

Dr. Ken

1857.

William James Cumming

A very complete review of the subject, that

thereof.

1.

Morbid Anatomy

On the ~~etiology~~ and causes of apoplexy.

The correct application of the term Apoplexy is to the more or less sudden occurrence of a comatose condition dependent upon certain morbid states of the cerebrum not arising from mechanical injury. Its use however has not been always restricted to such cases, since by some the coma supervening upon Bright's disease has been so designated. The etymology of the term would guide us to consider as typical form of the disease that in which the fit is sudden or instantaneous; and in this respect markedly different from the gradual induction of coma in uraemic poisoning.

Physiological experiments show us that the condition essential to the production of such speedy phenomena is the sudden application of compressing force to the nervous centres or some part of them; the removal of which is succeeded by the speedy disappearance of the symptoms. And post mortem in-

investigation proves that the cause of these symptoms is rupture of some of the cerebral vessels followed by rapid extravasation of blood and compression of neighbouring textures.

The mere consideration of the peculiarities of the cerebral vascular system might lead us to suppose that the cause of the proclivity to such cerebral lesions was not far to seek. In the brain the vessels, instead of being as in other parts of the body surrounded by textures capable of affording them support and preventing their undue dilatation, are distributed through a substance whose softness and delicacy presents exactly the reverse conditions. The arterial and venous coats are here much thinner than in any other part of the body; the middle coat of the arteries does not exceed half its ordinary thickness, while such is the thinness of the external coat (elsewhere so unyielding) that by many its existence is seriously disavowed.

Other organs (than the cerebrum) moreover have a much greater intermixture of structures not essential to the exercise of their peculiar functions in order to afford them that amount of protection and capability ~~of~~ resisting external injuries which is supplied to the cerebrum by the presence of the bony cranium.

And therefore, while in other parts of the body we find the vessels receiving strong investing sheaths of fibrous and adipose tissue, in the brain the vessels are totally denuded of any such adventitious covering. Lastly, from the proximity of the cerebrum to the central organ of the circulation the danger of over-distention and rupture of its vessels from unusually violent action of the heart might be considered serious and imminent.

Exclusive consideration of these facts might make it appear that much less than a very advanced degree of morbid change in the vascular system itself was requi-

site to prove a serious source of danger. But it is also to be remembered that much of the danger of rupture is obviated by the ~~danger~~ existence of an undistensible carotid canal, by the remarkable tortuosity of the large arteries, the wide anastomoses of the circle of Willis, and the exceedingly minute subdivisions of the vessels on the pia mater before entering the brain substance. These facts, however, appear to me to be entirely of secondary importance to the proved physiological law that the cranium always contains the same amount of fluid, with the trivial exception that, by the displacement of a small amount of cerebro-spinal fluid, a ^{very} small increase of space may be obtained for the accommodation of the cerebral mass. The general mass of the cerebrum is not therefore liable to universal vascular engorgement and the peculiar ~~various~~ tortuosity of the larger ves-

sels simply subserve the purpose of breaking and diffusing the impulse of the blood sent from the heart. In conditions of health very powerful pulsations of the carotids are unattended with risk.

In considering the relations of Apoplexy to other diseases I shall first revert to that which was once believed to be the most common and direct cause of the apoplectic seizure - namely Hypertrophy of the heart. Dr Hope informs us that apoplexy supervening upon this condition is one of the best established facts of Modern Pathology. This includes the great majority of cases of hypertrophy of the left ventricle, that condition being rare in the right ^{ventricle}, which is much more liable to dilatation than hypertrophy. From observing the increased amount of muscular fibre and propelling power of the left chamber of the heart, it was supposed that the ar-

terial blood was thrown with great and abnormal impetuosity into the aorta and carotid arteries and that during some paroxysm of the disease the cerebral vessels were from their delicate structure unable to bear the increased strain, and that in this manner rupture was produced in some part of their course. The mistake was the result of imperfect knowledge of the cause of the cardiac lesion to which such effects were attributed. In health the body is duly supplied with nutriment in every part by the normal action of the heart; but when obstruction to the onward flow of blood has been created, ~~the~~ the organ is excited to increased action and the physiological result of increased function is hypertrophy. The true perception of the nature of disease of the valves of the heart in causing partial impediment to the propulsion of the blood and secondarily hypertrophy of the left cham-

Contribution of paper too

ber, is sufficient to show that the violent action of the heart instead of constituting a source of danger is a measure of the amount of difficulty in propelling blood from the heart. The over-action seems to be barely sufficient for the maintenance of the circulation. In order to prove that hypertrophy can have an injurious effect we must have a case of hypertrophy pure and simple without valvular disease; and this is exceedingly rare. I believe however that there is a tendency to progressive enlargement of the heart in people who have been habituated to hard work chiefly, and that as a general rule the organ continues to increase after the frame has attained its full development. This seems to have been observed by those who have had opportunities of conducting many post mortem examinations. But a corresponding result in the increased number of cases of apoplexy

Yes

Robert Taylor

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has not I think been made out.

But so far from hypertrophy as a result of valvular disease being a cause of hemorrhagic apoplexy, I can conceive only of the opposite result being in certain cases produced, namely, such defect of vascular supply to the brain as shall end in ramollissement. †

This however will be separately considered. If ~~hyper~~ apoplexy were so direct a sequela of cardiac hypertrophy, ^{as was once believed} the morbid appearances of the disease should be constantly encountered in our Pathological theatres - how rare is the conjunction it is unnecessary for me to say.

While engaged with the consideration of this subject it will be necessary to say a few words about a supposed apoplectic diathesis or habit of body. Plethora or a condition of excess of blood in the body is maintained by some to be distinctly a cause of predisposition if not a predisposing cause of apoplexy. It has never been proved however that such a

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condition exists, ~~or that the blood~~ and the term has been vaguely applied. It coincides however with the popular opinion that individuals with large and corpulent bodies, thick and very short necks and large heads are peculiarly apt to be the victims of apoplexy. Pathology has however totally overthrown this idea: at least it has shown that quite as large a proportion of those who have a different conformation are subject to apoplectic seizures; such for instance ~~who~~ as have long and thin necks and whose aspect is anemic and ill-nourished. Those again who are corpulent are very erroneously supposed to possess an excessive amount of blood in their systems. As a general rule those who possess little or no fat have much larger internal organs and a much greater amount of blood, and are less liable to syncope. Much danger is popularly supposed to be connected with shortness of neck from the

idea that the heart's impulse would be communicated with great violence through a short arterial tube and thus exercise injurious pressure on the cerebral tissue or tend to rupture the vessels. But the provisions of nature are amply sufficient to obviate this danger. The peculiar relations of the cranium to its contents together with the other conditions previously referred to render the cerebrum incompressible by any force which the heart can exercise upon it; so that on the hypothesis of there being no disease of the vascular system, danger from such sources as those alluded to is purely imaginary.

Dr Williams has made a statement regarding the nature of epilepsy which if confirmed would establish an affinity between the paroxysms of this disease and those of apoplexy in so far as the mechanical conditions which produce the individual fits are concerned. He attributes if I mistake

not the phenomena of the disease to the pressure on the cerebrum occasioned by violent pulsation in the carotids preceding the individual fits of epilepsy. A great inaccuracy must have been committed in matter of fact in as much as the strong beating of the carotids does not come on till the paroxysm itself has subsided. It has been pointed out to me while observing an epileptic fit that the immediate effect of the convulsions is to banish the pulse; and if as if to restore the circulation remission is accompanied by increased action of the heart. After the fit a state of coma results which may be ^{viewed as} due in some slight measure to compression from increased action, but which I would prefer to attribute to excessive exhaustion resulting from the extraordinary vehemence of the nervous phenomena.

Proceeding with my intention of first considering those theories which have been advanced for the explanation of

the causes which produce apoplexy and which I consider either to have been dis-
proved by the advance of knowledge or ~~which~~
~~were insufficient to afford adequate evi-~~
~~dence of their being admitted as~~ ^{to be} inade-
quate as theories of disease, I shall
 not attempt any rigid classification ^{of}
 them, reserving this for such as I consider the
 approved and established theories.

The relation of another type of heart
 disease to apoplexy is next worthy of
 consideration. It is believed by many
 that Fatty degeneration of the heart is a
 prominent cause of apoplexy. Of this
 disease two forms have been distin-
 guished. The first is generally observed
 in individuals characterised by great obes-
 ity and consists of a deposition of fat
 around the heart in various amount,
 situated ~~one~~ between the pericardium
 and muscular substance. It is believed
 to cause absorption of the muscular
 walls, ^{by pressure} which become thin and softened
 and the energy of the heart's action

"The muscular fibres of the heart ~~says~~
or of the voluntary muscles may be
imbedded in adipose tissue and yet
may themselves be free from the least
degeneration." (Paget on Surgical Pathology)

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is thus much impaired. It is far from being proved however that only the corpulent are liable to this disease, or that they are liable to it in much greater proportion than others. But it is an undoubted fact that those who are excessively corpulent have comparatively small and feeble internal organs, and they are liable to syncopeal affections. The other and more formidable type of degeneration of the heart is a result of deterioration in the nutrition of the muscular tissue principally of the heart. It is chiefly dependent on hereditary predisposition and does not usually occur till the period of youth is passed. Microscopical research has proved it to be of gradual and progressive tendency to degeneration of the proper sarcons elements which become transformed into (or are replaced by) little oily particles which observe a similar arrangement to that of the constituents of the fibrillae

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which they replace*. As they increase they merge into larger oil-globules and thus the alteration of structure becomes as manifest as it is complete. To the naked eye there is only observable a diminution of the natural reddish colour of muscle and in place of it a pale fawn-colour is observed; the walls of the heart exhibit unnatural flaccidity, and the knife which is used to cut them will be found to be greasy.

The disease is a very insidious one and may have made fatal progress before anything wrong has been suspected: the patient being suddenly cut off by syncope during some unusual effort as a long walk or during straining at stool. The effect of exercise is to compress the arteries by muscular action and the heart is consequently excited to increased action in order to propel its contents through the system. The heart in this diseased condition is totally inadequate to the labour

* Paet

imposed upon it and the result is a total cessation of its action after a number of excited ~~to~~ contractions. Death is by syncope, beginning at the heart. It is strange that this class of disease should have been mistaken for apoplexy but patients were constantly stated to have died by apoplexy. Another common way of accounting for the result was to attribute death to spasm of the heart - a term which is merely a cloak for ignorance and not an actually existing disease. In other cases however the manifestations of the disease have been spread over a large space of time and as the disease advanced there have been dimness of vision, aching sensations in the head, vertigo, feebleness of intellect and loss of memory. A feeble condition of the circulation will probably be well marked and the first sound of the heart will be but feebly audible or as in some cases of fever not perceptible by the ear.

Yet we are enabled to draw a distinction
 between these cases once considered a-
 plectic, and those which are really
 and truly so; we are guided by the fre-
 quent recurrence of such nervous
 symptoms as those just described and
 by the fact that they are not productive
 of hemiplegia or any paralytic effects
 and Dr. Stokes observes that anti-
 phlogistic treatment, not un frequently
 incumbent in cases of real apoplexy,
 is here totally inapplicable while
 stimulation is ~~less~~ of decided benefit.
 Observation of
 The condition of the circulatory system
 taken in connection with the cerebral
 symptoms seems to me to be of the
 highest importance in forming a diagnosis.
 This class of diseases then, I consider,
 has been disproved by the advance of
 our pathological knowledge of its nature
 to be one of the conditions on which
Apoplexy depends. It ought to be
 classified with diseases essentially
cardiac.

17.

Serous apoplexy. Richoux was the first to describe what he considered a form of apoplexy which, from its supposed characters he termed the serous. This view became generally prevalent and even yet holds its place in the minds of many. It is in effect - that by excited secretion of the lining membranes serum may be acutely effused into the cavities of the ventricles of the brain or that of the arachnoid, and that ~~by~~ its presence, by exercising pressure on the cerebral mass, causes speedy coma and death. The observation of fluid in these situations led Richoux and the fact that the patients had died comatose led Richoux to institute a distinction between cases of apoplexy which were attended by cerebral hemorrhage (a form of the disease to which he believed the robust and full-blooded to be peculiarly obnoxious); and that form which he termed the serous and described as being often attended with some degree of general anasarca,

and a disease to which, not the robust, but the leucophlegmatic had a more than casual proclivity. Dr. Abercrombie acquiescing in its being ranked as a species of apoplexy believed it to result from loss of balance between the arterial and venous circulation in the cranium.

He considered the apoplectic symptoms to be the result of effusion and compression. Pathologists of the present day have however discarded the opinions of Rochoux. They hold that the apoplectic effusion, far from being the cause of death is innocuous in itself, of old standing and gradual accumulation; and they believe that the fatal event is produced by uræmic poisoning, coma from which is sometimes known to supervene with great rapidity. At the time when the belief in serous apoplexy was in the ascendant the whole of the Pathology of Bright's Disease was undiscovered, and no theory was advanced to explain the occurrence of

of head symptoms but such as would account for pressure of some kind on the Brain. If we were to look for the evidence of this effusion being rapidly poured out into the ventricles, or sub-arachnoid space, or both, we should expect to discover very of decided flattening of the convolutions - but this we never find. It appears to me that if the effusion from the serous membranes were sudden and copious, that the serum would be clear and watery. But on consulting Wood's Practice of Medicine I find that after stating that the fluid is rapidly effused and without any inflammatory action, ~~that~~ the damaging statement is made, that the fluid is sometimes colourless but often turbid and of a dirty yellow hue - in reality evidences of its being of old standing. But on the other hand, the observations of Sims have proved that the cerebral serous effusion is a direct consequence of slow shrinking of the Brain, the serum being thrown out in

exact proportion to the diminution of the volume of the cerebral mass and with the purpose of ~~not~~ occupying the space relinquished by it so as to keep the contents of the cranium at all times equal. In Wood's "Practice" this extraordinary amount of supposed effusion is said ~~to be~~ not to be marked by any evidence of congestion. It is not easy, ~~to~~ ^{yet}, to account for such rapid exudation without "any evidence of congestion." What has been already stated however regarding our knowledge of Bright's disease is sufficient to overthrow the beliefs in serous apoplexy. All the symptoms of these cases are what we now recognise as symptoms of Bright's disease and the coma we explain as the result of non-elimination by the kidneys of urea, leading to the presence of that deleterious ingredient in the blood. The transformation of this substance into carbonate of ammonia, in other words its decomposition in the system, is

now supposed to be the immediate cause of the induction of coma. The presence of urea in the blood seems to prove fatal with different degrees of rapidity and ~~it~~ it seems that the transformation of urea occurs much more readily in some than in others and is regulated by laws which are not yet understood.

Apoplexy as a result of acute Pericarditis. The earliest recorded case is that of Mr. Stanley in 1817. In 1821 a case in every point similar was communicated by Dr. Abercrombie to the Medico-Chirurgical Society of Edinburgh regarding which he remarks that "this dangerous affection may be going on rapidly yet insidiously while our attention is occupied by symptoms which have no relation to it." Dr. Latham next called attention to the deceptive nature of the cerebral symptoms in drawing off the suspicions of the practitioner from the real seat of disease. After attention had been so prominently

directly to this affection recorded instances of it rapidly multiplied. Dr. Hope was the first to dissent from this opinion and states that he had met with no such case in his whole experience. Recently special and extensive inquiries have been made regarding this subject by Dr. Taylor. The cerebral symptoms which he observed were, besides epileptic convulsions, frequent cramps in the legs and arms, confusion of mind and lastly stupor ending in fatal coma. The views of Dr. Taylor are quite in accordance with the most advanced Pathology of our own day and he has succeeded I think in proving very successfully that the Pericarditis and cerebral symptoms merging in coma are not related as antecedent and consequent but that the common source of both is renal disease and its necessary accompaniments. He has observed that the cramps and occasional loss of memory precede the

Pericarditis, which, it had been assumed by previous authors, its presence had given rise to. He also states that were it not for the undue prominence which has been given to the fact of there being Pericarditis in these cases, those who have recorded them would have assigned their origin to the Bright's disease which doubtless existed in all of them. Dr. Taylor next endeavours to show that the mass of cases of non-rheumatic Pericarditis is due to renal disorder. Without accepting this as thoroughly proved I have no hesitation in admitting that a very large proportion of them are. Pericarditis is a disease more apt than any other to be overlooked and closer observation and the employment of more accurate statistics might lead us to views ~~as~~ widely differing from those of Dr. Taylor. For example the frequency of the concurrence of pericarditis with pneumonia and with pleurisy by

the extension of the inflammatory process from one sero-fibrous tissue to another does not appear to me to ^{have been} ~~be~~ duly acknowledged hitherto. In a minor form, ^{pericarditis} ~~it~~ is very frequently present in the course of continued fevers. When acute and extensive the diagnosis between it and continued fever is often extremely uncertain, but if no renal complication co-exist death will not take place in the way of coma, but from syncope. Dr. Taylor, lastly, states his opinion that the majority of cases of non-rheumatic pericarditis are quite latent and that the mortality is much greater than among the cases of ~~non~~ rheumatic pericarditis. In commenting on this I do not think it proper thus to compare truly inflammatory pericarditis, ^(which the rheumatic is) with that form which accompanies renal affection. The risk in the latter is not nearly so great as this method of viewing it ~~not~~ would indicate. Renal pericarditis is more

properly hydropericardium than inflammation of a serous membrane and the risk is not so much the risk of pericarditis as of uraemic poisoning the concomitant of Bright's disease.

The extraordinary disturbance in the nervous system produced by acute or Rheumatic pericarditis does not at all entitle us to rank it as one of the diseases which ~~to~~ tend to the induction of apoplectic coma and the tendency to death is markedly and evidently by syncope. In the wider acceptance of the term 'Apoplexy', however, room may be found to admit the pericarditis consequent on renal disorder as forming along with that disorder one of the most frequent and deadly ~~causes of pericarditis~~ ~~precursors of apoplexy~~. But the strict and proper application of the term apoplexy excludes these (together with the conditions we have already reviewed) as leading to the true apoplectic seizure.

(Among others Dr. Williams)

Insolation. The results of undue exposure of the head to the sun's rays - insolation or "sun-stroke" - have been by many* supposed to be apoplectic. In support of this view he adverts to the fact that after death the vessels of the pia mater are usually found gorged with blood. This however is nothing but the condition of health and may be observed in many persons who have died suddenly, not having previously been the subjects of exhausting disease. But the phenomena of insolation do not present the characteristics of apoplexy so much as of death resulting from powerful effects produced dynamically on the nervous centres, death by shock in the way of syncope. The prostration of the whole nervous system and subserviently of the vascular and muscular is sudden and complete, whereas in apoplexy the pulse generally remains full and strong. But the weightiest argument adduced against the na-

ture of the disease being apoplecticiform is that advanced by J. Watson, namely, that "while the most approved remedies of apoplexy prove here unavailing, the largest amount of success attends the employment of means of an opposite tendency"— the cold douche to the head and internal administration of brandy and water.

We come now to consider those causes and conditions which have been assigned as those out of ^{which} apoplexy springs and which the present state of our knowledge entitles us to regard as the just pathological relations of that disease or rather ^{that} class of symptoms. In order to commence this subject properly it will be necessary to inquire into the peculiarities of the cerebral circulation, a theme which has been productive of so much discussion and which seems still to be open to investi-

gation.

The first whose attention was drawn to this inquiry was Monro Secundus and he first propounded the theory of the cerebral circulation. His views were tested experimentally by Dr. Keilic of Leith with the result of confirming the conclusions at which ~~Monro~~ ^{he} had arrived.

Dr. Abercrombie subsequently adopted and ably maintained the same views.

They are, as expressed by Dr. John Reid, these: "that the cranium forms a spherical bony case, capable of resisting the atmospheric pressure, as the only openings into it are the different foramina, through which the vessels, nerves and spinal cord pass; that the encephalon and its membranes, the blood contained within the vessels, the serous fluid secreted from the inner surface of the arachnoid, ~~and~~ ^{pia mater} and a small part of the cerebro-spinal fluid placed between the outer surface ~~and~~ of the arachnoid and the pia mater, fill up completely the interior

of the cranium." Since the cranium resists atmospheric pressure, no blood will leave the veins unless an equal amount be passed in by the arteries; for the pressure of the atmosphere upon the soft parts which cover the veins would effectually prevent its escape. The accuracy of these observations has been doubted and the experiments of Dr. Burrows were believed by many to have overthrown the theory of mowrs. The objections of Dr. Burrows have however been ably refuted by Dr. John Reid and I will here as briefly as possible refer to them.

Dr. Burrows objects that "in children with open fontanelles and in adults who have lost part of the bones of the cranium, we have no particular disturbance of the functions of the ~~cranium~~ brain from this gap in the walls of the imaginary sphere." In these circumstances however disturbance of the cerebral circulation could not be expected even by those who uphold the theory. Another principal ob-

jection is that those who hold the theory of Mowbray believe that "when hemorrhage takes place from the general system it does not affect the quantity of blood in the brain." The actual statement of Dr. Kellie was however that "if 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 plentitude of the cranium". "The proposition, says Dr. Reid, to have been correctly stated should have been in this form: "That when haemorrhage takes place from the general system, it does not affect the quantity of fluids within the cranium."

Dr. Reid has ably shown the fallacy of drawing inferences from such experiments as suspending animals by the heels immediately after death. The usual appearances moreover of the brain after death by hanging strongly militate against the views of Dr. Burrows and he has entirely

failed in attempting satisfactorily to account for them. ~~By admitting~~ Admitting that the majority of instances support the theory of Mours he is obliged to cite special cases of sanguineous apoplexy which occurred during hanging. Dr. Reid however replies that in these cases the vessels are prematurely weak and have given way; for as it is more difficult to obstruct the passage of blood along the arteries than along the veins, and as it has been proved by experiment that in asphyxia, as the blood passing along the arteries becomes more venous, there is an increased pressure upon their inner surface, if there be any tendency to sanguineous apoplexy, it is apt to occur at this particular time. There is no doubt that the cerebro-spinal fluid does pass between the spinal canal and cranial cavity; but the doctrine of unvarying quantity of fluid within the cranium is not overthrown by this. The quantity of the fluid is very small and the amount that might be displaced very trifling.

There seems therefore to be no solid reason for departing from the original theory, which was ~~on~~ based on such safe experiments and which has emerged so well from the keenest discussion.

Its bearing on the question of apoplexy is important and also on other cerebral disorders.

It seems to me that in a pathological disquisition the most convenient mode of arranging the ^{causes originating the} different forms of apoplexy will be to classify them according to their effects, their morbid anatomy.

We shall consider first those causes and conditions which tend to the production of simple apoplexy; second, those which lead to hemorrhagic forms; and lastly those which lead to apoplexy by defective nutrition of the cerebrum, to atrophic forms.

The scope of this essay will not include descriptions of the local lesions in apoplexy, with their results (as hemiplegia &c) but will be confined to the conditions on which apoplexy depends, from which it originates.

I. Causes which tend to the induction of Simple Apoplexy. These ~~are~~ Dr. Abercrombie was the first to describe a kind of apoplexy which leaves no appreciable cerebral lesion, and to this he gave the name of simple apoplexy. He attributes its occurrence to a loss of balance between the arterial and venous circulation in the brain. Now that such injury should be wrought in the brain as to cause speedy prostration ending in death and yet that no trace of injury should be perceptible on post mortem examination seems very extraordinary and equally unsatisfactory. The light thrown upon the sudden supervention of coma by the recent advances of Pathology and the discovery of the frequency of uraemic poisoning in connection with Bright's disease of the kidneys impress us with the belief that a very large proportion of Dr. Abercrombie's cases of death by simple apoplexy were in fact cases of death by uraemic poisoning. It can moreover be easily

imagined that less skilful practitioners would in cases of doubt and perplexity find this an admirable resort for satisfying their own minds and those of others; for the disease of the kidney not being then understood would not be ^{looked for or} detected, and the brain would be found to present its normal appearance; nothing else would be found wrong: ergo, the case was one of simple apoplexy. Whenever a thorough post-mortem examination was not instituted the liability to error would be greatly augmented. If the physician ~~only~~ ^{only} suspected ^{only} simple apoplexy he would only examine the head and he would be sure to be satisfied with the condition of the brain as confirming his diagnosis, whereas the fatal event might have been caused by the rupture of intra-thoracic aneurism or some other unsuspected disease. I do not however mean to express my disbelief in cases of simple apoplexy, and I consider that the re-

cent researches of Dr. Marshall Hall tend to put the subject in a very clear light and to account satisfactorily for the morbid effects. Dr. Hall holds, and I think none can dissent, that there is no danger, no tendency to morbid action as long as whatever may be the violence of the arterial circulation, as long as there is no impediment to the return of blood along the veins; & that the idea of a determination of blood to the head is quite erroneous and untenable. It would be very difficult to prove why ^{such} ~~this~~ vis a tergo should exist and what should occasion it: yet the belief in determination of blood to different sites maintained a long and absolute sway over the minds of medical men. Dr. Hall attributes the real state of things to impeded venous return, and this he has discovered to be induced by a spasmodic action of the muscles of the neck on the vein of that region, — an action evident in a vast many instances, though latent per-

The vehement pulsations in the arteries are only the result of the obstruction to the circulation, which the heart labours to overcome and the lividity and congestion that exist are evidences against the idea of 'determination.'

haps, and to be inferred from the similarity of its effects in others. This spasmodic condition he has designated 'Trachelismus.' It is frequently to be felt when it is not to be seen, on applying the finger." "It is ~~is~~ still more frequently to be traced and inferred by observing the lividity and tumidity of the integuments of the face and neck." Here then are some of the effects of a great constitutional condition, which I do not doubt to exist and in most cases to dominate the production of the apoplectic fit. The trachelismus is its result or paroxysmal manifestation; the result of the trachelismus is the induction of apoplectiform phenomena. I am by no means satisfied with Dr Marshall Hall's explanation of the cause of the trachelismus, which is nothing more than mental emotion and gastric irritation. I think that this is very inadequate, and though it may suffice to produce the paroxysm where the constitutional tendency exists, yet

to establish its truth it ought to be shown that a majority or at least a large proportion of cases of gastric irritation and mental emotion suffice to produce tracheismus with its dire results. To this constitutional condition reference will again be made. The form of apoplexy ~~now~~ at present under our notice I have called 'simple', because I see no reason why the term used by Dr. Abercrombie should be departed from; yet I do not give the same explanation for its occurrence. Instead of attributing it to loss of balance in the circulation, I consider this a mere secondary result of the constitutional condition which produces spasm of the sternomastoid and other cervical muscles, (so much more under the influence of emotion); and I prefer to speak of stasis or retardation of cerebral circulation as more scientifically correct than 'loss of balance' which doubtless exists. These forms of Apoplexy (the 'simple') are

what are so often termed 'threatenings' and I think they ~~are~~ do not ^{often} become more than threatenings very much ~~so~~ because in these cases ~~of~~ degeneration of the vascular system does not seem to have been present or advanced far. Dr. Hall states that there is this essential difference ^{in symptoms,} between the threatenings and severer forms of apoplexy, that "the former occur and recede ~~paroxysmally~~ whilst the latter proceed insidiously, and at last there is perhaps a crushing attack of pain, of pallor and of apoplexy; or of hemiplegia" - the result of a large laceration and extravasation. Beyond this I would not venture to push definitions or distinctions. I consider the coma in cases of retardation of the circulation to be dependent on the narcotising influence of the ^{continued} presence of venous blood in the brain and possibly ~~of~~ ^{on} pressure ~~of~~ ^{of} the cerebral substance, for we know that venous blood distends the capillaries to a very considerable degree. This congestion however of one part of the

Brain is not inconsistent with the theory of
 Mowbray, for it is evident that the con-
 gestion can only be partial and less blood
 will be admitted to other parts from the
 arterial system. I repeat however that
 cases of death resulting from this simple
 apoplexy alone are extremely rare. I
 am confirmed in my opinion of the coma
 being the result chiefly of the narcotic
 effect of the venous blood by the interesting
 observation of Dr Hall and Dr W. Tyler
 Smith that during sleep the muscles
 of the neck contract and compress the
 veins of this region, thus retarding venous
 return from the head, and inducing a
 sub-apoplectic condition.

Disease of the right chamber
 of the heart has a tendency to
 produce the same effects as
 tracheal aneurysm, and these not
 infrequently result. See Disease
 of the right ventricle causes delay in
 the onward progress of the venous circu-
 lation and there is a more or less

cyanotic condition and appearance constantly maintained. If the impediment be great the lung or the base of both lungs becomes more or less congested and ultimately solidified thus increasing to a very injurious extent the free transmission of blood through these organs. This again reacts on the cerebral circulation and venous ~~to return~~ from the head is greatly retarded.

When a condition of this sort is present, a sudden paroxysm of cough by suddenly arresting entirely the venous flow may ~~terminate~~ ~~etc fatally~~ determine the fatal result. If a diseased state of the cerebral vessels coexists ~~hemorrhage~~ laceration and hemorrhage will be produced and death from this cause is probably in the circumstances inevitable. When there is engorgement of the lungs it may be sometimes impossible to tell without dissection how much of the fatal event may have been due to asphyxia.

II. The causes and conditions from which Hemorrhagic Apoplexy results.

Of these the chief have been considered hereditary predisposition and a certain age and ~~also~~ accompanying these a degenerate condition of the vascular system; also, a certain way of living, exposure to vicissitudes and the influence of sex. Dr. Watson considers the most prominent of these to be age; the period between 50 and 60. So far as the atheromatous degeneration is concerned I think that hereditary predisposition and intemperate ~~temperate~~ habits are even of more importance in the production of disease than a certain age, though I would not extend the same observation to the calcareous form of degeneration as a cause of disease. There is no doubt or dispute that the male sex is much more liable to this disease than the female. The causes of predisposition are generally held to be to disease of the vascular system of the type of fatty degenera-

tion, are generally held to be habitual intemperance, ^{in alcoholic drinks} with undue exposure to the vicissitudes of weather, bad or insubstantial or insufficient food and defective clothing. Of course when as must very often be the case these ^{conditions and} tendencies concur in the same individual with that of hereditary predisposition, the proclivity to the disease must be greatly increased; and their ~~con~~ concurrence of causes in the same person must make it difficult to judge which is the more influential cause. An important fact in this inquiry is that the cerebral tissue has a remarkable attraction for alcohol or, more correctly stated, a remarkable power of detaining it. The result of all chemical analysis is to confirm this. This seems to me intimately associated with the remarkable frequency of disease of the cerebral vascular system.

The proximate effect of the use of alcohol is to load the blood with carbonic acid, for the acid is formed as usual by

the process of vital combustion, but is prevented being interchanged with the oxygen of the air during the presence of alcohol in the blood, for the exhalation of it becomes greater after the alcohol is gone. The venosity of the blood in drunkards is remarkable and it has been found to contain excess of carbon. To this has been attributed the dark colour of the blood, which is really due to deficiency of oxygen. Such blood passes with great difficulty through the capillary vessels and hence there is extreme danger of rupture occurring from increased distension. In confirmed drunkards there is generally present a diseased and varicose state of the vessels. The liability of drunkards to falls on some part of the cranium is attended, therefore, with the most extreme danger and the coma of drunkenness may be terminated by that of apoplexy. The permanent state of the vessels in drunkards renders them liable even in the sober state to ap-

apoplexy from injuries which to the temperate would prove innocuous. On the same principle excess of sleep is justly considered to favour the accession of apoplexy in the predisposed as in this condition less carbon is exhaled as an immediate consequence of the respiratory function being in a state of abeyance; all the secretions too are less active in sleep, hence this coupled with free indulgence in the pleasures of the table has no small influence in precipitating the attack.

Atheroma is a result of changes ensuing in the tissues generally in the decline of life and to be ascribed to defect, not to disorder of the formative process. Mr. Paget thus briefly describes it "In the place of a proper substance of an elemental structure, e.g. in the place of the contents of or the nucleus of a cell, or in the very substance of a simple membrane, a blastema or a fibre, minute particles or granules are seen which are recognised as consisting of oily matter &c. Numbers of these minute particles become aggregated and form a 'granule-cell' or—as it is termed after the cell-wall has wasted and disappeared—a 'granule-mass'. These products are derived from the degeneration of intercellular substance as well, and muscular tissue seems particularly liable to it. The arterial system

is particularly liable to atheroma. The vessels are usually affected in round or oval patches, which are more or less opaque and when microscopically examined present crystals of cholesterol along with fatty and granular compound cells. Wherever this has taken the place of normal tissue there is an absence of the muscular and elastic fibres constituting the middle coat of the arterial system. The vessel may indeed be thickened by the deposit of this cheesy-looking substance, but wherever it exists contractility is lost and when as often occurs the epithelial lining becomes eroded or ulcerated from defective nutrition, the force of the contained column of blood causes the unresisting walls to pouch at the weakened parts which are only prevented from speedily rupturing by the toughness of the external fibrous sheath. The tendency to rupture is thus always present and the exciting cause all that is wanted to bring about this result. In whatever way the return of blood from the head is checked rupture is imminent. The immediate

cause is stoppage of venous return and this may be produced by trachelismus, by violent exercise especially in the stooping position, by tight application of garments round the neck, keeping the head long turned to one side and fixed in that position; the venous excitement, paroxysms of anger &c which by acting on the emotional cervical muscles of the neck induce trachelismus.

Some have viewed the affection (atheroma) as inflammation of the vascular coats. This it cannot be, for exudation from the vasa vasorum would not be seated near the inner lining of the arterial walls, because the nutrient vessels do not penetrate further than the centre of the muscular coats, and if there were exudation, it would be chiefly confined to the outer ~~and~~ side. Moreover atheroma appears in patches all along the vascular system, and symmetrically disposed on opposite sides, thus proving that it is a result of constitutional disease.

M. Rochoux has ascribed hemorrhagic apoplexy to a peculiar softening of the cerebral substance. Cruveilhier and Casswell with more reason consider it as an effect of ~~the~~ attacks accompanied with hemorrhage.

The so-called red softening is undoubtedly the result of previous extravasation of blood, the ^{elements of the} extravasated ~~having~~ blood having been in great measure reabsorbed excepting some of its colouring matter, the haematins, to which there is little doubt the red appearance is really owing. It would be difficult to account for its appearance on any other hypothesis and I am not aware of any valid objection to this one. That cerebral hemorrhage often results from the giving way of a large vessel is undoubted and very often the actual source of it can be traced. Dr. Watson somewhat irrationally talks of blood being "exhaled!" I find that in his lecture on haemoptysis the same expression occurs and he endeavours to back his statement by saying that the pulmonary surface is often found perfectly entirely from the commencement of the trachea to the remotest divisions of the bronchial tubes!

That is, he thought so: for I cannot imagine how anybody could make such a searching examination and how blood-corpuscles are to escape from vessels unless rupture of their walls has occurred. The most frequent seat of hemorrhage is that which a knowledge of the anatomical characters of the brain would indicate: in those parts which are 1st more vascular than others containing larger as well as more & numerous vessels and 2^d those in which the cerebral substance is softer and more delicate and afford less support to the vessels. These conditions are most marked in the corpus striatum and next in the optical thalamus. The frequency of hemorrhage in these situations was first noticed by Morgagni and reasoning on the facts enabled him to discover the cause, and subsequent observations have confirmed his statements. The converse of this was distinctly proved by Kochoux who found

that the white substance is least liable to become the seat of hemorrhage.

The arteries at the base of the brain are sometimes affected with aneurism, especially the basilar and internal carotid arteries. In the event of rupture the patient has not the slightest chance of recovery.

Although the brain is incompressible from any force the heart can bring into operation against it, it is far from being absolutely incompressible and whenever rupture of a vessel occurs the neighbouring textures must be to some extent compressed: when however the amount of blood effused is large, it is obvious that as the brain is not easily compressible over its general surface, the space occupied by the extravasation must be gained at the expense of the arteries and veins and hence in addition to the fearful amount of shock produced, the accessory danger ensues of the entire supply of blood to the brain being cut off.

The veins are much less liable

~~In 1850 Mr. Paget brought forward the view that in many cases of~~
 than the arteries to atheroma. I think because they have not the muscular coats of the arteries, and it seems that the muscular is the tissue most obnoxious to this disease.

When cerebral hemorrhage occurs in connexion with hypertrophy of the left ventricle which is certainly very rare it must be attributed to the coexistent diseased condition of the vessels not to the cardiac affection.

Cerebral hemorrhage may occur along with the calcareous degeneration of vessels, which become brittle and inextensible in old age, there being often bony rings formed encircling the vessels with intermediate calcifications. Excitement of the circulation, but far oftener retardation arising from tracheismus tend to rupture and apoplexy.

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The exemption of the female sex as a general rule from apoplectic disease is a most singular and interesting fact. It has been clearly proved too that this exemption is not lost in the case of such as have spent their lives in habitual intemperance, as in the case of soldiers' wives in India, where a higher allowance of pay allows them to indulge in such propensities and where every facility exists for medical officers to observe the effects in the production of disease.

The symptoms of this disease are varied and inconstant nor can we satisfactorily account for them from our pathological knowledge of the disease. They consist* in slight and often transient paralytic affections, double vision, a dropping of one eyelid, occasional inarticulate speech, weakness perhaps of a single finger: headache, giddiness, unnatural sounds in the ear, numbness or tingling in the extremities; partial and strange defects of memory, temporary confusion of thought & some impairment of intellect.

III Causes inducing Apoplexy by defective
nutrition of the Brain.

In 1850 Mr. Paget brought forward the view that in many cases of Apoplexy preceded by cerebral softening the previous condition was one of fatty degeneration of the cerebral capillaries. He describes minute particles of oil scattered over the vascular coats, the vessels being completely beset with and lost in them, in the most diseased parts.

Professor Bennett on the other hand holds

that they are the result of an exudation from the vessels, which has gone through its natural course and been developed into an immense multitude of minute cell-forms. I do not think that analogy would lead us to suppose that such prodigious masses of oil-globules would result from the degeneration of the walls of these minute vessels.

As age approaches the effects of gradual failure of the vital powers become manifest, commencing in those tex-

times whose vascularity is lowest. Hence
 the lower chemical composition of the
 matter that takes the place of the
 former normal structure. The com-
 paratively low vascularity of the walls of
 vessels renders the nutritive process tar-
 dy and as a result of this again the
~~facility for the deposition of mineral~~
~~matter is at a maximum.~~ Most fav-
 orable conditions are afforded for the
 deposition of mineral matters. Connect-
 ed with this is the slow and imperfect
 reabsorption and excretion from the sys-
 tem of ~~mineral matters~~ of the effete
 earthy matters. Thus the presence in
 the economy of abnormal quantity of
 the mineral constituents of the body
 is a measure of the deficiency in the
 nutrition and replacement of those
 constituents which acknowledge a truly
 plastic origin. In these circumstan-
 ces deposition of the phosphate and
 more especially carbonate of lime oc-
 curs in the interstices of tissue, more

especially those of low vascularity. The lowest are the extravascular. Intermediate may be considered the walls of the vessels, and hence their liability in advancing age to calcareous degeneration. Dr^r Williams states that the most complete specimens of petrification supervene upon the deposit of a plastic fibrin in lymphatic glands, on snow surfaces, in the lungs, and on and under the lining membrane of arteries; if these deposits escape fatty degeneration and softening, they are very apt to be ultimately converted into masses or plates of calculous ^{or stony} matter in which there may be little or no animal substance. He considers the presence of calcareous matter as a result of degenerating albumen exerting a chemical attraction over the phosphate or carbonate of lime in the adjoining fluids. To establish this theory it is necessary to prove that there has been exudation of album-

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inous fluid, the truth of which does not seem to have been established any more than that albumen possesses any such selective power over earthly matter as has been attributed to it.

The vascular system when affected by calcareous degeneration becomes rigid, inflexible, inelastic and uncontractile. The affected parts have not the cheesy appearance and feel of atheroma but are hard, gritty, and white. It only remains to bear in mind the peculiar liability of the cerebral vessels to disease, the peculiar delicacy of their walls and the soft consistency of the surrounding brain substance, to understand the very great liability of the aged to apoplexy. Whereas in atheromatous disease dilatations of the walls are seen to be common from the softness, want of contractility and yielding nature of the diseased structures, in cretaceous degeneration the opposite conditions exist from the re-

distance to distension of the calcified walls and their tendency is to diminution of the calibre of the vascular system.

The circulation is impeded by the occluded condition the vessels and if from any cause the return of blood is checked the danger of rupture of the unyielding vessels is imminent and extreme.

But that which falls more natural by to be considered under the present division is the liability to total occlusion of one or more vessels by the encroachments made on their caliber by concretions occupying their walls, thus diminishing or cutting off the supply of blood to certain parts of the brain. Usually within a few days the natural consequences occur in the disintegration and destruction of the part, the effects varying with the amount and importance of the part destroyed. The same effects have been produced by the operation of tying the common carotid, the ramollissement being

clearly the result of atrophy. This condition is characterised by blanching of the affected parts from deprivation of blood and it has been styled "white softening" in contradistinction to the red. Sometimes the deposit of earthy matter exists in a series of bony rings round a vessel leaving intermediate portions unaffected. This determines a very strong tendency to pressure on the walls, the healthy portions being chiefly distended. Excessive fatigue seems most commonly in operation as the cause of atrophic apoplexy. If as is usually the case the vessels have been in such a state that well sustained and equable action of the heart is necessary to the due maintenance of ~~the cerebral~~ vascular supply, it is evident that the action of the heart when in abeyance will not send to the brain the requisite amount of nutriment, ~~and~~ because of the obstructions in the vessels proving too great an obstacle to the weakened and retarded current. The lack

Very frequently when erosion exists
and the current of the blood has become
more than usually languid deposit
of fibrine may occur or of phosphate
of lime in addition and these

of adequate nutrition of the vascular walls may be followed by disintegrations or exfoliation of the calcareous matters contained in them and thus perfect occlusion result.

During stages of depression, then, irreparable mischief may have occurred and the process of disintegration ^{of cerebral tissue} once commenced rapidly progresses. Similar fatal effects may be traced to the depressing agency of certain emotions or the supervention of severe or protracted and debilitating forms of disease.

I think there can be little doubt as to the far more formidable consequences of an attack of this sort ~~or~~ ^{as} compared with ordinary cases of cerebral hemorrhage.

Coma generally supervenes much more gradually than in sanguineous apoplexy.

If with these conditions of the vascular cerebral system there coexist hypertrophy of the left ventricle the liability to apoplexy is greatly augmented on account of the tendency to frequent irregularity of supply of blood to the cerebrum, ~~and frequent approach to intermission.~~

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Dr Law of Dublin was the first to draw attention to hypertrophy of the left ventricle with valvular disease as a cause of ramollissement. He has detailed cases in which the aorta had diminished in size from the less amount of blood passed through it by the ventricle and the retardation of the circulation was such as to produce engorgement of the lungs with pulmonary apoplexy, while the cerebral tissue exhibited white softening, the existence of which had been fore-shadowed by hemiplegia. The ^{aortic} valves were in several cases covered with warty vegetations preventing their application to the sides of the artery and forming a material obstacle to the transmission of a full stream, while regurgitation was freely permitted from their insufficiency. There seems to be no ground for doubting the occasional production of such effects by extreme valvular disease, especially when hemiplegia was explained after

death to have been caused by white softening: at the same time we cannot concede that these cases abound; ~~the~~ disease of the heart usually proving fatal from engorgement of the lungs and secondary effects on the cerebral circulation or by asphyxia resulting from pulmonary engorgement or apoplexy.

Now Dr. Kirker holds that cerebral softening may result from detachment of a fibrous ^{vegetation} ~~deposit~~ from the valves of the left side of the heart ^{which may} ~~and~~ be carried by the arterial circulation to be deposited in some cerebral vessel and thus cause ramollissement. He

1. put forward in support of this view the suddenness of the cerebral symptoms, which he ~~explains~~ ^{explains} by the sudden blocking up of a considerable vessel. The other
2. argument is the absence of local disease in the vessels of the Brain and of general disease in the arterial coats elsewhere. Now the first argument does not appear to be free from

objections, as in several cases of ramol-
 lissement, ^{without such cause} coma comes on with great
 rapidity. A case of this kind once fell
 under my own notice in Dr Bennett's
 ward the winter session before last, and
 I have a very distinct recollection of the
 circumstances connected with it. With
 regard to the second argument, it
 does not seem that particular care
 was taken to adduce cases in which
 the vascular system was entirely free
 from disease; but such admissions
 are made as that "a few specks of
 yellow ~~of~~ deposit were found on the
 coats of the vessel where the obstruc-
 tion occurred - and at the same time
 there were scattered deposits in the coat
 of the aorta, with thickening of the
 aortic and much disease of the mi-
 tral valve." Moreover no evidence
 has been brought to prove that the
 vegetations on the valves are apt to
 be easily thrown off or that they have
 been frequently found elsewhere than

in cerebral vessels.

Therefore while we cannot say that it is impossible for these vegetations to be carried into the cerebral arteries and plug them up, we must add that very inadequate evidence has been adduced to prove that this often happens. Until we have stronger proofs of its liability to occur we must consider it the rarest possible ^{cause of} cerebral lesion.

William James Cumming.
1857.