes that earthy deposits may take place in the vessels of the aged, without previous fatty or atheromatous degeneration. This I also believe, and that when cerebral hemorrhage occurs, it is in connection with atheromatous rather than earthy alterations, though the two be co-present. It is undeniable that the tendency to calcification of the vascular and other textures increases directly with the age; but beyond a certain time of life—over ninety-five—the liability to apoplexy greatly declines, as I shall immediately prove by reference to statistics.

I have specially noted the cerebral vessels as natural to the unassisted eye, in persons of 52, 54, and 55 years of age. Nevertheless my investiga-

tions lead me to conclude that individuals rarely pass their fiftieth year without the access of structural changes of atheromatous or other description, capable of being detected, by minute dissection, in the walls of the cerebral arteries.

With regard to the presence of *arcus senilis*, or fatty degeneration of the cornea, Mr. Canton* believes, with Mr. Paget, that it is the best exponent of proneness to extensive or general fatty degeneration of the tissues. According to Mr. Canton, it is not until about the age of fifty, as a common rule, that it begins to be formed. It may, however, be noticed much earlier, or, on the other hand, a century may be arrived at without the cornea being invaded by fatty degeneration. He has remarked a well-marked double arcus in a youth of 16, and in his brother a boy of 12; and he records instances of persons aged 82 and 103 in whom there was not a vestige of the arcus senilis. Amongst the Chelsea pensioners in fifteen cases where the ages varied from 68 to 96, he found the arc was entirely absent. According to some, the arcus has been met with in infancy. Mr. Canton does not deem it an infallible indication, as it may be prematurely formed and increase, without the constitution, *apparently*, being undermined by debility, and he would not imply that its existence is necessary in all cases of fatty degeneration of internal organs.

According to Dr. George Johnson,† Bright's disease is a fatty degeneration of the kidney, and he uses these terms synonymously. He points out that it rarely

* Canton on Arcus Senilis.

† Med. Chir. Trans., vol. XXIX.

happens that a patient dies of Bright's disease without presenting more or less atheromatous or fatty degeneration of the arteries, though this condition of the arteries is by no means confined to such subjects, as, in various degrees, it is an extremely common morbid appearance in persons *above the age of* 30, who die in the London Hospitals.

In certain instances, as Dr. Johnson has also shown, the renal degeneration depends on fibrinous or albuminous accumulation in the tubuli and cortical substance; but it is here unnecessary to consider the varieties of morbid kidney which are collectively grouped, by different authors, under the head of "Bright's disease."

Dr. Basham* also has called attention to the connection of kidney disease with granular and fatty degeneration of the arteries of the brain and of the heart fibre. For a full account of the subjects of atrophy, of degeneration of vessels and organs, and of senile changes, the reader is referred to the admirable reflections in Mr. Paget's "Surgical Pathology."†

* On Dropsy connected with disease of the kidney, &c.

† I beg leave to quote the description of this gentleman as to the changes observable in the minutest cerebral vessels. "In a single fortunately selected specimen one may see, in different branches of a vessel, all degrees or states of the degeneration—the less and the more thickly scattered minute oil-particles, the clusters of such particles in various sizes and shapes, and the larger particles like drops of oil. . . . The vessels most liable to this disease are, I think, the arteries of about 1-300th of an inch in diameter; but it exists generally at the same time in the veins of the same or of less size. It is not rare to find vessels of from 1-2000th to 1-3000th of an inch in diameter, having parts of their walls nearly covered with the abnormal deposit" (p. 107).—See also Mr. Gulliver in Med. Chir. Trans.

Rostan and Abercrombie thought that arterial ossification was the source of that condition of the brain which terminates in ramollissement, and none will disavow the view of Dr. Todd, that any state of the cerebral arteries or of the general system, which may impair the nutrition of the brain, is favourable to the production of white softening. Dr. Todd observing how frequently this softening surrounds apoplectic clots, deemed it, in such cases, to be the most probable precursor of the effusion, and Rochoux insisted that there is always a previous softening of the brain (*ramollissement hémorragipare*) in cases of sanguineous apoplexy. Cruveilhier, on the contrary, thought the softening always (*toujours*) consecutive or simultaneous, and only primitive (*sic*) in rare exceptional cases.

It certainly is an exceedingly difficult point to solve; but as the vessels are *always* (?) found diseased, it is a reasonable presumption that the surrounding tissue is morbidly involved, from defective nutrition. It would clearly be next to impossible to demonstrate it as a fact, in the walls of a hemorrhagic cavity recently lacerated by sanguineous extravasation. Dr. Bennett thinks it probable that softening sometimes precedes, sometimes follows cerebral hemorrhage. It should be recollected, that, on second or third attacks, hemorrhage may recur in the same locality, and obliterate, from its magnitude, the vestiges of pre-existent softening, cysts, or cicatrization.

Out of 426 cases of effusion of blood into the brain, collected by Dr. Aitken,* from Andral, Tacheron,

for 1843, and Mr. Moore on Diseases of Arteries, in Holmes' System of Surgery, vol. III.

* Practice of Medicine.

Serres, Lerminier, and the "Pathological Transactions of London," 202 occurred into and on a level with the *corpora striata*, 66 into these bodies, 44 into portions of the hemisphere above the *centrum ovale*, 39 into the *thalami optici*, 16 into the lateral lobes of the cerebellum, 12 into the mesocephalon, 11 into the posterior lobes of the cerebrum, 10 in front of the *corpora striata*, 10 into the *pons Varolii*, 5 into the meninges, 5 into the middle lobe of the cerebellum, 3 into the cerebral peduncles, 1 into the peduncle of the cerebellum, 1 into the *corpora olivaria*, 1 into the pituitary gland.*

The previous argument appears to me to warrant the following conclusions, which I consider to be scarcely controvertible.

That apoplexy (cerebral hemorrhage) is unquestionably very rare in early years, and is admitted to be most common after the meridian of life. It is, in fact, "l'apanage de la vieillesse." At any age the proportion of apoplectics to the population and deaths of that age is limited.

Thus, on referring to the last census year, which is convenient for the purpose, and fairly represents the annual mortality, I find the population of London on April 8th, 1861, was 2,803,989.

The mortality in London for 1861 was 65,251.

The deaths from apoplexy (so registered), 1,486.

* Consult also Rochoux, in Dict. de Méd., tome IV. pp. 111-2, art. "Apoplexie cérébrale." For a description of hemorrhagic effusions, their consequences and changes, see art. "Nervous System," in Cyclop. of Anat. and Physiol., by Dr. Todd; and Rokitauski's Path. Anat., vol. III. p. 387 et seq.

Of the 65,251 deaths from all causes there were :—

Under 1 year	15,076
1 year	7,105
2 years...	3,500
3 „	2,049
4 „	1,360
Total under 5 years...	29,090

5 years...	2,464
10 „	1,092
15 „	1,411
20 „	1,780
25 „	4,158
35 „	4,791
45 „	4,881
55 „	5,442
65 „	5,419
75 „	3,779
85 „	868
95 „ and upwards ...	76

Of the deaths from apoplexy there were, of both sexes :—

Under 1 year	98
1 year	43
2 years...	14
3 „	17
4 „	10
Total under 5 years...	182

5 years...	22
10 „	7
15 „	18
20 „	18
25 „	59
35 „	122
45 „	200
55 „	322
65 „	302
75 „	203
85 „	30
95 and upwards... ...	1

So that about one death in every 44 was certified as apoplexy; but it is certain that many cases, especially in young subjects, returned under this head, were referable to other cerebral disorder, and, on the contrary, it is not unlikely that a few cases of apoplexy were included under "Brain disease."

Now, although the returns are, in a measure, fallacious, it is evident that the fatality of apoplexy varies greatly in proportion to the deaths at different ages. From the Registrar-General's return of 1861 it is shown that under 20 years one death from apoplexy was recorded in about every 148 deaths in the metropolis; between 20 and 45 in rather less than 54 deaths; whilst *between the ages of* 45 *and* 85 *one death from apoplexy occurred in about every* 19 *deaths*. That is, according to the annual tables, the disease was nearly eight times as fatal (how many times more frequent?) in proportion to the deaths, as it was under 20 years. Or, to calculate by another method,—still computing by the census,—I find that in the year 1861

the deaths in London, *at all ages*, from apoplexy, *in proportion to the population*, were 1 to about every 1,887 persons living. Under 20 years of age the deaths from the same disease were about 1 to 5,179; between 20 and 45, 1 to about 5,631; whilst between 45 and 85 they attained to 1 in every 481 of the population. From the age of 95 upwards the frequency of the disease markedly diminishes. It will be observed that of the seventy-six deaths beyond this period only one is ascribed to apoplexy, and in the mortuary returns for ten years—from 1853 to 1862—the total number of deaths *from all causes* and *from apoplexy*, in persons above ninety-five was as under:—

	Deaths.	Apoplexy.
1853	56	1
1854	61	0
1855	53	2
1856	42	1
1857	54	2
1858	74	1
1859	62	0
1860	59	3
1861	76	1
1862	62	0
	599	11

Hence, at this term of life, if we accept the return, apoplexy proves fatal once in about 54·5 deaths, which testifies to a remarkable decrease in the liability to the disease at advanced periods; and this decrease would, I am assured, be more conclusively manifested were

uræmic cases excluded, which, contrariwise, are frequent in age. (Though not bearing directly on the subject, it may be observed, that of the mortality over 90 years nearly two-thirds are referred to old age, the remainder being classified under "causes not specified," "bronchitis," "dysentery," "dropsy," "paralysis," and ordinary casualties and diseases.)

From the above data we must infer that the conditions of causation are very exceptionally present in the young, only occasionally in the middle-aged and old.

Interrogating pathology, and commencing our inquiries at the seat of disease, we find alterations in the coats of the cerebral vessels to be very constant in advanced life, almost uniformly absent in infancy and early manhood, although in rare cases, atheromatous or other arterial degenerations may be developed in young subjects. Apoplexy, however, does not obtain sufficiently often in proportion to the frequency of disease of the vessels, as to justify us in maintaining such lesions to be alone adequate for the rupture. That a contrary notion has existed, is to be explained by the imperfect mode of investigation pursued by the older authors, whose inspections were oftentimes restricted to the cranium.

Extending our researches to the heart, we find cardiac diseases to be frequent in youth and middle life—often absent in old age.

In the former, notwithstanding there is little or no liability to apoplexy, though young persons with heart affections often succumb before the decline of life to their disease or its complications. Thus alterations in the walls and orifices of the heart seem to be insufficient to induce intracranial hemorrhage. When we

reflect on the multitudes with cardiac ailments who infest our hospitals and dispensaries — many with hypertrophy of long standing and of enormous extent, —and on the infrequency of apoplectic seizures in the progress of their complaints, we are unable to resist the conviction that hypertrophy or other disease of the heart, *per se*, rarely terminates in such direction. We should not fail, at the same time, to recollect that before forty or fifty years of age degeneration of the coats of the cerebral vessels is far from common.

Dr. G. B. Wood remarks that comparatively few cases of disease of the heart end in apoplexy.

Cardiac diseases are very common in the Pennsylvania Hospital, and patients frequently die of their consequences; but during the period of his attendance upon that institution he cannot recall a case in which apoplexy resulted. It is almost superfluous to repeat that patients suffering from disease of the heart, who attend public charities, are almost always under the afternoon of life.

Considerations of a like partial nature induced Dr. Kellie, of Edinburgh, to deny the affinity between hypertrophy of the heart and apoplexy. It must likewise be admitted that heart affections in the earlier periods of life and mid-age are chiefly accompanied with obstructive or valvular disease, so that the momentum is only apparently, not actually, augmented; but even in absence of impediment at the orifices, the systemic capillaries are healthy, so that the local forces equalize the propulsion of the blood, and the minute vessels are not liable to rupture, as their tunics are unaffected by degeneration. In other words, of persons under forty, how few are cut off by apoplectic

effusion! Of the aged, almost universally atheromatous or calcified as to their vessels, how few comparatively perish from the same disease!

The proposition, I believe, to be true that apoplectics are molested by morbid alterations in the heart *plus* atheroma or other degeneration of the arteries; but it is not conversely true that patients simply labouring under cardiac symptoms are frequently the victims of cerebral hemorrhage.

It is a combination of the two pathological states that constitutes the proclivity which, under such circumstances, is indubitably great, more especially with hypertrophy of the left ventricle. In apoplectics the changes in the heart and capillaries react upon each other. All are aware that in the accomplishment of the circulation, although the chief power emanates from the heart or *vis a tergo*, much energy is developed by the *vis inhærens* of the capillaries. In health, the capillary forces of the brain are, in a great degree, independent of the action of the heart, and tend to accommodate the current to, and rectify any disturbance or irregularity propagated from, the circulatory centre; but when impaired by disease of the vascular coats, they are subject to local perversion and arrest, and proportionately interfere with, and are interfered with by, any abnormity in the propelling power of the heart and in the ratio of capacity of the cardiac chambers.

Thus it has been well observed,* "The connexion of cerebral with heart disease has been much noticed,

* Med. Chir. Review for January, 1862 (No. LVII.)—"On the Cerebral Circulation."

and yet in how many instances of the latter are brain symptoms deficient. Some explanation, therefore, is wanting to show why, if there be any such direct elation between lesions in these two organs, it should not display itself with more constancy. This explanation, we believe, is principally to be found in the state of the blood-vessels, the walls of which, if abnormal in any way, produce irregular circulation, disordered function, and diseased nutrition in the brain." Yet one of our most prominent and respected teachers denies any relation of effect and cause between apoplexy and hypertrophy of the left ventricle, and affirms such views to be entirely erroneous.* Dr. Watson, like many others, is inclined to believe (I think wrongly) that the hypertrophy is usually combined with some impediment at the orifices. He thinks that the right chambers of the heart are also often involved, and that the connexion rather subsists between apoplexy and such cardiac alterations as obstruct the flow from the head through the *veins*. If this be absent, he refers the attack exclusively to disease of the cerebral arteries. *He acknowledges the frequent co-existence of cerebral hemorrhage and hypertrophy*, but ascribes the latter to diseased and dilated conditions of the aorta, and thinks the associated changes are the concomitant effects of the same cause.

Dr. Watson says disease of the right chambers of the heart is rare without disease of the left. The opposite is certainly not true; and in another passage this physician remarks, "it seldom happens that the whole heart is diseased."

* Lectures on Physic, 4th edit., vol. I. pp. 423-4.

Although hypertrophy may be accompanied by, or arise from, impediment at the mouth of the aorta in early or mid life, in those advanced in years it is mostly pure and simple, *i. e.* free from valvular obstruction. That the heart is involved, if at all, in the causation of apoplexy through the veins or from hinderance to the return of venous blood, is asserted generally, not proved. Patients with enormous emphysema and dilatation of the right heart, though subject to vertigo and symptoms of disordered cerebral circulation, probably from imperfect aëration and depravation of the blood, are infrequently the subjects of apoplectic effusion, as I can maintain from enormous experience of this class of diseases in the wards of the St. Marylebone Infirmary. Those authors who have turned their special attention to diseases of the heart, as Hope, Bouilland, and others, are also at variance with the sentiments of the above distinguished physician.

Dr. Watson recognizes the hypertrophy as a compensatory process, a measure of the difficulty with which the blood is circulated through the arterial system, and he likewise allows the frequent co-existence of hypertrophy and cerebral hemorrhage. If the hemorrhage be merely dependent upon a diseased condition of the vessels of the brain, we ought in practice to meet with many more cases of sanguineous apoplexy in absence than in presence of cardiac hypertrophy regarding the (almost) universality of arterial degeneration, and the *relative* rarity of disease of the heart in aged persons.

I am unwilling to accuse the kidneys of invariable participation in the pathogenesis of cerebral hemor-

rhage, but am strongly inclined to suspect their complicity in many cases. Morgagni, Dr. Bright, Dr. Williams, Dr. Kirkes, and others, have noticed their frequent implication, though numerous writers are altogether silent on the point. Dr. Abercrombie but once, casually, alludes to them, and Dr. Watson does not at all advert to these organs. If diseased, they may prove direct ætiological agents, or modify and aggravate the symptoms of hemorrhagic extravasation by uræmic complication. Albumen may be often discovered on the employment of catheterism, and I have found it in abundance when the kidneys were apparently healthy to the eye and weighed $9\frac{1}{4}$ oz. in a man of seventy. Of course this albuminuria may be transient, and merely betoken functional derangement; or of old standing, and denote degeneration of the renal tissue. If the latter, the kidneys may be immediately concerned in causation, or produce deranged blood formation, or originate a combination of evils.

Consulting Quain's Anatomy, I learn the weight of the kidney is computed to be about $4\frac{1}{2}$ oz. in the male, rather less in the female.

Dr. Clendinning says the two kidneys, in the male, average $9\frac{1}{2}$ oz., and in the female 9 oz. Dr. Reid makes it $5\frac{1}{2}$ oz. in males, not quite 5 oz. in females.

The following is the record of the kidneys in eight of my apoplectics, whose ages averaged $65\frac{1}{2}$ years.

UNITED WEIGHT.	CONDITION.
6 oz.	small, firm, pale.
7 oz.	firm and pale.
$9\frac{1}{4}$ oz.	healthy.
6 oz.	pale, cystic, granular.

UNITED WEIGHT.			CONDITION.
8 oz.	...	...	very firm, rather pale.
Not weighed		...	congested.
5 oz.	...	...	granular.
Not weighed		...	congested.

Rejecting those not weighed, the average is nearly 7 oz., —much below the standard of health, even when making allowance for the period of life, as, unlike the heart, the kidneys appear, according to my observations, to become rather smaller as life advances, independent of real disease, and undergo atrophy or degeneration of a character identical with that occurring in the vessels, cartilages, bones, muscles, fibrous tissue, and elementary textures—a link in the chain of senile phenomena, which, by irregularities, dissipation, "fast living," or other deteriorating causes, may be much earlier induced, or from deficient vital power prematurely lead to those phases of altered nutrition which are more or less natural and constant in age. Under similar conditions, original robustness may enable one individual to resist more firmly than another injurious influences and the ravages of disease; or like constitutions may, —through intemperance or disease on the one hand, hygienic rules and habits on the other,—arrive at differing periods at similar physical states, the latter slowly acquiring those changes, procrastinated to a green old age, which in the former are anticipated by reckless and extravagant destruction of tissue in excess of repair, and rapid enfeeblement and exhaustion of the powers of life.

I append extracts from autopsies,—of which I possess the requisite notes,—performed by myself on persons over fifty years of age, who did not die of dis-

ease related to apoplexy, uræmia, or other cerebral affection, to show the state of the kidneys compared with those of apoplectics of almost the same average age (64 and 65½ years).

Name.	Sex.	Age.	Disease.	Kidneys.
T. M.	M.	65	Dilated flabby heart. Died suddenly.	Rather small and granular. Not weighed.
J. R.	M.	73	Gradual death from age.	Small, healthy. Not weighed.
J. M.	M.	60	Emphysema bronchitis. Slight pleurisy.	Healthy. Not weighed.
E. H.	M.	78	Angina Pectoris.	Congested. 5 oz. and 5½ oz.
J. R.	M.	54	Exhaustion from destitution.	Large, pale. 5 oz. and 5½ oz.
—	F.	55	Drowning.	Healthy, but congested.
W. J.	M.	52	Pleuritic effusion. An Idiot.	Moderate size, congested.
S. W.	F.	75	Hæmoptysis. Pulmonary apoplexy.	Pale. 4½ oz. and 4¼ oz.

The result of the evidence adduced is unfavourable to the integrity of the kidneys in the apoplectic as

compared with ordinary subjects of the same average age, yet it does not supply proof of direct connexion between the renal changes and cerebral hemorrhage, as in the patient above mentioned, aged 70, the kidneys appeared healthy to the eye and their weight was $9\frac{1}{4}$ oz. In two other cases, in which they were not weighed, they are merely stated to have been congested.

It is interesting to meditate on the point of departure of the morbid changes presented in individuals dying of sanguineous apoplexy. It cannot be rebutted that the waning activities and inclination to inferiorly organized structures, in aged and impaired constitutions, are sufficient to explain the vascular changes in the arteries of the brain. It is a legitimate analogical deduction that simultaneous actions and alterations ordinarily ensue, in the aged, throughout the system, by which a due, but reduced, equilibrium is observed. Non-occurrence or inharmonious adjustment of these kindred changes we can readily surmise, especially in atheromatous subjects, may lead to cardiac disturbance and resulting hypertrophy.

At any rate, it must be acknowledged that the cerebral vessels degenerate, more or less late in life (a constant? factor), but do not dispose to apoplexy unless there be (an inconstant factor) co-existent deviation in the central circulating organ.

Doubtless, obstructive cardiac disease, in certain cases, may pursue its ordinary course, and, if the patient survive, terminate in apoplexy when the period or circumstances accrue which engender the requisite co-efficient in the vessels of the encephalon. In many cases, from constitutional depravation, the blood pri-

marily becomes deteriorated more or less gradually, causing paratrophy of the arterial coats and of the general system. The heart, *pari passu*, undergoes hypertrophy (or rather enlargement and thickening, for its muscular substance equally suffers) from the impediment offered to the transit of impoverished or otherwise altered blood through the coincidentally impaired capillaries. Or, from mal-nutrition, the ventricular wall may be unable to resist the retrograde effect of the opposition exerted in the minute vessels, on account of the diminution in the circulating forces, and experience passive dilatation.

It were unphilosophical to attribute cardiac changes to a simply increased vascular action, in exclusion of local or peripheric obstacles; and the fact that they are mostly remarked in persons not actively employed, countenances a systemic cause of production. In the so-called simple hypertrophy of the left ventricle, where there is no disease at the cardiac orifices, it is clear that gradual retardation of the blood in the capillaries, from impairment of nutrition, or constant presence of toxic materials in the circulation, as alcohol or urea, may give rise to enlargement or dilatation of the organ. Such obstruction, long continued, entails and finds its acme in atheromatous degeneration. In other instances there is probability that the kidney alone is the original starting-point, from the effects of an impression interfering with its depurating function, by which the blood secondarily becomes disordered, leading to progressive alterations in the walls of the heart and synchronous degeneration in the arteries.

Many imagine that the hypertrophy gives rise to the granular condition of the kidneys, but the hypertrophy

is more likely a consequence than a cause—a consequence of capillary stasis and of the compensatory effort to surmount the hinderance to the onward current of blood.

It is highly improbable that non-valvular cardiac disease—the common concurrence in apoplexy—arises from congestion or stagnation in the systemic or renal capillaries, in absence of distemper of the blood, unless we allow (simply) a faulty innervation, which is an entirely unsupported conjecture. Can we concede the tissues to be degraded to the extent implied in extensive atheromatous deposit, whilst the *pabulum*, the blood, be healthy ?

In certain cases of poverty of blood and general debility, it has been pointed out by Dr. Owen Rees, that the extractive, those ill-understood matters of the blood, may be found in the urinary secretion, without the presence of albumen. For instance, in chlorosis and diseases of the heart. They, of course, indicate a drain upon the circulation, and I mention them here, as it would be of cardinal value to subject the blood of apoplectics to careful analytical examination. According to Magendie and MM. Audral and Gavarret, there are diminution of fibrin and increase of the globules in apoplexy.

Difference of opinion has existed as to the proximate cause of the rupture of the vessel or vessels in apoplexy. Abercrombie supposed it to take place from disease of the arteries, without previous derangement of the circulation. Dr. Cheyne believed that the extravasation was a *consequence* of an excited state of the minute vessels of the brain; and Dr. Copland thinks a depressed

state of the vital energy and of the cerebral circulation occurs at the commencement of the seizure, owing to some condition of the ganglial nerves which supply the vessels of the brain and the brain itself (an apoplectic orgasm or *molimen apoplecticum*). This speculation is derived from certain German pathologists, who extend it to all the organs of the body, in which hemorrhagic extravasation may occur, holding that apoplexy depends on sudden paralysis of the sympathetic nerves of the part and consequent increased action of the blood-vessels, terminating in effusion of blood. Dr. Watson regards sanguineous apoplexy, in most cases, to result from the rupture of a vessel of appreciable magnitude.

My own views are that the *assumed* depression of the vital energy, if announced by symptoms, is owing to softening or invasion of the hemorrhagic attack. If not attended by symptoms, it seems to me utterly imaginary and unnecessary, in presence of morbidly friable vessels and the co-operative cardiac lesion.

CHAPTER II.

DIAGNOSIS OF CEREBRAL HEMORRHAGE.

It is certain that premonitory symptoms are *sometimes* absent, although the following are systematically enumerated as respectively preceding the attack in various cases. To me it appears contendible whether they generally occur, and if so, should be regarded as true prodromata, rather than indications of pre-existing disease of brain, heart, or kidney, or of the actual occurrence of effusion.

Rochoux, much of the same opinion, maintained that it is rare to observe precursory symptoms in cases of cerebral hemorrhage. He writes, "Sur soixante-neuf apoplectiques dont j'ai recueilli les histoires, onze ont présenté des symptômes précurseurs, et parmi eux, cinq étaient habituellement sujets à des vertiges qui ne se sont pas sensiblement augmentés aux approches de l'attaque. Ainsi, pour être tout-à-fait exact, il faut dire, que six malades seulement ont éprouvé des symptômes précurseurs, et qui reduit les individus, dans ce cas, à moins d'un dixième."—(*Dict. de Méd.*, tom. IV. p. 104.

The symptoms referred to are weight and pain in the head, vertigo, tinnitus, deafness, throbbing of the temporals or carotids, epistaxis, lacrymation, diplopia, heteropiæ, muscæ, ptosis, trembling speech, sighing,

yawning, vomiting, change of countenance, deviation of the mouth, starting of the limbs, excitement, agitation, numbness of the extremities, vacillation, stumbling, chilliness, flushing, pallor, grinding of the teeth, especially during sleep, wakefulness, drowsiness, incubus, incoherence, and loss of memory.

Were this group of symptoms ever aggregated in one person, it must be allowed that though they point to cerebral disorder, they are not pathognomonic of imminent apoplexy. It may be they are honoured as traditionary, as the chief of them were distinctly described long long ago by Cœlius Aurelianus,—who added that they are often absent,—and, with additions, centuries after by Ettmuller; but the former sagaciously explained that they equally presage the advent of mania and epilepsy (sed hæc communiter antecedunt vel præeunt etiam his qui in *epilepsiam* vel *furorem* venturi videntur).—Lib. III. cap. V.

The character and intensity of the symptoms of hemorrhage, of course, vary, according to its suddenness, situation, and amount; death being most certain and rapid, as a rule, when the extravasation occurs at the base of the brain; next, into the ventricular or arachnoid cavities; being less so when limited to the neighbourhood of the cerebral ganglia or the substance of the hemisphere. The issue is likewise much determined by the coincident morbid condition of the individual, as very feeble or previously hemiplegic persons will sometimes succumb to a very trifling sanguineous effusion—a fact apparently unnoticed in articles on the disease.

A patient may suddenly stagger and fall, and die

almost instantaneously in very rare cases,* but more commonly he becomes soporose and gradually merges into coma. The face is usually flushed, the pulse at first slow, full, hard, thrilling, and as it were resentful; but it may be small, weak, and quick in feeble cachectic patients and exceptional instances. The face also may be pale, livid, or bloated, and there is often marked hebetude of expression. The breathing is generally infrequent, and more or less stertorous; but it may be calm and natural, or suspirious at the outset. Frequently there is hemiplegia, the opposite side remaining unaffected, or it may be rigid and convulsed, or there may be rigidity of the limbs with or without convulsion, or the upper extremities may be flaccid, the lower rigid or convulsed, or there may be, mostly at a later period, general loss of muscular power. The pupils may be natural, contracted, or dilated, or one contracted the other dilated, and sensible or insensible to light. The mouth frequently deviates, but occasionally this is not observed. This state may terminate in perfect recovery (?), recovery with paralysis, or death.

Or the patient may suddenly become hemiplegic and speechless, with or without loss of consciousness, or unconsciousness may be transient, or again gradually pass into coma, which may be recovered from, or prove more or less speedily fatal. Or perfect recovery may ensue (?), or the patient may suffer from permanent hemiplegia and loss of speech, or imperfectly recover,

* Dr. Abercrombie relates one (CXVI.) where death occurred in five minutes, and one of my own (see page 35) was almost instantly fatal.

or the paralysis may be succeeded by early or late rigidity and contraction of the muscles.

In very sudden and extensive effusion (apoplexia fulminans, apoplexie foudroyante) the patient may become pale and faint, the surface cold, the pulse small and feeble. Frequently there is vomiting, yawning, and some impairment of speech and consciousness, which more or less quickly lapse into absolute coma, preceded, accompanied, or followed by hemiplegia, convulsions or general paralysis, spasmodic twitchings of the face and limbs, whiffing expiration, foam at the mouth, clammy perspirations, widely-dilated pupils, involuntary evacuations, and speedy dissolution.

It must be remembered that types of apoplexy more abound in books than in practice.

In our present state of knowledge it is impossible to localize cerebral extravasation, or always to affirm positively even that hemorrhage has occurred. In fact, it may be absolutely asserted that sanguineous apoplexy does not furnish one diagnostic, or rather pathognomonic symptom; yet collectively a certain series of objective phenomena, in most instances, will render an opinion tolerably certain. Cases are, however, occasionally presented, which are but slightly pronounced, especially on invasion, and prove very embarrassing. It must in addition be remarked that *all comatose diseases in their last stages simulate true apoplexy, and cannot be discriminated in default of their previous history, which is unfortunately often deficient or absent in patients found insensible and brought to a hospital.* The age may be of some assistance, as cerebral hemorrhage is not common until after the meridian of life. The condition of the heart, if known

beforehand, may also aid us. After the access of the disease this is difficult to examine, and the value of physical signs is doubtful, as coma, from whatever cause, is usually accompanied by oppressed quasi-hypertrophous action of the organ, in consequence of altered innervation.

Cæteris paribus, in a case of coma, the ascertainment of previous temperate habits, non-existence of *arcus*, and the exclusion of cardiac ailment augur favourably. Flushing or pallor of the countenance is an unsafe guide, without due correction, as the face is for the most part pale in the worst or ingravescent attacks, and it may be pallid, with lividity, throughout, if the hemorrhage be complicated with uræmia.

The most certain indications of intracranial hemorrhage are *sudden* hemiplegia, with more or less immediate and profound loss of consciousness—with or without rigidity or convulsion—stertorous breathing, deviation of the mouth, flushed face, and a full slow pulse. Tonic or clonic contraction of the muscles of the limbs frequently testifies to the co-existence of ventricular or arachnoid sanguineous effusion, and general paralysis of the extremities is usually associated with diffuse hemorrhage or the moribund state. Not any constant or special symptoms (as Gall and Serres maintained) attend apoplectic extravasation into the cerebellum, which may serve to distinguish it from effusion into other parts of the brain. (Brown-Sequard.) In a very circumscribed apoplexy, the slightness or almost negation of symptoms may render a decision difficult or impossible. If an attack, apparently apoplectic, *entirely* subside, *i. e.* without sequelæ,

hemorrhage, if pre-existent, must have been exceedingly limited, and in the immense majority of cases the symptoms will depend, not on hemorrhage, but on other disorder.

The most common affections with which apoplexy may be confounded in practice, are uræmia and poisoning by opium. In *uræmia*, the premonitory symptoms, if ascertained, are headache, vertigo, *muscæ volitantes*, anomalous pains and epileptiform attacks in many cases. The aspect is usually sallow and cachectic, the ankles often œdematous, and the urine more or less charged with albumen. In the uræmic paroxysm the coma is more gradual, and generally less profound (*sopor*) than in apoplexy. There is absence of paralysis at the outset, or it is less marked. Subsultus or convulsion is common, there is less stertor, and the breath fumes with hydrochloric acid—a very unsatisfactory test. Œdema, or anasarca, should not be overlooked in the diagnosis.

In *opium* and ordinary narcotic poisoning, the age and the odour of the breath may assist. The countenance is usually pale and placid, but ghastly, the skin moist. The patient appears as if in sound sleep, from which he is rousable in the earlier stages. Paralysis is absent. The pupils are not *always* contracted, even from opium. At a later period, the stupor passes into stertor and absolute coma, and without knowledge or history of the case it may be undistinguishable from uræmia or apoplexy. I once saw a woman (under the care of four medical men) with contracted pupils and the symptoms of narcotism, who was treated with the stomach-pump, coffee, walking about and galvanism,

and, after death, the appearances were those of extensive hemorrhagic laceration of the brain. In *alcoholic poisoning*, coma may be at once induced if the spirit be concentrated and rapidly swallowed. The odour of the breath, absence of paralysis, and circumstances of the patient will enlighten us on most occasions. *Concussion* or *compression* is generally recognizable as the result of violence. A person may, however, be intoxicated and experience concussion from a fall, and be found by strangers in a state of insensibility. A case, thus diagnosed, occurred to me a few years since in a woman of 50. The surface of the body was rather cool, the breathing calm, pupils sluggish, moderately dilated, and the pulse small. There were complete insensibility without paralysis, and smell of spirits (?) in the breath. Galvanism produced movements, but no manifestations of sensation. The stomach-pump was employed, but nothing noticeable removed. Remedies were discontinued, as the case was mysterious and bewildering, and the patient was warmly covered up in bed. She arose the next morning perfectly well. After recovery the account she gave was untrustworthy.

It should ever be kept in view that apoplexy may be complicated with uræmia, uræmia with opium-poisoning or alcoholism, and intoxication with narcotism or concussion; instances of most of which I have observed in public practice. For the determination of the cause of the coma, under such combinations, few definite rules can be appointed. The symptoms must be judged by the light of experience, without which, in cerebral diseases especially, little advantage redounds from the most copious and methodical illustration.

Cerebritis, or partial inflammation of the brain, occasionally runs a chronic course, and may be almost latent until coma supervene, headache and febrile disturbance being the only complaint until suddenly aggravated into unconsciousness. It is mostly traumatic. Of course it is usually distinguished by the persistent pain in the head, hebetude, vomiting, tonic contraction of the flexor muscles, and slowly progressive symptoms.

A man, admitted into the St. Marylebone Infirmary, on account of a surgical affection of the leg, abruptly became as it were apoplectic and rapidly died. On examination, extensive red softening was discovered in one of the hemispheres, and it was subsequently learnt that he had been much injured about the skull three months previously. Abscess, or suppurative inflammation of the brain, presents the same symptoms as cerebritis, and in like manner may run a latent course in rare cases.

Meningitis cannot be mistaken in the early stages. It tends, however, finally to coma. It is mostly traumatic or tubercular. *Inflammation of the dura mater* is almost always the result of injury or of extension from the ear, diploë, or brain, strumous or syphilitic. It is most common in young and middle-aged subjects. It may be very insidious and suddenly announced. In adults, if associated with syphilis, the cachexia and existence of caries will decide the point, and epileptiform attacks are common from gummatous infiltration of the fibrous membrane. Disease of the cranium may, in very rare cases, cause erosion of the meningeal artery and consequent hemorrhage.

Ramollissement, or white softening, is known by the previous existence of symptoms, more or less

marked, which are much discrepant, according to the site and extent of the disease. There may be impairment of intellect, irregular pains in the extremities, hesitation or defectiveness of speech, loss of particular words, inability to remember names altogether or for some interval, misapplication of words, incoherence, disposition to melancholy and tears, passing unconsciousness or actual epileptiform or apoplectiform attacks, paralysis or tonic or clonic contraction of the limbs, etc. Coma dependent upon softening is usually attended by less regular or less complete phenomena than apoplexy, but softening often terminates in sanguineous extravasation.

In *embolism*, or fibrinous occlusion of the cerebral arteries, the *usual* concomitants are headache, vertigo, and paralysis, without complete abolition of consciousness—at any rate, at the commencement.

Intracranial tumours may pursue an almost unobserved course, until the advent of fatal coma, or they may give rise to headache, vomiting, vertigo, amaurosis and other affections of the special senses, insanity, epileptiform convulsions and gradual paralysis.

Variation in their size, shape, structure, and situation will be attended with corresponding variety of symptoms. They may be specific, as syphilitic, cancerous, tubercular ; or osseous, fibrous, hydatid, or aneurismal. With malignant tumours there is mostly an organic look and intense constant or remittent headache, even in the earlier stages. I have seen an enormous fibrous tumour springing from the inner surface of the dura mater, cause deep indentation of the hemisphere, and yet be unaccompanied by symptoms of mischief until within twelve hours of death. In a case

of hydatidiform cyst of the brain in a child the symptoms were similar to those of hydrocephalus.

Aneurisms are a special form of tumour tending to rupture and hemorrhage, when they constitute apoplexy. They may implicate the basilar, cerebral, meningeal, or smaller vessels. Sometimes they produce very rapid death from rupture, preceded or not by complaint of cephalalgia, occasional vertigo, or other disturbance. In a case of aneurism of the anterior cerebral artery, which did not burst, the patient under treatment for another disease gave a sudden piercing shriek and almost immediately died. In a case of aneurism of the posterior cerebral, also unruptured, which was the size of a *boss* or *taw*, the patient suddenly became inarticulate, but remained slightly conscious and could swallow voluntarily. She died after about sixteen hours in a state of coma and exhaustion. The basilar was reduced to a mere thread, and the tumour above projected into the third ventricle, between the thalami. Below it was seen in front of the pons between the crura cerebri. It was solid, and exhibited the concentric laminæ from pale fawn to dark brown in colour, with a small black clot at the anterior part. In every case of tumour the characters of the presence of the growth differ in expression; for as Dr. G. B. Wood observes, the tumour does not yield the signs directly, but the brain upon which it acts.

From epilepsy the diagnosis is almost always clear. In *epileptic coma*, which might prove perplexing, there is commonly, besides the previous symptoms and history, foam at the mouth, peculiar pallor of the countenance, chilliness of the surface, weak pulse,

absence of paralysis and of decided stertor, and rapid recovery.

From *hysteria* and *syncope* the criteria will be manifest on full investigation, and in *malingering* or feigned unconsciousness, the closure of the eyelids, mobility of the pupils, absence of bitten tongue, and the sensibility of the impostor to snuff, tickling, pinching, and to pain and dread, proclaim the counterfeit.

In very exceptional instances *pneumonia* may, in consequence of cerebral disorder, exhibit a masked form, and be mistaken for apoplexy. A case of this nature, with coma and stertor, admitted into the St. Marylebone Infirmary, was seen by a well-known physician, who diagnosed hemorrhage into the brain, and indeed designated the locality. After death the only morbid appearance, on strict examination, was extensive double pneumonia. There was no reason to suspect that this was secondary to typhus, albuminuria, or otherwise intercurrent.

Lastly, to exhaust the diagnosis, it may be added that passive or symptomatic cerebral hemorrhage occasionally ensues in the advanced stages of *purpura* and *scurvy*.

CHAPTER III.

TREATMENT OF CEREBRAL HEMORRHAGE.

This is full of uncertainty. The diagnosis must of course be undoubted, in order to confirm the efficaciousness of remedial measures. If the patient die, little credit is reflected on therapeutical efforts, and if he survive, it remains unsettled whether extravasation did or did not occur. Even granting the extravasation, it cannot with precision be decided how far the treatment insured recovery. In arguing thus I am supported by Heberden and Cullen. Hesitation is increased on bearing in mind that all cases of sudden *coma* have often been, and yet are, regarded as apoplexy, and treated without distinction on an active plan. I admit that the judicious practitioner will individualize his cases and be guided by less arbitrary principles; but the force of established practice, good or evil, exerts influential, if unfelt, action on all, and greatly obstructs rational innovation in medicine.

Perhaps more than in any other disease, the treatment in apoplexy has been conducted in accordance with stipulated and undeviating precepts. Abstraction of blood, drastics, enemata, emetics, elevation of and cold to the head, blisters and sinapisms, almost complete the round of remedies resorted to during the

attack; but until within the last century errhines, volatile salts, and vinegar were, in addition, almost invariably recommended to arouse the flagging motion of the animal spirits; and many of the ancient and mediæval physicians, from Aretæus downwards, strongly advocated the application of the actual cautery to the vertex or occiput.

Of these agents the lancet occupies the foremost place, and with some it has been the custom, in all or almost all cases of supposed cerebral hemorrhage, and even in other forms of coma, to adopt venesection as a primary procedure. There can be no doubt that this inconsiderate practice, as in pneumonia and other diseases, has proved on the whole more harmful than salutary; and I feel pleasure in expressing conviction that the medical mind at the present day by no means ratifies the notion that withdrawal of blood in cases of cerebral extravasation is indispensable, or of anything like universal application. It is due to the physicians of a former age to observe that many of them also exercised a prudent reserve and moderation in the employment of venesection. To exemplify, Heberden (Comments., chap. LXIX.), after a sound practical description of apoplexy and palsy, remarks that the habit of taking away blood must be founded either in experience or theory; and if he were to judge from the cases which have occurred to himself, he should say that the occasions where it could be supposed to do good have been extremely few, and that large bleedings appear to him to have several times been prejudicial. His predecessor Ettmüller, though deeming venesection necessary in *sanguineous* apoplexy, declares that bleeding and vomits unseasonably administered

have murdered a great many. Dr. Fothergill was also much averse to bleeding as a general rule.

Dr. Cheyne, on the other hand, was an ardent supporter of blood-letting as the most effectual remedy in all cases where the patient was not actually sinking. More recently Dr. Cooke highly recommended venesection in *strong* apoplexies, but he admitted that it may be carried too far, and that the probable powers of the patient should be considered. Even Celsus (book III.) doubted that detraction of blood was always useful. He writes pithily "vel occidit, vel liberat;" and Aretæus (De Morb. Acut. Curat., lib. I. cap. IV.), though greatly favourable to bleeding in apoplexy, says it is better to err in moderation (κρέσσον δὲ ἐς τὸ ἔλασσον ἁμαρτάνειν).

It were superfluous to traverse the opinion that phlebotomy is demanded on all occasions. Often it would accelerate the fatal issue when the patient has been much reduced by previous paralysis, long confinement to bed, or other causes; and in the last stages, or in the hopelessly collapsed and moribund, it is clearly indiscreet to interfere. But the important questions for consideration are, is true sanguineous effusion ever benefited by bleeding? If recovery follow bleeding, is it in the relation of effect and cause, or is the recovery ascribable to natural endeavours? In other words, did any apoplectic ever recover after bleeding, in whom recovery would not have ensued, as a *spontaneous* consequence, had the patient been entrusted to the resources of nature?

This must ever be equivocal. Inferences of probability may be drawn from an impartial calculation how far venesection diminishes the force and action of the

heart, and how far such diminution can arrest or mitigate effusion from ruptured arteries, influenced by the peculiarities of the endo-cephalic circulation. In a small circumscribed apoplexy, in the substance of the hemisphere, in a moderately vigorous patient, the prognosis is favourable, whether bleeding be or be not employed; whereas in profuse hemorrhage, in extravasation near the base, or in multiple attacks, the prognosis, on the reverse, is unfavourable under any mode of treatment. It is only then in medium cases that abstraction of blood can be considered to exert any important influence, beneficial or injurious, and it is pertinent therefore to review its probable operation.

It is ordinarily held that blood-letting relieves congestion of the vessels and tends to check further apoplectic effusion. Whatever view may be embraced as to the cerebral circulation or the concurrence of congestion in hemorrhage, it is beyond doubt that the symptoms depend on solution of the cerebral fibres or extravasated blood.*

Bleeding to excess cannot at once absorb or directly obviate the effects of the blood already effused, and it cannot completely arrest the vascular current in the

* I am, of course, aware of the view of Monsieur Serres that the effusion follows the apoplexy, not the apoplexy the effusion. This is an adoption of the opinion of Weikard, who maintained that the extravasation of blood was a sequel and symptom of spasmodic contraction of the dura mater, from arthritic, rheumatic, or other subtle material, or fluid, or tumour, causing irritation, not pressure. Dr. Burrows, again, holds that the apoplectic coma depends usually not on the hemorrhage, but on the pressure of vascular congestion: to which I reply, that, in fatal cases, the brain is often pallid, at any rate *after death*, except in the vicinity of the lacerated tissue.

brain without entailing fatal syncope. If of any advantage, it can only so far modify or retard the flow in the vessels as to avert or lessen subsequent extravasation. Venesection depresses the impetus of the heart by reducing the quantity of the blood and impairing its quality (from decrease of the globules and increase of the fibrin); it must thus to some extent influence and lower, in common with other organs, the functions and nutrition of the brain, already in abeyance. This is practically confirmed in some cases of cerebral hemorrhage, where blood-letting is succeeded by augmentation of cardiac excitement rather than sedative action. The reason is readily interpretable. Various impoverished states of the blood are connected with irritable conditions of the heart, and in apoplexy the presence of degenerations discloses that the circulating fluid is often qualitatively defective in a grave degree.

In most instances we are previously unacquainted with the condition of the heart, and although Dr. Burrows enjoins it to be examined in apoplectic attacks, it is a very fallacious guide, as its action is always laboured, and in cases of coma, from softening or other causes, it may present an inorganic murmur *at apex*, from altered innervation, being at the time free from disproportion or valvular disease. He (Dr. Burrows) narrates a case (pp. 150-4) which he terms apoplexy, and in which he conceived hypertrophy of the left ventricle to exist; but from the record of the autopsy it appears to have been softening, and the heart was natural in size, with slight hypertrophy of the left ventricle (*sic*).

Dr. Burrows directs that the advisability of venesection should be determined by the state of the heart; but as I have already hinted, it is of doubtful significance, and it is better to trust to the strength of the pulse, unless calcified, the symptoms, powers, and constitution of the patient.

In the robust, if the symptoms be comatose more than syncopal, the lethargy profound, and paralysis be present, bleeding is I think justifiable. It will probably diminish the amount and stimulating properties of the blood, moderate the action of the heart, and may check further effusion. Again, the diagnosis is not absolutely certain, and the coma may depend on noxious matters in the blood, a portion of which will thus be removed from the circulation. Further, if the apoplexy be of sthenic character, and attended by marked stertor or serious apnœal phenomena, depletion will relieve the engorgement of the lungs and cavities of the right heart. By assuaging the circulo-respiratory difficulty, the system will be better enabled to encounter the detriment inflicted upon the brain; and after cessation of effusion and rally from shock, the unconsciousness may be protracted merely by asphyxial disturbance. By removal of the secondary effects, venesection may thus indirectly restore the patient.

In certain apoplectiform attacks, in which insensibility is speedily recovered from—during or soon after abstraction of blood—I believe, the symptoms subside in consequence of depletion allaying the cordo-pulmonary distress, which has become the chief, though secondary lesion, or that they arose from ramollisse-

ment, cerebral embolism, or other species of coma, the duration of which is usually transient, independently of treatment.

As to the utility of blood-letting in reference to the ruptured cerebral vessels, much disagreement exists amongst practical men, in the main representing their favourite theories. Drs. Clutterbuck and Burrows may be selected as partisans of extreme physiological schools. The former believed that blood-letting does not diminish the quantity of blood in the brain, but reduces the velocity and impetus of the cerebral circulation by influencing the general system. He thought that the circulation in the brain is in great measure independent of the heart. He recommended moderate blood-letting to reduce arterial action, but not without discrimination of cases. Dr. Burrows, on the contrary, alleges that even in hemorrhage the coma depends in most instances on vascular congestion, and that the quantity of blood within the cranium is extremely variable at different times and under different circumstances. He is, in consequence, more sanguine as to the propriety and beneficial operation of venesection. It is curious that Dr. Abercrombie, though his views accorded rather with those of Dr. Clutterbuck than of Dr. Burrows as to the cerebral circulation, adopted, almost without reservation, large and repeated blood-lettings in apoplexy and coma generally. He enjoins practitioners not to be deterred by a weak, languid, compressible pulse, or the general infirmity of the patient. Portal, on the same side, thought venesection not only the best remedy, but the best prophylactic (premier remède préservatif); and Cruveilhier regards early venesection as the most effectual

treatment—first in the saphæna, then in the arm, and lastly the jugulars.

From these and intermediate opinions it may be fairly educed that the value of bleeding in the treatment of apoplexy has not yet been accurately computed, or its mode and extent of effects unquestionably demonstrated. It may seem needless and presumptuous to attempt to raze phlebotomy from its ancient pedestal, as from time immemorial it has been venerated as almost the *remedium unicum* in apoplectic disorders; but I humbly urge in apology that my depreciation and scepticism are not dictated by a spirit of revolutionary speculation, but from unfortunate familiarity with the cheerless prospect offered by the recognized treatment of cerebral hemorrhage. In addition, the performance of venesection is impracticable or contra-indicated, where the requirement of succour is most peremptorily urgent; but I acknowledge that this in part obtains from the inherently irreparable nature of the mischief. Hence Hippocrates and Aretæus in the same words (the latter using the aorist form) observe, "λύειν ἀποπληξίην ἰσχυρὴν μὲν ἀδύνατον, ἀσθενῆ δὲ οὐ ῥηΐδιον," and Sauvages, echoing their sentiments, declares "apoplexiam fortem tollere ut plurimum impossibile, carum vero non facile est."

Local blood-letting by cupping, arteriotomy, or leeches, is sometimes combined with general depletion, or substituted in its stead, on account of the age or debility of the patient, or the intensity of the prostration.

Monsieur Littré strongly recommended bleeding from the septum narium (la pituitaire de la cloison) with a bistouri caché or by means of leeches, which

Cruveilhier previously proposed and practised. It may be reminded that the countenance may be highly flushed and congested, without, of necessity, any participation of the brain, and as there is but little vascular connection between the surface and the encephalon, the benefit of topical withdrawal of blood—if it be useful—must be almost exclusively conferred by impression on the nerves distributed to the cutaneous surface, producing a reflex contraction of the coats of the cerebral arteries. To be brief, on the whole, if bleeding be requisite, venesection appears in many respects preferable, from the considerations already adverted to; and as delay is hazardous, the promptitude with which the lancet can be wielded is an additional argument in its favour.

In the Gulstonian Lectures for 1864, "On the Uses of Blood-letting in Disease," Dr. Markham philosophically discusses its action, and nearly assigns its position as a therapeutical agent, but principally in connection with inflammation.

In reference to abstraction of blood in apoplexy, I feel compelled to append the following commentary of Dr. Machlachlan:—"Bleeding seems to be still more rapidly falling into disuse in the treatment of diseases in advanced life, and is justly very much less resorted to than heretofore in the apoplectic and paralytic affections to which old age is liable. This is a natural consequence, because, in the first instance, phlebotomy has almost always been guardedly advised in the old by practical men; and in the second, because within the last twenty or thirty years the nature and causes of these cerebral disorders have become better known, and the inutility, if not the impropriety of blood-letting,

has been fully demonstrated in many varieties of apoplexy and palsy—not only practically, but by the pathological discoveries made on *post mortem* examination, showing how irrational and absurd the expectation of benefit from such a proceeding" (p. 42).

With regard to the remainder of the treatment, emetics have been occasionally administered; but they may be at once dismissed, as, although I have never witnessed their exhibition, I am convinced they are always to be condemned in true apoplexy. Yet physicians of the seventeenth and commencement of the eighteenth century, as Ettmüller, Sydenham, and Allen, recommended vomitories after bleeding (*ex. gr.* vomitorium propinetur statim, Syd.), though they appear to have been denounced by Sylvius, Boerhaave, Morgagni, and Barbette, and later by Cullen, Cheyne, and Cruveilhier. I presume that they gained reputation from having been employed, with apparent success, in overloaded states of the stomach (interfering with cardiac action or otherwise inducing a syncopal condition), or in narcotic poisoning, uræmia, or other pseudo-apoplectiform attacks.

Cathartics are most important in removing vitiated secretions and sources of nervous irritation, and by abstraction of serum from the vessels they place the system under the best circumstances to resist and sustain the injury experienced by the brain; at the same time favouring, by inspissation, coagulation at the orifices of the ruptured arteries. They may also act beneficially in some unknown sympathetic manner. In the treatment of apoplexy, I believe them to be the

least objectionable, indeed the safest and most valuable articles in the materia medica, and as the diagnosis is, at times, obscure or incorrect, it is a satisfactory reflection that their employment is almost always exempt from danger, and alike profitable in other comatose affections. Altogether, in proportion to their usefulness, their operation is less depressing than any other class of remedies in the whole range of therapeutics. The most cogent drawback is that their action is not immediate. One or two drops of croton oil, alone or in combination with calomel, should be placed on the tongue, and repeated, if necessary, in one or two hours. In the mean time an enema of chloride of sodium and castor oil may be injected, or one of turpentine,—which is stimulant and styptic,—if there be collapse apparently dependent on diffused hemorrhage, or on the feebleness and low physical resistance of the patient. In the latter case, ammoniated draughts may be also given. If the croton oil do not act early, it may be expedited by spoonfuls of a mixture of jalap or senna, and tartrate of soda, cautiously administered. At the same time the head is to be elevated by pillows, and if flushed or hot, cold lotions or ice may be applied to the scalp and forehead, but it is difficult, *with certainty*, to explain or estimate their reputed benefit. Cruveilhier, apparently anticipating vaso-motor therapeutics, thought the constriction of the vessels of the exterior of the cranium exercised a similar effect on the intracranial vessels, and therefore highly recommended ice to the head, not constantly, but in an intermittent manner. In one case, after failure of all other remedies, he affirms that it produced a "resurrection."

Sinapisms to the legs and hot bottles to the lower extremities may be likewise useful as revulsives. Blisters about the head are of doubtful service at the earliest period, but after an interval may counteract the tendency to succeeding inflammatory irritation. They should be attached to the nape of the neck rather than to the integument of the head.

After recovery, the patient must be carefully watched, the diet regulated, and general hygienic measures observed, to guard against a recurrence; and the cerebritis and hemiplegia are to be treated with due solicitude and circumspection.

In conclusion, it may be broadly enunciated that the great points in the therapeutics of cerebral hemorrhage are to diagnosticate between oppression and depression, and to avoid the *nimia diligentia medici*, without drifting into expectancy.

CHAPTER IV.

PROPHYLAXIS.

Notwithstanding the controversy which subsists with regard to the character, pathological significance, and even the presence of cardiac lesions in cases of apoplectic extravasation—which I have striven to reconcile as dependent on the imperfect methods in which thanatological inquiries have been conducted and on the aspects whence the subject has been approached—unanimity almost prevails amongst the moderns as to the invariable (?) existence of atheromatous, or other degenerations in the vessels of the brain. Such degenerations, in consequence, are generally acknowledged to be an important ætiological element in the seizure. Preventive medicine, dating with clearness from the immortal discovery of Jenner, has gradually unfolded that the virulence of diseases of the zymotic, endemic, and developmental classes may be much restricted or abated by sanitary efforts, and it is now well ascertained that disorders of degenerative type, if not inveterate or greatly advanced, are also susceptible of amelioration or curtailment by hygienic and remedial agencies. It is no small triumph of our art that we are enabled to discern and utilize the objective signs afforded by examination of the patient, and so control

or remove the incursions of degeneration, as to ward off or procrastinate the liability to cerebral hemorrhage —a disease, the onset of which is so abrupt and fearful, and the result for the most part so disastrous.

It is appropriate to consider the nature of the degenerations, which are induced or encouraged by deviations from the healthy state, and how far it is competent in the medical practitioner to combat and restrain their progress.

In extreme age, the tendency to lower organized tissues is probably normal and universal; but this age must be held to fluctuate according to the habits and original constitutional vigour. Mr. Paget observes, that "the changes of natural degeneration in advanced life have a direct importance on all pathology; because they may guide us to the interpretation of many similar anomalies, which, while they occur in earlier life, we are apt to call diseases, but which are only premature degenerations, and are therefore to be considered as methods of atrophy, as defects rather than as perversions of the nutritive process, or as diseases only in consideration of the time of their occurrence" (p. 71).

That corporeal strength, features, and attributes differ, is manifested in the various temperaments, the predominance of certain functions and physical peculiarities, and the proneness to special forms and types of disease in different individuals, each person being, in a certain and extended sense, idiosyncratic.

The proclivity to certain morbid processes is doubtless to some extent hereditary or transmissible from the parent to the offspring, but it is as satisfactorily determined that this tendency is subservient to modification, to aggravation, or to correction, according to

the physiological and moral conditions to which the progeny is subjected.

Nevertheless it is to be enforced, that irrespective of the disturbing influences of food, soil, climate, exposure, mental and bodily occupation, and other extrinsic agents (weighty as such influences indubitably are), the duration of life is a variable quantity on constitutional grounds, and that each, on this account, would attain an age more or less protracted, even if allotted to the sphere of conditions most conducive to longevity. It cannot be doubted, says Dr. Roget (Cycl. of Med., art. "Age"), that some are destined to a shorter and others to a longer term of existence, independently of all adventitious causes that may occur to disturb the regular course of nature in the demolition of the fabric and the termination of life.

The degenerations of the vessels—with which tissues I am alone concerned—may be atheromatous or calcareous, and in a general manner, I may observe that atheroma or fatty transformation is rather indicative of premature senility; calcareous or earthy changes, of natural decline of life. I must not be understood to affirm, however, that calcification is not encountered at very early periods, or that atheroma may not be attendant on age; or that, again, atheroma may not be converted into earthy deposition, but simply that the fatty form of degenerescence rules rather in those old by anticipation, the earthy in those advancing to hale senescence.

The sources of premature degeneration are numerous, and some weight must in the first place be attached to the innate constitutional powers. Thus

the descendant of a long-lived family, *i. e.* in which both parents were derived from ancestors living to advanced age and were themselves longæval, has, unless overtaken by accident or acute disease, great probability of surviving to the full term of existence (fortes fortibus creantur). Descent from one parent of short-lived progenitors may vitiate this expectation, as children at times exhibit almost exclusively the paternal or maternal constitution.

Amongst the predisposing and exciting causes is, *par excellence*, the abuse of ardent spirits and other alcoholic fluids. It is not to be inferred that I recommend total abstinence or deprecate fermented stimulants *in toto*, as much depends on the occupation, exertion, and general health, as well as on the wear and tear of the nervous system. Moral causes, as mental inquietude, anxiety, grief and ennui, unquestionably exercise considerable influence, and also dissipation, syphilitic affections, and mercurialization, especially in those disposed to dyscrasia. Luxurious living, privation, renal disease, and gout also tend to originate and foster degenerative changes, as well as overcrowding, insufficient air and light, and, perhaps, immoderate smoking.

Dr. C. J. B. Williams believes that organic disease, as hypertrophy of the heart, atheroma, &c., may issue from the continued excitement of sthenic plethora or hyperæmia in those in the prime of life or of sanguine temperament, and Mr. Paget thinks it very probable that the more complex and vascular tissues, such as the muscles, can become hypertrophied by excess of blood. If these views be, and they probably are, cor-

rect (though it may be inquired if, in such cases, the blood is normally constituted), the secondary result of reaction from simple overstimulus, or a plethoric state, is tendency to debility, deterioration of tissue and decay, equally and in common with the effects of intemperance and dissipation, and also of privation and deficient nutrition, in which latter case, even languid performance of function becomes overstimulus to the enfeebled system, directly inducing gradual degeneration.

Amongst the agents enumerated I would, I say, adjudge the most potent causative influence to alcohol and to extravagant expenditure and depression of the nervous energy—of that which regulates the processes of nutrition and secretion.

The emulation, ambition, absorbing greed of gold, and the accompanying cares and brain-tension of the present generation, from rapid competitive improvements in inventions, science, commerce, and other mundane affairs, eminently conduce to overtask the intellectual powers and promote inordinate waste of the nervous system, the nutrition of which becomes more or less impaired. Indirectly, the functions languish from derangement of the nervous force, and mal-assimilation and its consequences result, the progress of which, unarrested, is inevitably downwards towards degeneration of tissue.

With regard to alcohol, the aphorism should not be forgotten that "aliud vinum, aliud ebrietas," and that the moderate use of wine and beer, especially by persons of feeble constitution, by those much occupied in mental and bodily exertion, or those suffering from grief and anxiety, is probably beneficial and preventive

of undue destruction of tissue of nerve and brain. Even in large quantities its injurious action is neutralized or diminished by great physical labour; yet it is absolutely certain that its abuse, especially in the undiluted form, is a most fruitful source of many of the most grievous ills of poor humanity. If imbibed to excess, it may be detected in the blood, bile, urine, brain, liver, and other organs. It gradually produces depravation of the blood, impairment of the nervous centres, and, amongst other disorders, fatty degeneration of the kidneys, and of the arteries of the brain.

By degrees—

> "The life of all the blood
> Is touch'd corruptibly." *

The virtues of temperance were not disregarded by the older physicians; in proof of which I refer to, but cannot venture to quote, a lengthy but very graphic declamation against intoxicating drinks in the "Synopsis Medicinæ Practicæ" (MDCCXXX.) of Dr. Allen, p. 166, as it forcibly depicts their baneful action and the difficulties which encompass us in the treatment of those addicted to vicious beverages.

Yet some members of the profession have hesitated whether arterial degenerations are the ordinary or necessary accompaniment of age, and it has been questioned whether drunkenness is a leading cause in the production of atheromatous changes. Thus Mr. Hodgson (Diseases of Arteries and Veins) seems to think if the deposition of calcareous matter in the arteries of persons advanced in life were the natural

* *King John.*

effect of protracted existence, its extent would be proportionate to the age of the subject; but I think it explicable by the original constitutional power, degree of temperance, and general mode of life of the individual.

Mr. Moore again (Holmes' Surgery, vol. III.) believes that atheroma prevails chiefly in persons who are in the decline of life, and that it is associated with defective nutrition of the normal structures of the body, and with an accumulation of fat; and that there can be no question of its frequent, if not invariable occurrence in drunkards, but that there must exist, however, some more hidden fault in the formation or purification of the blood, to which, rather than to alcohol, the disease must be attributed.

My previous observations will, I think, sufficiently illustrate why some persons are more readily affected and sooner succumb than others to the effects of alcoholism and other pernicious influences; and for further consideration of the subject of intemperance as impairing nutrition, and for explanations why drunkards *occasionally* attain old age, in spite of their malpractices, the reader is referred to Canton on Arcus Senilis, chap. VI.

In a person suspected, or suspecting himself, to be inclined to apoplexy, we should direct our attention to the state of the heart, the condition of the renal secretion, and the presence or absence of the arcus senilis. If the patient be not much over fifty, the arcus may be taken, as has been before alluded to, to indicate a more or less general fatty degeneration of internal organs. The upper part of the cornea should be care-

fully explored, if the eye appear to be free on cursory examination. Absence of the arc is, of course, a favourable point, and its presence, though important, must not be hastily estimated without inquiry into its history and duration, and the corrective testimony of corresponding signs and symptoms.

If the urine be free from albumen and other foreign matters, it is also favourable; and if albumen be present, the secretion should be tested on more than one occasion, to ensure a decision that it is not an accidental phenomenon.

If examination of the heart result in the discovery that this organ is free from hypertrophy or other disease, the prognosis is immensely propitious. The habits of living, and the symptoms which impelled the patient to seek advice, should be elicited, and it is to be remembered that what are termed "prodromata" are not to be viewed as direct forerunners or intimations of impending cerebral hemorrhage, but as tokens of intracranial disturbance from disease of the vessels, and consequent mal-nutrition, or of other organic alterations, which may, or may not, lead to extravasation. If the heart present physical signs of enlargement or dilatation, arcus be distinctly visible, and there be complaint of languor, apathy, feebleness, low spirits, incapacity of active exertion, and general infirmity of purpose, the prospect is peculiarly unfavourable, especially in conjunction with albuminous urine. Yet under these circumstances much may be occasionally done by observance of injunctions adhered to for a considerable time.

In a person out of health, with commencing arcus

of recent date, in whom the heart is not markedly altered, and the urine not albuminous, recovery and removal of the zone on the cornea may frequently be accomplished by appropriate treatment. The circumstances of life, state of plethora, habits of the patient, and mental condition, must guide us, and hygienic directions be based on each particular case. A just mean of temperance is to be enjoined, with light unstimulating nitrogenous diet.

Inordinate distraction of mind is to be moderated and grief alleviated as much as possible, as mental tranquillity is of primary moment. All sources of annoyance and excitement are consequently to be avoided. Moderate exercise, short of fatigue, should be taken, according to the strength. Nutrition must be rectified and assisted by iron, the mineral acids, and phosphoric acid or the hypo-phosphites, in some cases, to restore the tone of the nervous system. Iodide of potassium and arsenic are most useful if there be proof of syphilitic complication. Rhubarb pills or mild salines may be taken if required. Change of air is to be recommended, and chalybeates drunk at the spring are desirable, as the gentle amusement and society of a watering-place contribute to cure. In fact, attention to what our forefathers entitled the *six non-naturals*, and preparations of iron are the most efficient remedies. In early stages we may thus rapidly improve—

> "A body yet distemper'd,
> Which to his former strength may be restored,
> With good advice and little medicine." *

In conclusive demonstration that fatty degeneration

* *Henry IV.*, Part II.

can be arrested and dispersed by analeptic methods, Mr. Canton has noted, in certain cases, that the *arcus has disappeared* with amendment of health. This, although on first reflection improbable, is sustained by the fact that tonics and judicious treatment remove the symptoms and restore health and strength, failure of which, if dependent on degeneration of tissue, can only be recovered from by improvement and cessation of the pathological cause.

Although atheromatous degenerations are, in a measure, eradicable by obviation of the origin of production, it is highly improbable that calcareous deposition can ever be rendered amenable to therapeutics.

The belief that people with large heads and short necks are signally prone to apoplexy, though transmitted from remote ages and generally accepted by authors, I conceive to be only so far correct, inasmuch as such persons are liable (are they peculiarly liable?) to cardiac and atheromatous lesions. In like manner, the disease is reported to be hereditary; and I accede to the doctrine of morbid inheritance to this extent—that it indicates a disposition to the ancestral physical constitution, gouty, tubercular, degenerative, or otherwise—a liability to the pathological tendency, rather than to the particular disease.

Under these heads it may be introduced that Boerhaave insisted that short necks were *always* found in apoplectics. Ponsart affirmed that small heads disposed to apoplexy. Portal thought the disease so strongly hereditary that he made it (art. IX.) a variety of the affection, whilst Rochoux, with whom I entirely concur, believed, with regard to temperament and *embonpoint*, that there is no appreciable external sign

which discloses a liability to apoplexy; and Rokitanski declares that the so-called apoplectic constitution is mere hypothesis.

It is to be deplored that fatty and atheromatous invasion of the tissues has often extended too intimately and become desperate before the physician is consulted; for it is extremely difficult to impress upon patients the danger of the remote or deferred consequences of intemperance, and of those habits and indulgences which lead to degeneration.

The first warnings of declining health, if obscure, and (to them) almost inappreciable, generally pass unheeded, and even when the gravity of the disease is unmistakably declared, they often, until it is irretrievable, affect indifference.

> "That what we have, we prize not to the worth
> Whiles we enjoy it; but being lack'd and lost,
> Why, then we rack the value, then we find
> The virtue that possession would not show us
> Whiles it was ours." *

> "Virtutem, incolumem, odimus,
> Sublatam ex oculis, quærimus, invidi." †

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* *Much ado about Nothing*, Act IV.

† Hor., Lib. III. Carm. XXIV.

PART II.

ON (SO-CALLED) NERVOUS APOPLEXY, ON CONGESTION OF THE BRAIN, AND SEROUS EFFUSION.

(*Reprinted from the "British and Foreign Medico-Chirurgical Review,"* vols. xxxvii.–viii.)

"The condition which we denominate coma is not characteristic of any one condition of the brain, but may exist in connexion with diseases which are very different or even opposite in their nature; it does not prove the existence of any compressing cause, and particularly has no necessary connexion with effusions in the brain."—*Dr. Abercrombie*—"Researches on Diseases of the Brain and Spinal Cord," p. 4.

PART II.

ON (SO-CALLED) NERVOUS APOPLEXY, ON CONGESTION OF THE BRAIN AND SEROUS EFFUSION.

To render the argument more complete and intelligible, at the risk of repetition, I prefix a very brief abstract of the presumed peculiarities of the endo-cephalic circulation.

Drs. Kellie, Alexander Monro, Abercrombie, Clutterbuck, Bennett and John Reid, and Drs. Burrows, Watson, Williams, Todd, Copland, Kirkes, Carpenter and Neil Arnott, whilst disagreeing, more or less, respecting the variability of the circulation within the cranium, its amenability to atmospheric pressure, and the importance of the cerebro-spinal fluid, are in harmony so far as the general admission of an intracranial *plenum;* and with most, a disproportion, or want of just balance, of blood in the different orders of vessels is regarded as the cause of congestive pressure (how demonstrable we are not informed), and the proximate origin of many comatose diseases. But in opposition to these surmises, it cannot be doubted that the circulation in the brain, and the degree of vascular turgescence of its ganglia, vary even under normal physiological processes, which deviation may involve alterations in the relative complement, or in rapidity of flow, of blood in arteries, veins, or capillaries, or in its absolute amount in the entire series of vessels, as Mr. Durham

has conclusively shown that the brain during sleep is in a comparatively bloodless condition. The circulation, as elsewhere, must be also much controlled by local (nutritive) forces, and the total volume of blood supplemented by the rise and fall of the serum in the cephalo-rachidian cavity, though the quantity of this is limited, and it can be but gradually effused or absorbed. It must, in addition, be borne in mind, that the manner of distribution of the blood in the brain, *during life*, is a consideration quite apart from the differences which are observed in the amount of fulness of the vessels *after death*, as states of post-mortem engorgement may depend on mere plethora, position, or mode of dying, whilst an exsanguined condition of the brain may be alike perfectly consistent with plenitude of the vessels, if there be dilution or poverty of blood from hemorrhage or spanæmia.

Under ordinary circumstances, it is evident that adequate provision is ensured against the exigencies, whatever they may be, which arise from fluctuations in the ingress of blood to, and its egress from, the brain, as during excited action of the heart and lungs, in running, coughing, straining, and under various intellectual and emotional activities.

When we reflect that those pursuing the most laborious employments, and exposed to the most violent exercise, as artisans, athletes, tumblers, divers, players on wind instruments, and children, are not prone to apoplectic seizures, I contend—as will be more developed in connection with pathological and traumatic lesions—that there is not a shadow of evidence that simple modifications in the cerebral circulation are capable of exerting any material pressure on the brain;

nor, *à fortiori*, that such (hypothetical) pressure has any influence in the causation of apoplexy.

Apoplexia nervosa aut absque compressione (apoplexie nerveuse sans matière ou sans lésion appréciable of subsequent French authors) was a term invented by Kortum in 1785, to include those cases of coma and *sudden death* in which no morbid appearance could be afterwards detected within the cranium, and where death was supposed to be due, according to the fantastic pathology of the period, to arrest, retardation or extinguishment of the animal spirits—*i. e.*, of the more subtle and volatile parts of the blood separated in the brain—to relaxation of the nerves, to spasm of the meninges, or of the nerves and vessels of the encephalon. Cases so viewed (quandoque nullum est vitium conspicuum in cerebro nec in cerebello) are recorded by Willis, Bonetus, and other older writers.

Morgagni * treated "De apoplexiâ quæ neque a sanguine neque a sero est," not in a *nervous* sense, but to illustrate certain obscure and insidious cerebral and other diseases. Thus—

Art.	II.	Pus in skull.
,,	IV.	Much pus in left ventricle of brain.
,,	VI.	Disease of dura mater. Liquefaction and extensive disease of brain.
,,	XI.	Sanious matter on the surface of anterior lobes. Brain very flaccid (summâ flacciditate).
,,	XV.	Substance of right hemisphere very brown (valde fusca).
,,	XVII.	Sudden death (homo eodem quo cadebat puncto temporis est mortuus).
,,	XIX.	Sudden death (cor magnum et flaccidum).

* Lib. I. epist. V.

The last two cases show the truth of the assertion of Heberden,* that *all sudden deaths are put down to the account of apoplexies*, when we find the illustrious Morgagni so committing himself.

Dr. Abercrombie believed that apoplexy might prove fatal without any morbid appearance, or with appearances so slight as to be altogether inadequate to account for the attack. For such cases he employed the term of *simple apoplexy*, and deemed them to depend upon a cause which acts simply upon the circulating system of the brain, producing a derangement, which takes place speedily, and is often as speedily removed. He relates cases (97, 98, 99, 100, 101) to support his views; but the histories and post-mortem details, which are most scanty, do not, I submit, irrefragably establish them as examples of primary simple apoplexy. In all, coma was the only leading symptom, and case 101 was undoubtedly uræmic, as the patient had anasarca and effusion into the thorax and abdomen. The others might likewise have been of renal origin (state of kidneys not recorded), or the result of narcotic poisoning, softening, plugging of the vessels, or of some other unrecognised cause of disease.

Dr. Clutterbuck, in like manner, affirmed that there are numerous instances on record of apoplexy proving fatal, where no change in the structure or condition of the brain could be detected, that was at all adequate to explain the symptoms, or to account for the death of the patient. He did not, however, confirm this statement by actual cases, but evidently followed Dr.

* Comments, ch. LXIX.

Abercrombie, as he observed an arrangement and classification similar to that physician.

Dr. Cooke, nevertheless,* expressed an opinion that the spasmodic, convulsive, or nervous apoplexy ought to be classed amongst the symptomatic affections. Dr. Hope believed that a patient might die with symptoms of congestion, yet after death not a vestige of any morbid condition in the brain might remain. He thought the refrigeration of the blood, and its accumulation in the great vessels might remove all appearance of increased vascularity.

Dr. Hughes Bennett is of opinion, that in individuals *who have died of apoplexy*, the brain may present in every respect a healthy appearance, the most rigorous and careful inspection failing to discover any morbid change. He candidly admits, however, that such instances, recorded by Morgagni and others, were, until lately, attributed to want of care during the examination. He considers the pathological condition of the brain, in these cases, to be temporary pressure on the encephalon by over-distension of its vessels with arterial or venous blood, either from *increased* or *diminished* action of the heart. After death, of course (he argues), this is not to be detected, the tonic contraction of the arteries is alone sufficient to empty them of their contents, and turgidity of the veins may remain or not, according to the symptoms immediately preceding death and the position in which the body is placed. He regards pressure to be the chief agent in the production of *all* cerebral diseases! Dr. Burrows also does not seem to be surprised that apoplexy, from vascular congestion, should prove fatal,

* Nervous Diseases, vol. I. p. 269.

without any lesion to be subsequently detected in the brain; but Dr. John Reid* sensibly remarks, that the disappearance of congestion is a supposition, not a fact, of Dr. Burrows, as no proof is offered in support.

In reference to apoplexy, Dr. Watson observes that we may detect no deviation whatever from the healthy structure and natural appearance of the brain. The congestive pressure (if it, indeed, existed) has left no prints of its action.

Dr. G. B. Wood, of America, respecting the occurrence of nervous apoplexy, states that it is generally admitted, death may occur with all the phenomena of apoplexy, without leaving any observable lesion in the brain. He deems such cases to be, at least, very rare, and mentions the suggestion which has been already advanced, that most of them might be referred to the existence of Bright's disease, and Dr. James Arthur Wilson, above thirty years since, wrote to prove that many apoplectic attacks, without lesion in the brain, were due to this cause.

To come more directly to the subject. The statements concerning nervous apoplexy, as far as I can ascertain, are never supported by indisputable facts—that is, by recorded cases free from cavil, and I challenge pathologists, who endorse them, to supply the deficiency. On the other hand, I shall narrate three or four histories, militating against the hypotheses which have been propounded to explain the absence of morbid appearances in certain forms of fatal coma.

I previously venture to append a reflection of the

* Monthly Journal of Medical Science, August, 1846.

observant Heberden, to indicate that a physician of the last century was not satisfied with the pathological doctrines of the period. "Theory may teach, but will find some difficulty in proving, that apoplexies must arise from a compression of the brain, owing either to a distension of the blood-vessels, or to extravasated blood from their rupture, and that the energies of the nerves can be deadened by no other cause beside fulness."*

Case 1.—I was requested by Dr. ——, of St. John's Wood, to accompany him to a post-mortem examination of a gentleman, aged thirty, who had died, with apoplexoid symptoms. The doctor had been hastily called to this patient, and found him in a state of coma. He had been lying in bed, on account of pain in the knee (rheumatic?), and had taken medicine from a druggist in the neighbourhood. He was apparently well, two hours before swallowing a draught, and was playing with a child who had entered the bedroom. He was soon afterwards found insensible, snoring loudly, and he died, without change, in spite of the remedies adopted.

An examination was considered necessary, which was performed two days after death.

There were no morbid appearances within the cranium,—to which we naturally first appealed, with the exception of trifling congestion of the veins of the pia mater posteriorly, probably from gravitation. There was no softening or serous effusion. The viscera elsewhere were also healthy, and there was not any obvious

* Art. "Paralysis et Apoplexia."

cause of death. As there had been pain about the left knee, it was cut open, but there was no trace of inflammation, or of purulent effusion within the joint.

Dr. ——, before the autopsy, unknown to me, went to the druggist, as he thought the symptoms referred most suspiciously to poisoning, and on examining his book, which was blotted, found a draught entered containing morphia. The symbol opposite to the morphia was gr. ss according to the druggist, gr. iss according to the doctor.

The friends, however, desired the affair to be hushed up, as the young man was on the eve of marriage, and the druggist was in some way connected with the family. According to my position in the matter, I was bound not to interfere, but I, nevertheless, rather freely expressed my sentiments.

At my suggestion the certificate was filled up—

{ Rheumatic pains.
{ Coma. (P.M.)

and was so registered. I entertain no doubt that this was a case of narcotic poisoning, and would have been to our forefathers a case of *nervous apoplexy*.

Case 2.—Henry H ——, aged twenty-eight, had been a groom, but unable to work for two years past in consequence of disease of the left knee-joint, for which he had been in and out-patient of St. Mary's Hospital. He died suddenly on April 9th, 1860, whilst sitting up in bed drinking a cup of tea. This was unexpected by his wife and friends, and the medical men at the hospital refused a certificate. He was never known

to have suffered from any cardiac affection. The autopsy was performed forty-eight hours after death, in company with Mr. Gascoyen. Rigor mortis well marked in extremities and jaw. Face pale. No foam at mouth; features placid. No indication of violence, but sugillation of posterior upper part of trunk.

Head.—Calvaria rather thick. Dura mater slightly adherent. Slight subarachnoid effusion. Much prominence of veins of pia mater. Brain substance normal. No extra vascularity. Scarcely any fluid in ventricles. Sinuses at base rather gorged. No morbid appearance in cerebrum or cerebellum.

Thorax.—Right lung healthy, but lower lobe congested posteriorly. No adhesions, no effusion. Left lung same condition, but old pleural adhesions. About a drachm of serum in pericardium, no adhesion. Heart flabby, weighs eleven ounces. No valvular disease, but wall of left ventricle rather thin, flabby, and the organ generally larger than natural. A dark, but partially fibrinous, clot in right auricle, which distends its cavity and passes down into the right ventricle, interlacing firmly with the columnæ carneæ, and, at the latter situation, light in colour. Much black semi-coagulated blood also in right cavities. Stomach moderately large, almost empty. No erosion or ulceration of mucous surface, but pinkiness of the lining membrane in parts, as if digestion had been recently proceeding. Odour merely cadaveric.

Intestines mostly pale, contain some fluid, lining membrane healthy. Bladder empty. Spleen natural. Liver pale, healthy. Gall-bladder distended. Right kidney healthy, pale. Left kidney healthy, not so pale. No fluid in peritoneum.

Case 3.—M. A. L——, aged thirty-two, died suddenly in bed. There were no external marks of violence. The *brain* was healthy, as were also the membranes. There was some serosity at the base of the skull, and a little sanguinolent fluid in the ventricles. The *lungs* were much congested, and generally emphysematous; the bronchial tubes extra-vascular, and contained frothy mucus. There was about a pint of serous fluid in either pleural cavity. The *heart* was flabby, much enlarged, containing clots and dark fluid blood on both sides. The left ventricle was hypertrophied, and dilated. The right ventricle was also dilated. The aortic valves exhibited warty vegetations. The other valves were normal. There was no fluid in the pericardium. *Stomach* was very large, containing much semi-digested material. Intestines healthy. The spleen and liver were much enlarged, the kidneys healthy. There was some serum in the peritoneum.

Case 4.—J. M——, a man about sixty years of age, suffering from reducible hernia (right), and slight bronchitis, died suddenly on January 2nd, 1854.

Head.—Brain natural, not unusually congested. Substance healthy. About two drachms of fluid in each ventricle.

Thorax.—About an ounce of turbid serum in pericardium. Heart weighs rather over ten ounces, and is dilated, thinned, and flabby. The muscular tissue is pale, and there are yellowish spots on the endocardial surface. In the heart generally, there is substitution of fatty for muscular substance, especially in the parietes of right ventricle, midway between base and apex. There is no valvular disease. Much dark fluid blood

in right cavities, and some on left side. Lungs are congested and emphysematous. The bronchial tubes afford evidence of chronic bronchitis. There is no softening of the mucous membrane. Old adhesions of pleuræ on both sides, and several ounces of fluid in each cavity. Slight exudation of recent lymph on anterior surface of left lung near its apex. Some congestion of liver. Stomach contains some yellow semi-digested matter; its mucous membrane is not softened, but here and there a little injected.

Intestines healthy, presenting slaty discoloration. The hernia presents trifling ulceration on its inner surface, but no evident injection. Kidneys, spleen, and bladder healthy.

In the majority of instances, I believe, the coma in (so-called) nervous apoplexy to be due to uræmia, which, as Dr. Richardson remarks (Asclepiad, vol. I.), the unlearned as yet call apoplexy. This physician does not particularly refer to the complaint in the old, or in connection with the small contracted kidney (of which I shall speak further), but in one of his cases, in a woman, aged thirty-four, with enlarged kidney, there was *no evidence of congestion of the brain, nor of effusion; the membranes were natural, and the ventricles contained no fluid.*

He also notices that the effects of the scarlatinal poison may simulate apoplectic coma, and in two memorable instances of my own, I can affirm that one child, about eight or nine years old, died from sudden invasion of epileptiform coma (toxæmic) whilst malignant scarlatina was prevalent in the house.* In the

* See Dr. West, p. 24, for an analogous case.

other case, a boy about fourteen, convalescent from the disease, was also *suddenly* carried off, from arrested renal function and accumulation of urinary elements in the blood.

Occasionally, fibrinous occlusion of the cerebral artery—the middle one, most usually—may cause death, especially in young adults, and be referred to nervous apoplexy, if the vessels remain unopened by an incautious observer. Dr. Markham in his work "On Diseases of the Heart" (Appendix II.), records, in detail, a striking instance in a woman of fifty, of infarction of the innominate, right common carotid, left internal carotid, and middle cerebral arteries, in which there were apoplectiform phenomena with hemiplegia, and death in sixty hours.

In Dr. Abercrombie's thirty-fourth case, there was plugging of the basilar. Further information on this subject may be obtained in Dr. Kirkes' original paper in the "Medico-Chirurgical Transactions," vol. XXXVI.

In the present state of medicine, it is unnecessary to enter at length into the manifold causes which may provoke more or less pronounced comatose symptoms. If the appearances within the cranium be of negative character, we may expect the cause of death to be manifested in other organs, or be owing to shock, syncope, lightning, or a physically undetected, or undetectable poison.

There are, of course, numerous sources of fallacy. Thus, as I have already noticed, under diagnosis, death may depend on an anomalous type of pneumonia, or severe concussion, with, or without intoxication. Delirium tremens also with our predecessors, and (unobserved) cerebral softening, have also, it is almost

certain, been regarded as nervous apoplexy, as well as cases of homicidal, suicidal, and accidental narcotism. Pulmonary apoplexy, too, has given rise to supposition of cerebral apoplexy, and under such a diagnosis, we must conceive that the pulmonary structure is not submitted to careful incision to correct the error. Cases of angina pectoris, of cardiac coma, and apnœa, of pressure on the recurrent nerve, may have increased the list, as also the toxic coma of gout and pericarditis, cases of catalepsy, and even hysteria, and the collapse ensuing on rupture of internal aneurisms, as the most renowned pathologists of yore often confined their examination to the brain, and afford no history of symptoms.

Even impaction of food in the œsophagus may prove a perplexing cause of death. I once examined the body of a man, who, I was informed, staggered, and fell dead in the yard, outside a cottage. This appeared to point to heart or brain affection, and I had received no further account of the previous circumstances. A cautious but fruitless scrutiny was made of all the viscera, before the foreign body was detected—a piece of beef weighing two ounces, firmly implanted behind the larynx.

The brain, I admit, may not present any appearance sufficient to explain the cause of death, but it is not, therefore, to be concluded that the coma (apoplexy) is a primary neurosis, in the sense employed by Kortum and his supporters—that is, dependent on some intrinsic undefinable disturbance, but *symptomatic* or consecutive to some well-recognised, if ascertainable, morbid operation. Perhaps, the old proverb contains some truth, that "Venus nimia in senibus multum ad

apoplexiam disponit," that excess in this particular, as in any other, may so depress the functions of the nervous system, already nearly exhausted, as to decide the term of life; but this is rather death commencing at the brain, previously atrophied, or otherwise undermined (necrencephalus), than mere idiopathic nervous apoplexy.

To return to the most common mode of production of coma (cerebral hemorrhage excluded) Dr. Basham, "On Dropsy connected with Disease of the Kidneys," points out that renal disease will give rise to symptoms which often appear to have little reference to the renal functions, and which may be, and frequently are, accepted rather as evidence of disease of the cerebral organs. In these cases, *dropsy is altogether absent*, or the *anasarca is so trifling* in amount, as to excite but little attention. Symptoms referable to the brain are those which exclusively attract the notice of the patient, or to which the care of the practitioner is directed. Headache, frontal or sincipital, dimness of vision, simulating the approach of cataract, slight convulsive attacks of epileptiform character, followed by more aggravated paroxysms of convulsive disorder, constitute the class or series of symptoms; the other is expressed by headache and imperfect vision, followed by dulness of intellect, sluggishness of manner, frequent sopor, or drowsiness, or occasional stupor, terminating usually in fatal coma. The absence of dropsy, and the insidious approach of these symptoms, are well calculated to misdirect the attention of the medical practitioner, and the remedies employed to avert the simulated and suspected apoplectic seizure may be those which hasten forward the fatal effects of

uræmic poisoning. The form of renal disease which usually develops these symptoms is the atrophied, shrunken, and nodulated kidney (p. 147).

Dr. Basham does not allude to its frequency in old persons, or in those who have prematurely degenerated, but thinks, in conjunction with the late Dr. Todd, that the wasted kidney is closely connected with the gouty diathesis. Yet he owns, that it may be met with in persons who have never exhibited gouty symptoms. He does not consider that the shrunken kidney represents an advanced stage of Bright's disease, as in this, he believes, the kidney is usually increased in weight. On the contrary, Dr. Handfield Jones maintains that the contracted granular form is the one most typical of true degeneration. Agreeing on the whole with Dr. Prout, he does not regard it as necessarily connected with renal hyperæmia, although it may be thus complicated or intensified. Dr. Bright* described three forms of diseased kidney connected with albuminous urine. In the first, the organ is natural in size; in the second, enlarged; in the third, contracted. Dr. Bright expressed uncertainty whether they were distinct or only modifications, and more or less advanced states of one and the same disease. Dr. George Johnson and Dr. Maclachlan insist that the small contracted kidney is the variety *most incident to age*, and agree that it may proceed to extreme degree in absence of the supervention of dropsy.

Without venturing to merge deeply into renal pathology, I feel confidence in asserting that all who are accustomed to examine aged subjects will allow that

* Medical Cases, vol. I. pp. 67-69.

the small contracted kidney is, in them, by far the most common appearance, that it is absolutely frequent, and a more or less essential characteristic of senile degeneration, though, in many instances, it may not manifest itself at all during life, or until the occurrence of epileptiform or comatose symptoms. Its advent at earlier periods, I hold to be indicative of low vital power and premature physical impairment.

In gout, the kidney may present similar alterations, in consequence of the excessive demand on the urinary functions and the diathetic influence, but that such degeneration peculiarly or invariably depends on this complaint is unquestionably erroneous.

As with atheroma, in advanced life, it is difficult to declare how far the alterations in the kidney are physiological, how far pathological in their nature, but they clearly partake more of atrophy and senile changes, than of active disease. It is confirmed by all observers, that these renal conditions may not, and often do not, give rise to dropsical effusion, and Dr. Maclachlan remarks in his "Diseases and Infirmities of Advanced Life," that "the great majority of persons affected with the shrunken kidney die comatose, without any subsequent appearance in the brain, the symptoms proceeding entirely from the poisoned state of the blood" (p. 583).

In briefly considering the subject of *cerebral congestion* as an efficient *cause* of fatal apoplexy, I would, at the outset, inquire if any practitioner can advance an undeniable example—verified by accurate detail of symptoms and exhaustive autopsy—of death from mere primary sanguineous congestion?

Dr. Abercrombie lays little stress on cerebral congestion as a cause of apoplexy; in fact, I am not aware that he once employs the term amongst his post-mortem appearances, though in one or two instances he refers to *increased vascularity*. He ascribes apoplexy (p. 206) to simple (nervous) apoplexy, in which there are no appearances to be subsequently detected; to serous effusion, which he regards as a consequence of simple apoplexy, and not a primary state; and to sanguineous extravasation. Dr. Clutterbuck referred the symptoms of apoplexy to impeded or interrupted circulation in the brain, leading to suspension of the sensorial functions, from pressure operating upon the blood-vessels, so as to hinder mechanically the passage of blood through them.

Dr. Sims, physician to the St. Marylebone Infirmary, in 1835, held that a loaded state of the blood-vessels of the brain is sufficient to produce all the symptoms of sanguineous apoplexy, and to occasion death without extravasation; and that convulsions in children arise in general from cerebral congestion, and are essentially cases of sanguineous apoplexy.* He admitted that the presence of serum, to which I shall presently refer, is not absolutely explanatory of the symptoms observed during life. By a parity of reasoning, the appearances of congestion are not of certainty indicative, being more probably a *consequence* than a *cause* of the morbid phenomena, and the cases of Dr. Sims in which cerebral congestion was observed are imperfectly recorded, and in several, the thoracic and abdominal viscera were not inspected. In some, in

* Med. Chir. Trans., vol. XIX.

addition, other diseased appearances were noted in the brain. In the case of children, above noticed, the convulsions, it is generally agreed, are nearer allied to epilepsy than to apoplexy, originating in eccentric irritation, not congestion. In many instances, death is due to laryngismus stridulus. For my own part, I have more frequently seen coma, in children, connected with spanæmic or deteriorated quality of blood, than with evidences of vascular engorgement of the brain. Moreover, the convulsions, in them, for the most part precede the so-called congestion, not the congestion the convulsions.

Dr. Burrows does not mention *any particular case* of cerebral congestion causing apoplexy, but appears rather to regard it as an axiom, and assume it as a postulate. The cases referred to Dr. Abercrombie, Dr. Cooke, and the rest, are not described, but seem more fairly attributable to disease of the heart or uræmia than to perverted circulation in the head (congestion). I have already remarked, that Dr. Abercrombie records no case which he imputes to cerebral congestion, and after careful perusal of Dr. Cooke's learned treatise on "Nervous Diseases," I cannot find any of the cases which are referred to by Dr. Burrows, or which will, in any way, corroborate his views! Dr. Maclachlan again, though he treats of *congestive apoplexy*, gives no illustrative examples.

Dr. Burrows goes so far as to believe, that apoplectic coma is rarely dependent upon the extravasation of blood (p. 92), but in the vast majority of cases, upon the pressure induced by vascular congestion. He relates two histories (pp. 92 and 96), *in which both the patients died comatose*, to support his views;

but they serve to demonstrate what he strives to disprove, and the intervals of *impaired* consciousness, which occurred in their progress, are explicable by the peculiarity in the site and mode of the effusion.

Of a certainty, as Dr. John Reid points out in the article before quoted, "In analysing closely the matter contained in most treatises on practical medicine, facts and opinions are with difficulty separated from each other, and no satisfactory evidence can be found of the evidence of truths upon which important inferences are founded."

Dr. Watson adopts the tenets of Dr. Burrows, that pressure is the ordinary cause of apoplectic coma; and that pressure can, and does, *sometimes* cause coma is incontestably established, but that cerebral congestion, pure and simple, can induce coma, *quoad* pressure, is a vastly different proposition.

Andral, in his "Clinique Médicale," gives five cases under the head of cerebral congestion, which I beg to remark, in all reverence to this distinguished physician, are not to be unequivocally relied on for determining the point disputed. In only one case are the kidneys mentioned (gorged with blood), and of four he observes, "Is it not a circumstance worthy of notice, that the four cases of cerebral congestion now reported regarded individuals labouring under chronic affections at the time the brain became congested in them? In three of them, hæmatosis was for a long time vitiated; they were meagre, bloodless, and appeared to be in a condition entirely opposite to that which is usually laid down as favouring cerebral congestions." Two of these four were phthisical subjects, and the fifth case

is a history of cerebral symptoms intercurring in ordinary pulmonary tuberculosis.

Dr. Bright,* under the head of "Pressure," mentions two cases which are said to have been due to cerebral congestion. In Case 86, there was giddiness, loss of sight, and of consciousness, in a stout-made man, who was, however, exsanguined. After depletion, tonics did most service. In this case, I humbly suggest, that the assumption of *simple* congestion is not satisfactorily proved, and in Case 87, in which there was unconsciousness with convulsion, and slight, but persistent, paralysis, the diagnosis is favourable, I think, to limited hæmorrhage.

Cases 88, 89, 90, 91, related as samples of cerebral congestion, were dependent on narcotism. Cases 92, 93, 94 are cases of bronchitis with cerebral congestion; 95, 96, 97 are cerebral congestion from emphysema; 98, cerebral congestion from hooping cough; 99, effusion of blood on the brain in a child in connexion with the same disease, the account being scanty and the history wanting. Case 100, is congestion in the vessels of the brain in a man dying of hepatization and gangrene of the lungs after a severe scald; 101 is vascular congestion of the brain in fever, and 102 cerebral congestion in fever. These are the whole of the cases of congestion of the brain to be found in Dr. Bright's work. Of all these, only the first two, *which recovered*, are admissible, and these are fairly open to question. The remainder are secondary, and connected, in almost all instances, with respiratory disturbance, and many of them might be dependent on

* Reports of Med. Cases, vol. II. part 1, sec. II.

cadaveric states. In all, at least, the cause of death is evident, without the necessity of resorting to cerebral congestion for explanation.

Dr. Copland says, that in a large proportion of cases of simple primary apoplexy, *i. e.*, without extravasation, *excessive injection of the vessels* of the pia mater and *engorgement of the whole vascular system* of the encephalon are the chief lesions. This (he owns) is of comparatively rare occurrence. It constitutes the *coup de sang* of the French, and is observed in those cases of *coup de soleil* which prove rapidly fatal. But Dr. Aitken, in his "Science and Practice of Medicine," justly maintains that it is erroneous to describe Insolatio or Erythismus tropicus as of the nature of apoplexy. The opinion possesses additional weight, as this physician would give a very wide signification to the term apoplexy, and use it to characterise a group of symptoms, irrespective of the anatomical conditions upon which they may depend. Thus he would include congestion of the brain, hemorrhage, sudden serous effusion, local cerebritis, tumours of the brain, meningitis simple and tubercular, the progress of various zymotic and constitutional diseases from blood-poisoning, anæmia, disease of the heart, and vascular obstructions under this head.

Death occurs in cases of insolatio, according to the concurrent testimony of military surgeons, from syncope or apnœa, and the chief morbid appearance usually observed is excessive engorgement of the lungs. In many cases the brain is healthy, without trace of congestion or accumulation of blood. Dr. Morehead states, that the blood in the vessels is always fluid (Aitken). The essence of the disease seems to be intense exhaus-

tion, from the combined effects of over-fatigue and subjection to a very high temperature—intracranial changes being adventitious, not causative, or even constant.

Is it permissible to adduce cases like the preceding —cases of insolatio, of cerebritis (?), of tubercular disease of the brain or its membranes, of delirium tremens, of uræmia, of secondary or post-mortem engorgement of the vessels of the brain, &c., as examples of cerebral congestion constituting primary apoplexy? It is undoubtedly important to define accurately the limit and meaning of the word, as one of our most distinguished practical physicians, Dr. Watson, denominates by "apoplexy," "coma occurring suddenly, or coming on (at least) with rapidity," which would include every affection attended with sudden loss of consciousness, whether arising from hemorrhage, tumour, intoxication, typhous complication, coma of epilepsy and hysteria, injuries to the head, softening, or even asphyxia, syncope, the terminations of cerebritis and hydrocephalus, narcotism, or the action of deleterious gases.

Practitioners generally make a distinction between *apoplexy* and *genuine apoplexy*, so as to render justifiable a remark of the late Dr. Jones Quain, that when we look into works on medicine, we are struck with the confusion which exists in the nomenclature of cerebral diseases. More precision has been attempted by our French neighbours, though Littré complained ("Dict de Méd.," art. Apoplexie) that "Le mot apoplexie, souvent aussi vague dans la bouche du médecin, que dans celle du malade, se prête à toutes les interpretations de l'ignorance."

"In the nervous centres, as elsewhere," says Andral, "before the production of hyperæmia or anæmia, we must conceive a primary modification of the force, whatever it is, which subjects the cerebral circulation to certain rules. In the midst of these numerous currents, of these oscillations of globules, which pass within the organic tissues, how many causes constantly presented, and whose influence is entirely unknown to us, may derange a current and modify the distribution of the globules? * * * When we thus examine minutely the grounds of the question, we soon see that hyperæmia and anæmia in the brain, as in other parts, are themselves but *secondary phenomena*—mere effects. But these effects, inconstant and variable, do not necessarily follow the action of the cause; they may be wanting, and yet the symptoms will still continue, for they depend less on the state of cerebral hyperæmia or anæmia, than on the organic modification which precedes them, and which causes them. Thus, our post-mortem examinations show us, for the explanation of identical symptoms, sometimes a state of hyperæmia, sometimes a state of anæmia, sometimes nothing unusual in the quantity of blood contained in the brain; and in this brain, moreover, no lesion appreciable by our present means of investigation; the reason is, because these means do not show us all; by them, we as yet discover nothing but effects; the material modification which incontestably precedes them, requires not their production in order that disturbance may take place in the functions of the organ. However, once produced, the different lesions which the present state of anatomy is calculated to reveal, may give rise to phenomena which depend on them alone, and which

establish their diagnosis." * This quotation contains the gist of my argument, viz., that the (so-called) nervous apoplexy and cerebral congestion are but secondary states, not the origin of the apoplectiform symptoms. I am aware that the doctrines of Andral may be urged, in some degree, on either side of the question, but they at least favour my views as to the uncertainty which besets the subject, which uncertainty most pathologists will admit. Dr. Watson, in his twenty-first lecture, adverts to the difficulties with which the study of the maladies and disorders of the brain and nervous system is surrounded; and Dr. Brown-Sequard acknowledges, that less is known about the anatomy and physiology of the brain, than about any other organ. The *onus probandi* rests with those who subscribe to the theory of cerebral congestion as a cause of fatal apoplexy, which they adopt, I suspect, on the score of conventional obedience to systematic writers. I therefore reinsist, that general objections and counter-assertions are pointless and futile, unless fortified by the crucial proofs of clinical and pathological observation. I speak with some confidence, the result of special investigation, as during a residence of five years at the St. Marylebone Infirmary, where I had medical care of nearly a thousand aged persons, and great opportunities of noting maladies from their commencement, of ascertaining the appearance after death, and of observing disease in all forms, at all ages, in a hospital of 200 beds, I never witnessed a fatal case which could be clearly ascribed to simple apoplexy or cerebral congestion.

* Translated by Dr. Spillan, p. 92.

Physicians, swayed probably by surgical considerations and physiological experiments, have been altogether too *mechanical* in their opinions concerning apoplexy and cerebral congestion, and have not dwelt sufficiently on the importance of the *quality* of the blood. *All* has been assigned to *pressure*, from augmentation or deviation of the normal amount of blood in the several orders of vessels, without emphasis on the *cause* of the pressure, *if it obtain*. For example, if cerebral congestion causes coma or apoplexy (congestive) from pressure, how can it be explained that inflammation of the brain-substance, or cerebritis, does not, in its earlier stage, produce coma, although there must be notably increased blood in the capillaries and other vessels of the part, and (theoretically) consequent pressure? Verily the pathology of the brain is encompassed with difficulties, but these difficulties are oftener tendered and acknowledged as explanations or finalities, than appreciated as monitors to caution us against misinterpretation, and incite more diligent and profitable inquiry.

It may be said, that venesection has been *advantageously* had recourse to (the contrary might be also stated) in many cerebral attacks, supposed to depend upon congestion; but I reply, that although abstraction of blood may withdraw a given quantity of this fluid from the vessels, it is inexplicable how it can rectify the vascular balance in an (assumed) congested venous or arterial apparatus. Is the practice correctly founded, or does it hold good in spite of the erroneousness of the theory? And I ask each individual member of the profession, how frequently he has employed the lancet in simple primary congestion of the brain?

Much evidence might be collected in favour of bleeding in cases of sudden hemiplegia, many of which depend on discontinuity of the cerebral fibres or infarction of the vessels, and on theoretical grounds we should imagine such a line of practice positively injurious. In other than cerebral disorders, abstraction of blood has been indiscriminately had recourse to with the happiest advantage, if we credit the unqualified testimony of men of the largest experience. Thus in pneumonia, without distinction of cases, the school of Sangrado and Broussais has in its turn had a multitude of disciples; and in fevers, venesection has been carried to extreme by Clutterbuck and his followers, who imagined the febrile state to be associated with, or symptomatic of, inflammation of the brain. I regard the nervous manifestations in fever to depend rather on heteræmic influences, or, as Dr. Todd would say, disturbed polarity, than on congestion of the brain or encephalitis. The treatment of fever by bloodletting, is now generally abandoned and condemned: and this amelioration in practice cannot be ascribed to change of type of disease, but to clearer and more rational ideas on therapeutics. From these sentiments I must not be mistaken for a bigoted anti-phlebotomist, as on one occasion—if anything relating to our art can be certainly declared—I saved life in a severe case of inflammatory anasarca from exposure and renal congestion, by the prompt removal of a very large quantity of blood from the arm. But to return to the state of the brain in fever: Dr. Watson confesses, that he cannot tell how often he has looked, and looked in vain in the brain, for some palpable disorganization, or some effusion, implying pressure. All who are familiar

with the dead house of a hospital are aware, that this fruitless search for some physical explanation of the comatose state, after death by fever, is of very common occurrence (vol. ii., p. 807). In cases denominated congestion, in which benefit has been derived from, or has followed phlebotomy, I believe the relief to be at times due to the effect exercised on a commencing inflammatory state of the brain, where blood has been attracted more than determined to the head, its properties altered, and the nutritive operations interfered with and perverted, as in other tissues, during the inflammatory act. In many cases, the symptoms are probably uræmic, or arise from obstruction to the functions of the heart and lungs, producing disorders viewed as apoplectic. Alteration in the *quality* of the blood, under such circumstances, is capable of producing symptoms commonly referred to pressure from congestion, and we possess as much, if not more, evidence of the existence of the former, as of the latter. That enormous pressure may be exerted in absence of apoplectiform states (there being absence of altered quality of blood), is demonstrated by persons labouring under very large tumours within the cranium, compressing important structures, often remaining unattacked by coma until within a few hours of death. I can vividly call to mind a fibrous tumour as large as a billiard ball springing from the dura mater, and deeply indenting the hemisphere, an aneurism of the basilar artery nearly of the same size and a large malignant tumour of the cerebellum, amongst others, in confirmation. I know, it may be rejoined, that these growths were gradual, whereby the neighbouring parts were absorbed, or by degrees accommodated

themselves to the pressure experienced, or that their position did not interfere with the more important parts of the brain; but these attempted solutions appear highly unsatisfactory, if we are to concede grave and even mortal consequences to a *slight and transient* (*and after death, unrecognisable*) *congestion.*

Not that I deny the brain can become the theatre of congestion, but that congestion, pure and simple, can be reasonably inferred, much less *proved*, to be a sufficient cause of apoplectic coma. Also, that congestion can produce pressure, equal in extent to many vascular variations consistent with health. It is beyond dispute, that coma exists as frequently unaccompanied, as accompanied by any indication of pressure or congestion. If congestion be combined, it may with stronger probability be accepted as the result, than as the origin of the phenomena, to which it is, I affirm, non-essential and sequent.

The most, indeed the only apparent, valid argument of the congestionists is, that in elderly patients of a plethoric constitution, symptoms referrible to the head, as cephalalgia, vertigo, drowsiness, sense of fulness and temporary confusion of thought sometimes occur. But I demand, can even these be shown to depend necessarily on congestion of the brain? Can it be maintained that they are restricted to the plethoric, and that these persons are otherwise cerebrally sound? I admit, that patients with the symptoms I have specified have come under my own observation. I remember in particular (as a type) a sanguine old man, in whom cupping from the neck was periodically practised with marked benefit. I may mention another case which comes under the same category, though occurring in a

youth of fourteen, who experienced severe cerebral symptoms from assiduous application at the easel. Here purgation and rest from his pursuits afforded speedy cure.

In the first instance, the attack I consider to have been owing to alteration in the nervous substance of the brain or its arteries, or to an excessive composition of the blood, giving rise to nutritive impairment, but not to increment in the amount (congestion), but in quality of the fluids within the cranium. In the second case the symptoms are ascribable, not to congestion of the brain, but to protracted mental tension in a weakly boy, passionately addicted to painting. In either case, the ordinary nutritional changes in the nervous substance were deviated from, with consequent accumulation to greater or less extent of toxic materials in the minute vessels.

Many old persons in a state of coma are aroused and recover after bleeding, as attested by Morgagni, Forestus, and others of bygone times, who regarded them as specimens of *serous* apoplexy.

Cases of the kind I have observed and treated, but there is no proof that the attack in these is primarily dependent on inequality in the amount of blood in the cerebral arteries and sinuses (congestion or pressure), but there is considerable evidence in favour of a toxæmic state of the blood from ureal impregnation. The recovery after venesection is to be referred probably to the withdrawal of a portion of the poisonous agent from the circulation, by which time is gained to combat the renal degeneration. I am gratified to find that I am supported in this view by Dr. Richardson, who asserts that venesection is of the highest value in acute

uræmic coma (Asclepiad); but when patients recover consciousness after bleeding, I suspect most practitioners impute the attack to vascular congestion or pressure on the brain. In many cases, the employment of hydragogue cathartics, diaphoretics, and mild diuretics will restore the patient, and a course of iron produce more or less permanent improvement without abstraction of blood. Examination of the urine will reveal, in these cases, the presence of albumen, an indication of degenerescence of the kidney, which I pointed out, when considering the subject of cerebral hemorrhage, as a more or less constant senile change. Recurrence of the attacks in uræmia is especially apt to ensue, more particularly if the patient be not subsequently improved by the exhibition of steel medicines. I have treated a large number of old persons suffering from this affection—the majority without bleeding, and it is difficult to state definitely, under what circumstances the lancet is most demanded. As a rule, its employment is warrantable and serviceable when the coma is very profound, and the symptoms approach those of extravasation of blood; in fact, in certain instances, the diagnosis becomes impossible.

In milder forms of uræmia, compound jalap powder, elaterium or croton oil, with salines and nitric ether, are sufficiently efficacious with or without blisters or sinapisms, and stimulating enemata. On the whole, I would infinitely prefer to rely on alvine evacuants, than on bleeding in anomalous cerebral attacks. Every experienced man must be acquainted, how little benefit accrues on most occasions from the use of the lancet, until the active operation of cathartics. It is much more probable that the disorder is mitigated by the

removal of deleterious matters from the circulation, than by a derivative action decreasing the volume of blood and vascular supply to the brain. Cases of uræmia are analogous to those of ordinary narcosis, and do not require congestion and pressure to explain the morbid state. Congestion is not the essence of the disease, but alteration in the blood, and congestion and pressure are consequences, if present. By prolonged bodily exertion the force of the heart is increased, and the circulation in the brain quickened. Yet it does not lead to cerebral mischief (congestion). On the other hand, protracted intellectual activity is not unfrequently followed by symptoms involving the brain. Under either circumstance, it is likely that the vascular conditions of the encephalon experience modification, but in the latter case only is there *polar* disturbance or altered innervation, to which congestion, if real, is subordinate and consecutive.

Further signs ordinarily held to be dependent on, or conjoined with, congestion of the intra-cranial structures, as flushing and turgidity of the face, throbbing of the temporals and carotids, injection of the conjunctivæ, &c., are but fallacious indications and doubtful exponents of encephalic states. Careful inspections of cases of strangulation are narrated by Drs. Kellie, Monro Secundus, John Reid, and Watson, in which these external characters were found to be coexistent with *non*-increased vascularity of the vessels and tissues of the brain. In a very careful examination made by myself of a child, aged six months, strangled by its mother, the brain was found to be not extra-congested, a few veins of the pia mater were rather prominent posteriorly, and there was fine ramiform injection over

the surface of the hemispheres. No sub-arachnoïd effusion. Brain firmish for the age. About a drachm of serum in the ventricles, puncta vasculosa not abnormally numerous, a little dark fluid blood in the longitudinal and lateral sinuses, and about three drachms of serous fluid at base of skull.

In another child, aged three months, poisoned by laudanum, the brain presented a very pale, almost anæmic appearance. The ventricles were slightly moistened with serum, and there was about a drachm at the base of the cranium. In justice, I should add that the infant was flabby and ill-nourished.

Dr. E. A. Sansom (On the action of Anæsthetics, "Brit. Med. Journ.," Sept., 1864) concludes, from the result of careful experiments, that chloroform, ether, and volatile narcotics, act directly upon the blood. He maintains that the essential concomitant of a state of anæsthesia is sluggishness of circulation; that it is not a condition of hyperæmia of any organ. As in sleep, Mr. Durham has pointed out that the brain is comparatively anæmic, and that the blood in its vessels is not only diminished in quantity, but also flows with a decreased rapidity, so in chloroform narcotism. Dr. Sansom alludes to an interesting case, recorded in the "American Journal of Medical Science" for Oct., 1860, in which chloroform having been administered in extensive fracture of the cranium, it is stated that during the full effect of the narcotic the brain was remarkably pale; and whenever the anæsthetic influence began to subside, the surface became florid and injected.

Professor Ackermann of Rostock some time since

made experiments with the object of ascertaining the condition of the cerebral circulation in asphyxia, which afforded results also opposed to received opinions. He removed a portion of the skull of an animal with a trephine and substituted a piece of glass. After about twenty-four hours, asphyxia was induced by strangulation, submersion, compression of the thorax, &c., and the condition of the brain watched through the glass plate. Asphyxia was found to be invariably accompanied by a condition of anæmia instead of congestion. The congestion of the cerebral vessels, not unfrequently presented after asphyxia, he believes to be attributable to hypostatic hyperæmia of *post-mortem* character. ("Archives of Path. Anat.," quoted in "Brit. Med. Journ.," April, 1861.)

It would therefore appear that in many cases and forms of fatal coma, usually referred to, or said to be attended by, congestion, the latter phenomenon is rather incidental than invariable. If present, it must be regarded as a consequence. Opium, alcohol, chloroform, and carbonic acid cause insensibility from toxæmic influence; not from mere induction of congestion. The congestion is secondary, from dissemination of poisonous elements in the cerebral vessels, ensuing on diminution and arrest of the normal circulating forces—in like manner as in the lungs, asphyxia is not due to congestion, but congestion to the asphyxia or apnœa.

As I have adverted to the lungs, it is not irrelevant to observe that *pulmonary apoplexy* (apoplexie pulmonaire foudroyante) has occasionally proved fatal in a sudden manner and been mistaken for cerebral apoplexy. On subsequent examination, the brain and its

vessels have appeared normal, but lesion and hemorrhagic infiltration have been discovered in the pulmonary parenchyma. Such cases are recorded ("Dict. de Méd.," art. Apoplexie du Poumon) by Corvisart, Bayle, and Andral, where death was almost instantaneous.

In recognition of the above, and that the most eminent of our predecessors often instituted an inspection of the brain only in such and analogous cases, we can repose but little reliance on their statements and researches, although their observations have doubtless exercised important influence on the views and practice of succeeding generations of physicians. I speak not in depreciation of the labours of our illustrious forefathers, as few would accord them merit more liberally or be more sedulous to vindicate their learning and fame. The advance of scientific medicine has been of necessity gradual, and effected by accumulation of legitimate inference and theory, grafted on the facts of physiology and pathology. "Necessitas medicinam invenit, experientia perfecit. Quæ quidem primâ ætate rudis erat ac stupida; progressu verò temporis accedentibus in dies novis observationibus, sibique mutuò facem quasi præferentibus, cuncta præsertim regente, ac moderante rationis lumine, liberalis facta est et erudita." (Baglivi, "De Praxi Medicâ," cap. ii.)

It is, nevertheless, unquestionable that the promulgation and adoption of hasty assumption and unauthorized speculation, even amongst the ablest, have tended enormously to retard its progress. Much incorrect doctrine has sunk to oblivion, much has survived to our day. Owing to the inexactness which more or less enshrouds our knowledge of disease and medical

practice generally, divers current dogmata have been taught and circulated, *ex cathedrâ*, from professorial chairs, which are rather the expression of time-honoured error, or the phantoms of preconception, than the offspring of direct investigation, though most obsequiously assented to by particular sects of past and present practitioners. Even the distinguished Laennec believed that *congestions* arose, under certain circumstances, in consequence of the blood experiencing a rapid dilatation. On no other explanation could he account for the immense losses of blood sometimes occurring in hæmoptysis or menorrhagia, or the sudden hyperæmiæ of internal and external organs witnessed in epilepsy and hysteria.

I have admitted that, under certain conditions, especially traumatic, apoplectiform symptoms are undoubtedly presented as the consequence of pressure, but that such pressure is exercised through the medium of congestion is void of proof or probability. Pressure evokes disorder of nutrition, either from direct effects on the nervous centres, or from alteration in quality of the blood, from stagnation and arrest of the physical changes—a state more allied to anæmia than congestion. The physiology of sleep, the effects of profuse hemorrhage, observations on asphyxia and the action of anæsthetics, with many other trivial facts, warrant the conclusion that unconsciousness is more frequently attended by a bloodless than a congested condition of the brain.

Pressure induced by external lesion is almost invariably complicated by concussion, which alone, in absence of compression, is able to exert most of the phenomena of pressure, demonstrating that changes

apparently inappreciable, in the (polarized) constitution of the brain are equally represented by symptoms commonly attributed to mechanical causes.

Mr. Abernethy, in 1797, in his surgical and physiological essays (Part III., on "Injuries of the Head"), published a series of cases in proof that extensive depression of the cranium—of the parietal, frontal, or temporal bone—might exist unattended with coma, and that recovery might take place without trephining, the skull remaining depressed. In *one* or *two* instances only there was transient stupefaction from the injury, which may, with fairness, be referred to shock or concussion rather than compression. The inevitable inference Mr. Abernethy drew from these cases was that pressure does not always derange the functions of the brain, either at the time or at any remote period. On the other hand, Mr. Abernethy points out that severe symptoms sometimes ensue when the bone is elevated and pressure absent. He remarks that a surgeon might be led to ascribe the mischief in these cases to the depressed bone, if the trephine were not employed; and the success in those recovering without operation to the same instrument, if elevation had been practised. In conclusion, I think it impossible to maintain that vascular congestion or pressure is the *ordinary* origin of apoplexy or comatose symptoms, to the exclusion of nutritive alterations or perversions. Coma is frequently developed when states opposite to pressure or congestion are established, as in syncope, concussion, hydrocephaloid or pseudo-meningitis, sudden hemorrhage and ligature of the carotids; and also when there is no evidence of congestion or abnormal pressure, as in ramollissement, uræmia, cerebral embo-

lism, delirium tremens, fever, and during the incubation and progress of certain acute and specific diseases. On the contrary, in cases where pressure must exist there may be entire negation of comatose symptoms; and, in others, the post-mortem discovery of cerebral congestion may have been unpreceded by coma or any indication connected with the nervous centres. Excessive action of the left heart—if injurious where the minute vessels of the brain are sound—syncope or arrest of blood to the head produces effects, not from variation of pressure, but from modification of the process of nutrition. In such circumstances, by the upholders of the *pressure* theory, we are not enlightened why the cerebro-spinal fluid fails to afford the succedaneous efficacy with which it is, on all other occasions, physiologically invested. After I had ventured to commit myself to the above novel and heterodox views, I derived some complacency from ascertaining that Dr. Wood of Philadelphia has in some measure similarly expressed himself.—"Some appear to consider coma as dependent upon pressure alone. But as this cause operates merely by suspending certain functions, there is no reason why stupor, in its different grades, should not arise from other causes capable of interrupting the same functions, whether by an excess of excitement or a direct sedative impression. I have no doubt whatever that it often results from both these causes, altogether independently of pressure." (4th edition, vol. II. p. 683.) Yet the most recent authority, Dr. Maclachlan, follows the routine opinion. Thus—"Apoplexy appears in very opposite states of the system, or condition of the brain itself in regard to plethora or anæmia, but the *tangible*

cause of the disease is pressure" (p. 133). I would rather say, *intangible*, and Dr. Maclachlan, in a previous sentence, admits that "the proofs of the existence of pressure, in every case, are nevertheless very defective."

Reference to the *quality* of the vascular supply, toxic or spanæmic, or nervous disturbance, will, in all cases, satisfactorily elucidate the seeming contradictions and be equally explanatory of vertigo, delirium, epilepsy, puerperal eclampsia, and insanity—not to mention emotional states—which are incapable of solution on mechanical considerations or supposition of congestion.

Serous or pituitous apoplexy is a variety of the disease which has been for ages, and is even at the present day, recognized as a substantive cause of death amongst infants and adults by a large number of practitioners. It is a common verdict of coroners' juries, whose finding must depend on medical evidence, which for the most part merely testifies to the presence of serum in the cerebral ventricles, arachnoid cavity or sub-arachnoid space of the deceased.

I have been long led to conclude that, independent of the cause of death, it is more usual than otherwise to meet, in the adult, with some amount of fluid, increasing with age, in the lateral ventricles or at the base of the brain, and that its presence has no fixed relation to the degree of vascularity, or engorgement of the brain or its vessels. In young children, however, dying from diseases not of the comatose class, I have more frequently than in those of later years, remarked the absence of fluid from the ventricles and the base of the brain. On reference to my register of

post-mortems in persons over fifty years, not dying of apoplectic or uræmic disease, I find the following account of the fluids within the skull, when they have been noted.

T. M., 65. Brain healthy, serum in ventricles.

J. M., 60. Brain not congested. About ʒij of serum in each ventricle.

J. R., 73. Membranes healthy, about ʒj of serum in each ventricle and fluid beneath arachnoid.

E. H., 78. Vessels very turgid, and substance of brain vascular. Small amount of fluid in ventricles, and about ʒij at base of skull.

J. R., 54. Brain slightly extra-vascular. Sub-arachnoid effusion and a little fluid in ventricles and at base of brain.

W. J., 52. Ventricles contain moderate quantity of serum.

—, 55. No fluid in ventricles. This was a case of drowning, and in another female, aged 30, also drowned, there was no fluid in ventricles, and in both the brain-substance was natural, with fluid blood in sinuses.

—, 60. Brain-substance natural. Ventricles distended with serum, and about ʒij at base of skull.

It has been said that accumulation of fluid in the ventricles causes coma, whilst sub-arachnoid effusion has no such tendency, but in chronic hydrocephalus there may be serum in the ventricles from the commencement, with persistent absence of comatose symptoms. I have, moreover, met with cases of coma, not dependent on cerebral hemorrhage or uræmia, in which there was no fluid in the ventricles, and in the cases of drowning above cited it was absent. In cases of sudden death it may be or not present; and in many

cases, even of uræmia (or chronic Bright's disease), there may be neither serum in the skull nor any trace of anasarca. Whether death be due in these to urea or spanæmia (whether the blood be toxic or defective, or suffer from a combination of evils), it is at any rate from alteration of the *quality* of the blood, not from serous effusion or pressure.

Nevertheless, on consulting many eminent authorities on the subject of serous effusion, I was surprised to discover that they greatly accorded, and confirmed my own previously-espoused views, which I imagine, notwithstanding, to be at variance with widely-accredited doctrines. Abercrombie thought it was in the highest degree improbable that serous effusion should occur in the brain as a primary disease and accumulate with such rapidity as to produce the symptoms of an apoplectic attack. He observed that the quantity of fluid effused bears no proportion to the degree of the apoplectic symptoms. It is found in small quantity, though the apoplectic symptoms have been very strongly marked and long continued; it is found in large quantity where the symptoms have been slight; and finally there may be most extensive effusion in the brain, when there have been no apoplectic symptoms at all. The direct inference he drew from these facts was that in the cases of apoplexy with effusion, the presence of the fluid cannot be considered as the cause of the apoplectic symptoms (p. 216).

Dr. Clutterbuck likewise considered that serous effusion was not the cause of the apoplectic symptoms, but that these were produced by the morbid condition of the brain on which the effusion depended.

Dr. Cooke ("Nervous Diseases," vol. I. p. 268)

thought we must admit that the serous apoplexy very seldom occurs, and adds (p. 341)—"I have never seen a case exactly answering to the description of this form of the disease." Dr. Sims ("On Serous Effusion from the Membranes and into the Ventricles of the Brain," Med. Chir. Trans. vol. XIX.) pointed out the very great frequency of collections of serous fluid in the ventricles or membranes of the brain, in cases, at all ages, where death occurs from diseases not cerebral, and where no cerebral symptoms are known to have existed. Without denying that such a disease as serous apoplexy may occur, he thought, at least, that it was extremely rare.

Andral believed it by no means well determined how far the effusion is concerned in the production of symptoms, as it often appears unattended by them, whilst, on the other hand, we frequently observe these symptoms in cases where, after death, there is not any effusion worth mentioning to be found either in the ventricles or elsewhere.

Dr. Todd ("Cyclo. of Anat. and Phys.," art. Nervous Centres) says it is in vain for the pathologist to attempt to form an opinion respecting the quantity of the fluid found in the cranio-spinal cavity, unless the inspection has been made at an early period after death. Practical men are too much in the habit of attributing morbid phenomena of the nervous system to the influence of the pressure of a liquid effusion upon the brain or spinal cord. Many facts tend to show that in a large proportion of cases, especially in the adult, the occurrence of an increased quantity of fluid, either around those centres or within the ventricles, is a *result*, and that probably it is a result of a *conservative kind*, con-

sequent upon a morbid change which depresses the general nutrition of these organs themselves. Dr. Hughes Bennett again notices that effusion of serum is not unfrequently found after death from apoplexy. He ascribes it to obstruction of the blood and transudation of the more fluid parts through the coats of the vessels, the same as in other dropsies. He states that it is seldom found without engorgement of the veins and sinuses, and that it may take place immediately before or even after death.

Dr. Watson imagines that a moderate quantity of serous fluid poured out rapidly during life would certainly exert a degree of pressure adequate to the production of fatal coma, though how the serum comes to be so effused, he admits, it is not easy always to say.

Dr. Burrows does not think serous effusion to be a cause of coma, but vascular congestion. Dr. Copland does not consider the serous effusion to be the cause of the apoplectic seizure, but the consequence of that state of the circulation on which the disease more immediately depends. Indeed, he is of opinion that a considerable portion of the effusion takes place just before death, or soon after life is extinct; and he remarks that in apoplexy presenting on dissection *congestion* and serous effusion, these states may be often considered rather in the light of *post-mortem* changes than the pathological states, which had existed previous to death. According to Dr. Sieveking ("Path. Anat.," p. 222), there is no doubt that occasionally the sub-arachnoid fluid is attributable to cadaveric changes; it is therefore necessary to be circumspect in at once attributing its presence to antecedent morbid action. The amount and position, and more particularly the

concomitant appearances of the pia mater and arachnoid must assist us in determining the question in the individual case.

Dr. John Reid (*loc. cit.*), who offers no speculations, observes that the condition of the brain which causes the symptoms in (so-called) serous apoplexy, has not yet been fairly elucidated.

Sauvages, exactly a century since, in his "Nosologia Methodica," although his views were, of course, not untrammelled by the speculative and defective pathology of the period, clearly expresses his belief that the presence of serum within the skull affords no precise indication of the cause of the apoplectiform symptoms. "Quòd in cadavere sinus cerebri aquâ turgidi reperiantur, non sequitur apoplexiam ab illo sero fuisse inductam; cum ubi infarctus est vasorum sanguiferorum, ibi lympha facile e vasis suis elabatur, et quo longiori post obitum tempore sit extispicium cadaveris, eo uberior est ut plurimum seri effusi copia; vidi hydrocephalos ingentes sine apoplexiâ, viderunt et alii; veruntamen hæc species est omnium funestissima, quia labem inveteratam in cerebro, ac sæpius ætatem senilem supponit, in quibus casibus facultas motrix quæ sola morbis medetur, efficaciter est effœta et languida" (p. 498).

Henceforth, I trust that not any medical practitioner, if he meet with even copious accumulation of serum in the ventricles of the brain, or at the base of the cranium, will have the temerity, in default of knowledge or history of the case, to assign death to serous effusion. Nay, *if he be* aware that comatose symptoms have pre-existed, that he will yet refrain from estimating the presence of serum—as a *primary* morbid

act or sequel of cerebral congestion—to be adequate explanation of the fatal event, without inquiry or reflection, as to the cause of the effusion, and vigilant search for morbid alterations in the brain and other organs. By strict observance of these cautions, I am persuaded that "serous apoplexy" as a primary disease would almost disappear, if it were not actually expunged, from our nosological systems.

In further proof that I stand not isolated in these views, which will, I am assured, become general with increasing knowledge, I may state that Dr. Abercrombie (p. 145) maintains that we have no certain mark upon which we can rely as indicating the presence of effusion in the brain, and I beg to requote Dr. Maclachlan, a physician, be it remembered, ripe with over twenty years' experience amongst the Chelsea pensioners, who doubtingly asks (p. 146), Is there such a disease as serous apoplexy? This inquiry is, to my mind, an emphatic declaration that Dr. Maclachlan has never been able satisfactorily to recognize intracranial effusion as a primary and efficient source of coma. Mons. Littré (*loc. cit.* prop. xvi.), in 1834, also dubiously inquired—Existe-t-il des apoplexies séreuses? a question which Cruveilhier ("Dict. de Méd. et Chir. pratiques," art. Apoplexie, prop. xvi. 1829) had uttered some years before, and was inclined to answer non-affirmatively.

Dr. Copland, in his Dictionary, does not treat of serous apoplexy under a distinct head, nor does Dr. Aitken, more recently, in his "Practice of Medicine."

Rokitanski ("Transl." vol. III. p. 404) hesitates whether such a disease as serous apoplexy really exists, and whether it can be recognized in the body by the

post-mortem appearances without reference to the symptoms attending the death of the individual.

To turn to recorded cases. Those of serous apoplexy described by Morgagni ("De Apoplexiâ serosâ," lib. I. epist. iv.) are unable to support the title, and at the commencement of his epistle he observes—"Nec vero nos ii sumus qui quotiescunque intra apoplectici calvariam aqua invenitur, continuo ab hâc ejus morbum repetendum esse, existemus." To select a few of the most prominent. A case of acute fever (febris acuta), Art. VI.; sudden death during illness, Art. IX.; man of seventy found dead, Art. XI.; fatal drunkenness (?) in a young man, Art. XVI. Not to be prolix, many of the accounts are most meagre and the inspection most unsatisfactory, being confined to notice of the serum. All the cases, I think, are referable to causes other than mere serous effusion as the proximate morbid action, and Dr. Cheyne thought ("On Apoplexy and Lethargy," p. 48) that much objection lay against the cases of Morgagni as specimens of serous apoplexy. Dr. Cheyne considered that the causes of serous apoplexy are involved in great obscurity, and of its symptoms and anatomy he knew little more than the one case afforded (XVI.), which is related in the 4th section of his work. This case may be found in Copeman's "Collection of Cases of Apoplexy" (the 97th), and appears to have been one of complicated meningitis.

Dr. Bright (vol. II.) gives a number of cases (CIII. to CXXIV.) to illustrate the pressure arising from serous effusion independent of inflammation :—

CIII. Effusion under arachnoid and into ventricles after hanging.
CIV. Cerebral congestion from suffocation.
CV. Do. do.

CVI. Serous effusion from suffocation.
CVII. Do. do.
CVIII. Cerebral congestion from suffocation.
CIX. Effusion under arachnoid and into ventricles in disease of heart.
CX. Congestion and effusion in phthisis.
CXI. Effusion under arachnoid; renal disease.
CXII. Congestion and effusion; renal disease.
CXIII. Effusion, &c. do.
CXIV. Effusion, &c. do.
CXV. Effusion, &c. do.
CXVI. Effusion, &c. do.
CXVII. Effusion, &c. do
CXVIII. Effusion in a man with tubercular lungs.
CXIX. Serous effusion under arachnoid in a case of emaciation, with vomiting and disease of supra-renal capsules.
CXX. Effusion in a hard drinker with tubercular lungs.
CXXI. Hemiplegia; old disease of brain and effusion into ventricles.
CXXII. Serous effusion in diabetes; death from gangrene of the lungs.
CXXIII. Effusion in diabetes.
CXXIV. Do.

Now these cases, which are the whole under the head noticed in Dr. Bright's work, give no support to the view of serous effusion being a primary and substantial disease. The effusion, *in all cases*, was associated with well-marked morbid processes, as with hanging, suffocation, cardiac incompetency, phthisis, *uræmia*, exhaustion, disease of the brain, and diabetes. In many of the subjects the effusion was evidently *apud* or *post mortem*, and from the symptoms it is clear that it contributed no share to the production of the fatal issue. To verify with exactness in all cases, the conditions under which collections of serum within the cranium are accumulated is, however, more difficult

than to elucidate that they are insufficient to produce comatose symptoms.

I believe that the amount of serum within the skull is usually found to be more limited, relatively as well as absolutely, in young subjects, and that the quantity seems to be almost irrespective of the direct cause of death, though perhaps influenced by mode and rapidity of dying and fulness of the vascular system. In aged persons, in whom apoplectiform attacks are most common, the presence of serum even in large (but varying) amount is the normal consequence of shrinking or condensation of the cerebral substance, or atrophy. Even in uræmia the attendance of serous effusion is not invariable, and if pre-existent, it is unconnected with the induction of unconsciousness.

It is impossible accurately to estimate, says Dr. Maclachlan, the normal amount of fluid in the ventricles and beneath the arachnoid in any one instance; it is often difficult to say that it is increased in quantity. This is more particularly the case with that contained in the sub-arachnoid space. When it exceeds three drachms in either lateral ventricle, it may be regarded as abnormally augmented. It is generally in both situations greatly increased in old age, and there can be little question that its influence in the production of apoplectic affections has frequently been over-rated (p. 96).

The senile changes in the brain, which consist in wasting or diminution in bulk of the encephalic structures, necessarily require increase of the cephalo-rachidian fluid to maintain a *plenum;* and pathologists familiar with autopsies in the old, amongst whom I may mention Rokitanski, Drs. Sieveking and Maclach-

lan, allude to the augmentation of the cerebral serum in advanced life. This is often denominated "senile hydrocephalus," an accumulation which has long existed, and to which death can in no way be ascribed. (Maclachlan.) In such cases the arachnoid, subarachnoid space, and ventricles of the brain may be distended with fluid, so as in extreme instances to attain to as much as twelve ounces in quantity. In hydrocephalic cases, in young adults, even a gallon of fluid has been collected within the cranium without supervention of coma.

Now these accumulations, as in infancy, are undoubtedly gradual, are conservative, and in nowise indispensably connected with altered pressure or apoplectiform symptoms. If coma supervene, it is frequently to be traced to uræmic intoxication; and albumen may be detected on catheterism, which should always be performed, and urea discovered in the serum from the nucha after vesication, and post-mortem in the ventricular effusion. After death, as Dr. Basham cautions us, the contents of the bladder are universally albuminous.

Should the kidneys be sound, the symptoms are not to be referred to the effused serum, or pressure, but to arrest of nutrition and of function, from softening or atrophy of the brain; or obvious explanation of death may be presented in the other cavities, *i. e.*, dependent on causes enumerated under "nervous apoplexy" and "congestion of the brain," as cardiac or other disorder.

In testimony, even in our day, that a renal causation may be readily overlooked, I may mention that I have seen a physician of long hospital experience con-

found a case of tolerably typical uræmia with local cerebral disease. It is certain, also, that some inflammatory cerebral complaints, as meningitis, simple and tubercular, have been set down, when proving fatal, to serous effusion. Thus, until within the last thirty years, tubercular meningitis in children was generally regarded as essentially a dropsical effusion within the brain, and was in consequence designated "hydrocephalic apoplexy," "water-stroke," or "acute hydrocephalus." These erroneous views are not yet universally eradicated, although, like uræmia, the disease may terminate in death without the occurrence of effusion. Many of the cases of Dr. Abercrombie, under the title of acute hydrocephalus, are evidently tubercular, as there is history of phthisis; and most are in children. This physician upheld that it was not the mere presence of a certain quantity of fluid in the brain which gives rise to the symptoms of hydrocephalus, as he had seen the disease pass through all its stages, and end fatally without any effusion (p. 144). Under the head of apoplexy Dr. Abercrombie relates only three cases of serous effusion. In Case CII. a man of eighty, the symptoms appear to have arisen from uræmia, or senile softening. In Case CIII., also uræmic or cardiac (?), it was a man of seventy; and in Case CIV., a dropsical patient aged forty-one, the attack beyond question is ascribable to renal disease. It may be noted in corroboration of these conclusions, that in each instance the coma was not profound. It were unnecessary to recount the many forms of coma in which, after death, serum may be discovered on examination of the cranium. I may, nevertheless, repeat that it is very often met with in the diseases alluded

to under nervous apoplexy, and congestion of the brain, especially in uræmia, inflammatory and chronic cerebral disease, delirium tremens, and, more or less, in most elderly subjects, and after exhausting diseases. It may be found effused at any age and under the most varied circumstances, which, I imagine, it is not requisite to consider further, as I am most concerned with its presence in the cerebral cavities of those cut off by that species of coma commonly known as "serous apoplexy." I will therefore relate a few histories in persons past the prime of life, which tend to prove how frequently an apoplectiform attack (in which no morbid appearance in the brain but intracranial serum may be revealed on inspection) depends on extensive structural changes in the kidneys. In such cases, the invasion of uræmic intoxication may be very sudden, the patient having never complained of symptoms, or at least having never brought them under medical observation.

Case 1.—An old man, with prostatic enlargement, who had been accustomed to draw off his urine once or twice daily, with an elastic catheter, was attacked by erysipelas of the leg, which confined him to bed. A considerable amount of urine dribbled away, and as attention was not drawn to the condition of the bladder, and he had in the mean time ceased to introduce the instrument, he became by degrees unconscious, which was at first referred to his recent malady. Such a case, which might lead to coma or urinary mischief, is more likely to happen in a public institution than in private practice, as amongst the better class of patients there is greater eagerness to live, and the condition of

the urinary organs, if impaired, is certain to be brought prominently under notice.

The case is alluded to, as, if proving fatal and serum were subsequently discovered in the brain, death might be directly assigned to the effusion.

Case 2.—A woman, æt. 58, who had been two or three times under treatment for sub-acute rheumatism, was brought to the infirmary in an insensible state. The pupils were dilated, insensible to light; the limbs rigid without paralysis; mouth drawn to right side. The complexion was pasty, pale; there were twitchings of the facial muscles and grinding of the teeth; slight stertor, and the lower extremities moved on pinching the skin of the leg, but tickling the soles did not produce reflex movement. There were appearances and expression about the patient as of feigning insensibility. Pulse was feeble. According to report, she had had epileptiform attacks during the day. There was no further history. There was also very slight œdema of the ankles, and the urine was found to be highly albuminous, and contained casts.

She was cupped over the loins, a blister was applied to the nucha, sinapisms to the calves, and croton oil was administered, but she died ten hours after admission.

Post-mortem thirty-three hours after death. Rigor mortis well marked. No arcus. Brain nothing marked, but substance very firm. About an ounce of serum in sub-arachnoid space. Soft commissure very noticeable. Slight opalescence of arachnoid; vessels of brain apparently not diseased. Lungs congested and emphysematous. Bronchial tubes healthy.

Heart, ten ounces; cavities contain dark and decolorized coagula in all chambers,—most right. Surface encroached upon by fatty substance. No valvular disease. No fluid in pericardium. Liver large, yellowish-brown; centre of lobules pale; periphery injected. Kidneys weigh three and a half ounces each. They are lobulated and granular on section. The capsule, on removal, brings away renal substance. Cortical substance increased; medullary, at parts, almost obliterated. They are highly granular and contracted in appearance, after the removal of the capsule. Stomach contracted. Spleen small and firm. Other viscera normal.

Case 3.—A washerwoman, æt. 64, was admitted into St. Marylebone Infirmary. She was pale, cold, haggard, and I was informed that she had had a fit a short time previously. There was sopor, but not actual coma. The pulse was 100, full, weak, compressible. Respirations 11 to 12; noisy, gasping, with loud râles. Pupils were contracted and insensible to light. Arcus was very strongly marked. Considerable œdema of feet and legs. Mouth deviated, but there was neither paralysis nor convulsion.

She was wrapped in blankets, hot bottles were applied to the feet, a blister to the nape, croton oil was given, and she was ordered beef tea at intervals.

Next morning she was completely comatose. Pupils still contracted; surface warm; mouth drawn to left side. The face was somewhat livid. The action of the heart was weak and rapid, the pulse almost imperceptible. Respirations 18. Bowels had not acted. Two pints of urine were withdrawn, which, contrary

to injunction, were thrown away. She could not swallow, and was evidently sinking. She died in the evening, twenty-two hours after admission. Autopsy thirty-six hours afterwards. No external marks. Œdema of legs and feet. Pupils moderately dilated. Rigor mortis tolerably well marked.

Head.—Scalp and calvaria natural. Dura mater injected. Some sub-arachnoid effusion, and two to three ounces of fluid at base of skull. Arachnoid partially opaque. Prominence of large veins of pia mater. Brain-substance healthy, but puncta vasculosa unusually evident and numerous. About the centre of the brain, the substance a little softer than natural. No clot, no injury to skull. Some serum in the lateral ventricles. Vessels at base extensively atheromatous.

Heart.—Sixteen ounces, surrounded by fat. Hypertrophy of left ventricle, and aortic orifice narrowed from binding down of one of the semilunar valves. Mitral valve appears healthy; but the aperture is constricted, admitting only one finger. The aorta is dilated above its valves. Valves on right side normal. A partially decolorized clot in right auricle, extending into ventricle and interlacing with its fleshy projections. Very little blood on left side of heart. Coronary arteries atheromatous. No fluid in pericardium. Lungs healthy, with tenacious yellow mucus in trachea, bronchi, and divisions. Some serous effusion into right pleura; no adhesions. Left lung slightly adherent to costal pleura by recent exudation; and effusion into chest, on left side, of some ounces of turbid serum, holding in suspension flocculi of lymph. Liver healthy but pale. Spleen small, firm. Stomach contains some ounces of greenish fluid and much mucus, without

peculiar smell. Internal surface healthy. Gall-bladder distended with dark oily bile. Right kidney three ounces; small, hard, shrunken, granular, lobulated, and pale. The capsule separates with difficulty. Left kidney a little over two ounces, with the same characters to greater extent. Much fat around viscera. No fluid in peritoneum.

Case 4.—J. C——, æt. 79, died on February 15th. She was taken with a fit about 1 a.m.; apparently uræmic, and died in about four hours.

Post-mortem three days afterwards. Weather intensely cold. Face placid, pale. No external marks. Body tolerably stout. Pupils moderately dilated.

Head.—Dura mater adherent to calvaria. Some congestion of surface of brain, and some fluid between arachnoid and pia mater; the former opalescent at spots. Brain-substance very firm. Ice (frozen serum) in ventricles slightly sanguinolent. Much fluid at base of brain. Extensive atheroma of cerebral arteries, but no calcification detected. Dark clots in lateral sinuses.

Heart.—No valvular disease, and it appears to be perfectly healthy in size, neither hypertrophous nor dilated, although the lungs are extensively emphysematous, especially along the borders, and at bases in contact with the diaphragm, some of the sacculi being as large as a damson. Coronary arteries of heart atheromatous. Left cavities filled with dark coagulated blood. Slight amount on right side. A little fluid in pericardium.

Bronchi and divisions give evidence of chronic bronchitis, containing muco-purulent secretion. Stomach empty, contracted, pale. Kidneys large, pale, granu-

lar, and adherent to capsule. Uterus very hard. Other viscera normal.

Case 5.—S. K——, æt. 78, died on May 29th, with marked symptoms of uræmia and highly albuminous urine.

Post-mortem two days subsequently. Weather fine. Look placid, lividity of nails, much sub-cutaneous fat. Pupils moderate. Arcus well marked. Slight œdema of feet and legs. Rigor mortis well marked. Calvaria tolerably thick. Dura mater rather injected. Arachnoid opaque, and much serum between it and pia mater; the latter rather congested posteriorly. Brain thirty-nine ounces, rather less firm than natural. Puncta vasculosa numerous. Effusion of serum into ventricles and at base of brain. Dark semi-fluid blood in sinuses. Vessels at base atheromatous.

Amount of serum in cranium from three to four ounces.

Chest.—Lungs mottled-greyish, emphysematous, imperfectly crepitant anteriorly, and of coriaceous firmness. No fluid in pleuræ. Slight adhesions on right side. No tubercle. Bronchi dusky red, and contain some frothy mucus. Pericardium not adherent, contains an ounce of serum. Heart ten and a half ounces, large and very flaccid, containing much dark clot in right auricle, a little dark blood in other cavities. Orifices appear natural. There is a valvular opening in the foramen ovale, sufficient to admit the tip of the little finger. All the valves healthy. Muscular walls of ventricle are much encroached upon by fat, and the colour is pale, almost straw-coloured. Substance is very friable, and exhibits fatty degene-

ration under microscope. Coronary arteries are cretified and atheromatous, as is also the aorta. Stomach dilated, contains much mucus, its lining pale. Pancreas congested. Spleen three ounces, very diffluent and pale.

Right kidney, weight three and a half ounces; left, three ounces three drachms. They are rather small, pale in colour, firm and markedly granular on separation of the capsule, removal of which detaches the subjacent tissue. The cortical portion in some parts is not easily distinguishable from the medullary; the latter better marked and more natural in certain places. Microscope reveals fibro-plastic granular and fatty matter, but no casts.

Liver thirty-five ounces, friable, rather small; the centre of the lobules dark, the margin pale. Bile light-coloured and small in quantity. Intestines appear healthy. Uterus one ounce. Fallopian tubes seem impervious. Other viscera present nothing marked.

Case 6.—I relate the following case in this place as it is one of much interest, and Dr. Richardson records one, not unlike it, under "uræmic coma," which appears to have been benefitted by blood-letting. F. S——, æt. 58, pale and sallow. Has been a painter, but for the last three years in declining health, and very subject to gout and asthma, the latter coming on occasionally in most distressing and threatening paroxysms. States that he has never had colic or wrist-drop or other effects of lead-poisoning. Has slight arcus on upper half of cornea. Was intemperate in early life.

At the commencement of January, 1856, he was

attacked with vomiting, without obvious cause, which was not much influenced by remedies. He had no head-symptoms at this time. On January 12th he was suddenly seized with a fit, which, from the description, was of epileptiform character, being attended with complete unconsciousness, eyes turned upwards, general rigidity of the muscles, and foam at the mouth. Three or four days before this fit, convulsive twitches of the face and arms were noticed by the nurse to come on occasionally (three or four times in an hour). After the fit he was in a lethargic state; his speech was less distinct, and there was considerable mental hebetude. The pupils acted tolerably well. His urine was of low specific gravity, clear, straw-coloured, and albuminous. He was ordered compound jalap powder and acetate of ammonia mixture.

Up to January 17th he remained much in the same state, but had one or two fits daily of a similar kind. On the 17th, about six a.m., he was seized with violent convulsive or choreal twitchings of the muscles of the body generally, especially of the muscles of the face and of the upper extremities. The facial muscles were in constant movement, so as to produce a painfully ludicrous effect. He did not appear much distressed by these twitchings, and stated that he felt no pain. They did not succeed a fit, but came on suddenly without loss of consciousness. The tongue participated in the movement. He could protrude it when desired; it was brownish, furred, dry, similarly agitated to the muscles of the body, and did not deviate. Respirations 32, shallow, gasping, chiefly abdominal. The expression was somewhat anxious, but not so greatly as might be inferred. Conscious-

ness was much impaired, but some replies were more rational than others. Common sensibility was also impaired, he did not, or did not appear to feel smart pinching. Tickling the soles excited but little movement. He could rise in the bed when his legs were examined, which were not œdematous. There was no paralysis or rigidity. Pulse 96, feeble. He had not, and had never complained of, pain in the head. When asked, he stated indistinctly that the twitchings did not hurt him, in fact, that he did not feel them.

On the 18th he was seized with a fit during my visit. His head was suddenly drawn to the left, the movements increased in severity and rapidity, and general contraction of the face and trunk ensued. The muscles of the body became tonically contracted, so that he did not move in the bed, but was tetanically extended, the legs and arms likewise becoming rigid. The eyes were turned upwards; the face became of a dusky, pallid, death-like hue, not flushed. The tongue was not protruded nor the jaws ground together, but some foam escaped from the mouth. There was no stertor. The attack lasted about five minutes, the muscles relaxed, and coma and stertor remained, with return of the twitchings and general flaccidity of the limbs.

A few hours after I ordered him croton oil and a blister to the nape.

January 19th.—No more fits. The medicine has acted thrice. Has had no sleep, and the twitchings have continued, being at the present time stronger than yesterday, but confined to the face and upper extremities. His appearance much resembles that of a person recently dead subjected to galvanism. An attack of asthma has come on, and without aid he had

got up and sat on the side of the bed in his shirt, breathing 62 per minute. The surface was covered with perspiration. He answered "no" to all questions, and consciousness was considerably impaired.

He had no dysphagia, and had taken bread and milk, beef tea, eggs, &c.

He swallowed an ether draught, and soon afterwards moved voluntarily into bed. At 12½ p.m. I was summoned, and found him just dead. The twitchings ceased about a quarter of an hour before death, and he sank gradually, without convulsion.

Post-mortem, twenty-one hours after death. Weather warm. Face pale, placid; pupils not so dilated as immediately after death. Rigor mortis well marked. No œdema. Muscles of good colour.

Head.—Calvaria moderately thick. Dura mater very adherent to the skull and removed with it, but detachable by force. Lateral sinuses contained partly decolorized firm clots. About an ounce of reddish serum at base of skull. Vessels extensively atheromatous. Considerable sub-arachnoid effusion, so as to lift the membrane from the surface of the hemisphere. Arachnoid greatly opalescent, especially over the anterior lobes of brain. Vessels of pia mater not much congested. Brain-substance very firm, and very vascular on section, showing numerous bloody points, and the whole of the white substance had a slight but distinct pinkiness of hue. Ventricles contained but slight quantity of fluid, about a drachm in each. No abnormal appearance about thalamus, corpora striata, or other parts of brain. Brain weighed, after having been drained of serum, fifty-two ounces.

Chest.—Heart fourteen and a half ounces. Pericar-

dium contained about an ounce of serum. A large, partly decolorized clot in right auricle; small fibrinous clot in right ventricle, extending up the pulmonary artery. Left auricle contained black clots extending into pulmonary veins, and the left ventricle some dark grumous blood. No disease of valves or orifices, but left ventricle hypertrophied. Right ventricle also thicker than natural. The cavities do not appear much dilated. Muscular substance appears good to the eye. Walls firm. Extensive atheromatous deposit on inner aspect of aorta. Lungs extensively emphysematous and much congested posteriorly. No adhesions, no fluid in pleuræ, no tubercle. Bronchial tubes intensely injected, of a dusky hue, the duskiness becoming gradually paler towards the upper part of the trachea, where the mucous surface is quite pale. Stomach contained bilious-looking mucous secretion. Spleen firm.

Kidneys small and shrivelled, each weighed two and three-quarter ounces; they were very similar in appearance as well as in weight. The capsules did not separate easily, but brought off the renal substance in spots. The exposed surface of the kidney was largely granular, whitish-yellow in colour, and exhibiting very distinct mottling of the organ. The feel was very firm, and section gave a very distinct sense of induration of the tissue. The cortical substance had almost disappeared, and the medullary was much paler than natural. The liver pale, with fawn-coloured patches on its surface, extending for about half an inch into its substance.

The entire colon was contracted, being hardly of the calibre of the small intestine. It presented no other

unusual appearance, and the remaining viscera seemed healthy.

In recapitulation. It is admitted by every writer who acknowledges a plurality of apoplexies, and refers to frequency, that hemorrhage into the brain is by far the most common cause of apoplectiform attacks. If to this cause be added uræmia, and those cases in which, on strict inquiry and examination, obvious explanation of death is afforded—as ramollissement, narcotism and other toxæmiæ, tumours, embolism, aneurism, &c.; the remainder will be very limited, in which we have to interpret the fatal event by reference to "nervous apoplexy," "vascular congestion," or "serous effusion," and accompanying (hypothetical) pressure. These are the refuge of ignorance, and made available when the cause of death (in the brain) appears otherwise inexplicable. I ask, is the autopsy always completely performed, and are all the viscera and the vascular system thoroughly examined in these cases? Have they not become much more rarely recorded, with advancing knowledge of renal diseases, more accurate pathology and acquaintance with the phenomena of embolism, cardiac affections, and various blood disorders?

It is indisputable that (so-called) nervous apoplexy *may* depend on uræmia without effusion, on fibrinous occlusion of the cerebral arteries, and on undetected narcotism; not to enumerate cardiac and other diseases.

To accept the coma as symptomatic, is to recognize undeniable facts; to regard it as a primary idiopathic affection leaving no trace of its action, is a baseless conjecture. In these *nervous* cases, we can only allow

that there is no anatomical evidence that the brain is the *origin* of the coma. The attack is not to be imputed to an intangible, inconceivable neurosis, but rather to disease of other organs, which should be carefully investigated, and, if necessary, analyzed.

As to congestion and effusion, they are often present without antecedent symptoms of brain affection, and often absent after profound and protracted coma. Many hold that congestion tends to disappear after death, as Hope, Bennett, and Burrows, whilst Dr. Copland, on the contrary, thinks congestion partakes much of the nature of post-mortem changes. The appearance of congestion seems to be much regulated by the mode of death, properties and stasis of the blood, and many causes not satisfactorily settled. It is certain that symptoms almost identical may, in one instance, be observed with congestion; in another, with effusion; in another, with normal appearance of the brain—a weighty token that these appearances are accidental and extraneous.

The vascular condition of the brain varies much during sleep and wakefulness, during rest and exertion, and during intellectual repose and labour. Mr. Durham ("Guy's Hospital Reports," 1860) has demonstrated, in opposition to Dr. Cappie ("Essays on Medical Science," 1859), that venous pressure is not the cause of sleep. On the contrary, there is diminished quantity and velocity of blood in the brain.

Serous effusion, in senile and infantile hydrocephalus, may persist without interfering with consciousness. By most reflective men it is regarded as the *result*, frequently post-mortem, of other operations and not directly connected or associated with coma, as it may

be absent after death from coma, even in that class of cases wherein effusion is more commonly detected; and it may be presented in large quantity where consciousness remains until the last moment of life. On the whole, effusion appears to be more indicative of secondary transudation than of primary cerebral dropsy causing coma.

The pathological facts divulged by cerebral diseases on post-mortem examination, conjoined with their symptoms and phenomena during life, afford an irresistible demonstration that pressure is absent in a vast number of comatose affections. For example, in the toxæmiæ of scarlatina, renal disease, narcosis, alcoholism, fever, gout, and rheumatism, no appreciable change may be observed after death, and no proof can be adduced that pressure on the brain was exerted during life. Although the nervous centres present no lesion, or evidence sufficient to account for death, it cannot be with propriety referred to a primary neurosis of the brain, or simple apoplexy, but to a profound, probably structural, nervous change, though it may be inappreciable to ordinary methods of investigation. Further, in presence of pressure, symptoms of unconsciousness may be wanting, as in cases of intracranial tumour and aneurism, and even in *sudden* depression of the cranial vault. Again, limited extravasation into the substance of the brain, subsequently demonstrable, when death occurs at a more or less remote period, may produce a merely temporary unconsciousness (? shock), although the source of pressure cannot be removed until a more extended lapse of time. In ramollissement, I am not aware that the coma which may ensue is declared to be the result of pressure;

and in cases of embolia, the insensibility is commonly referred to interference with the circulation.

Do not these seeming inconsistencies admit of solution by nutritional considerations, by impairment, perversion, and deficiency of the blood; by the various faulty, diseased, specifically altered, and poisoned states of this fluid, from morbid causes already categorically described?

At any rate, the most prejudiced supporter of vascular pressure cannot deny that his doctrines are *at the least* equivocal, and the feeble averment that turgidity of the vessels sufficient to produce, may disappear after, death, does not contribute to allay the scruples of the unbiased investigator, who is, in justice, desirous to be informed of the *primum mobile* which can initiate and disperse a so conveniently evanescent congestion.

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