**Observations on cerebral apoplexy at different periods of life / by Richard Quain.**

3. From the Author

# OBSERVATIONS

ON

# CEREBRAL APOPLEXY,

## AT DIFFERENT PERIODS OF LIFE.

BY


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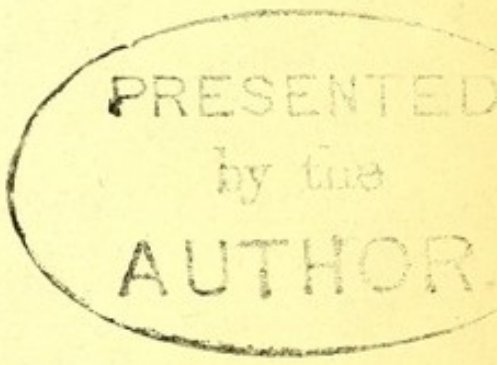


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# THE HISTORIES OF TWO CASES OF CEREBRAL APOPLEXY IN CHILDHOOD;

## WITH SOME GENERAL AND STATISTICAL OBSERVATIONS ON THE CAUSES OF THE DISEASE, AND THE RELATIVE FREQUENCY OF ITS OCCURRENCE AT DIFFERENT PERIODS OF LIFE.

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THE histories of the two following cases of cerebral apoplexy, occurring in childhood, are offered, with the observations which they suggest, as a contribution to the records of similar cases. Both of these cases presented well-marked and characteristic symptoms, each exhibiting at the same time, striking contrasts, not only in the condition of the patients in whom the attacks occurred, but also in the seat of the hæmorrhagic effusion. One of the subjects was an apparently healthy boy, who was suddenly seized whilst at play; in this case the hæmorrhage was found to be in the substance of the brain. The other was an unhealthy girl, who had long been delicate: here the blood was found effused in the arachnoid cavity. In the first case, hypertrophy of the heart existed, and the hæmorrhage was of the sthenic form; in the second, the patient was suffering from purpura, and the hæmorrhage was of the passive or cachectic kind.

This variety of circumstances has led to a series of reflections, which will be found in the following pages. They include some observations on the causes of apoplexy in general, more particularly in reference to the histories of these two cases. Some remarks on the occurrence of apoplexy at this early age, with references to several similar cases, will be given; and a series of statistical facts, which appear sufficient to prove apoplexy to be a disease of middle life rather than that of advanced age, will be found appended.

CASE I.—*Cerebral Apoplexy in a boy aged nine years, characterized by coma, convulsions, and paralysis. Death in seven hours. Large clot of blood in the right hemisphere of the brain. Hypertrophy of the left ventricle of the heart.*

Thomas Smith, aged nine years, a boy of slight conformation, but who is stated to have enjoyed good health up to the time of the present attack, was brought into University College Hospital in a state of insensibility. On inquiry, I ascertained that, between three and four hours previously, he had been trundling a hoop, when he was seen to stop suddenly, put his hand to his head, and fall to the ground, making an attempt to grasp the iron railings of a house He was subsequently seen by a surgeon, who recommended his removal to the hospital.

On his admission, there was coldness of the general surface of his body, particularly of his feet; his face was pale; his tongue slightly protruded from his mouth; his breathing slow, occasionally slightly stertorous; his pulse was small, and varied from 50 to 60 in the minute. The right arm and leg were convulsed; the left arm was flexed, but not apparently paralysed, as when removed from any position in which it happened to be, it resumed the same again; the left leg was paralysed, falling flat after being raised up. He was insensible to pinching, or to loud noises made close to his ear. The pupil of the right eye was widely dilated, that of the left much contracted; and both were equally unaffected by a lighted candle. No external marks of injury were found on shaving the head. The treatment consisted of a cold lotion to the head, warmth to the feet and trunk, and a turpentine enema. He died, with very little change in the symptoms, in four hours after his admission, and about seven hours after the first attack.

*Post-Mortem Appearances.*—The scalp and skull were quite free from any appearance of injury, and the membranes of the brain were healthy; but there was great congestion of the veins and sinuses of the dura mater. The cerebral substance of the left hemisphere was found to be particularly pale throughout. On slicing the right hemisphere, a large clot of blood (about three ounces) and a quantity of serum were found. This coagulum, which extended longitudinally between two and three inches, was placed externally to the right lateral ventricle, and passed downwards to the corpus striatum, but did not communicate with the ventricle. The cerebral substance surrounding the coagulum was not softened. On washing away the clot, the open extremities of several blood-vessels were seen. One in particular, larger than the rest, was traced through the corpus striatum, and was believed to have been the principal source of the hæmorrhage. The lungs were much congested. The heart was enlarged, weighing, when well washed, between five and six ounces. The left ventricle was much hypertrophied, The aortic valves were thickened; but, on being tested with water poured on them through the aorta, they were found perfectly competent for their function. The abdominal viscera were healthy, with the exception of venous congestion of the liver, spleen, and kidneys.

Remarks.—In examining the details of this case, we perceive the characteristic symptoms of an apoplectic seizure, in a child, identical with those which occur in the adult. There existed the like sudden attack, arrest of volition and sensation, collapse, convulsions, unequal action of the pupils, slowness of pulse and respiration, stertor, etc. A post-mortem examination, revealing extensive hæmorrhage in the brain, completes the history.

On inquiring into the origin of this sudden and fatal lesion in so young and apparently healthy a subject, and on looking over the list of the generally assigned causes of the disease, hypertrophy of the left ventricle of the heart meets our view. It was present, as we have seen, in this case; and was, in all probability, under the excitement of the play in which the boy was engaged, the immediate cause of the hæmorrhagic effusion. The relation between apoplexy and diseases of the heart has long been a source of difference of opinion among pathologists; some asserting that the association between the two is intimate; others,

with equal force, denying any relation between them, save coincidence. Lancisi, Baglivi, and Portal, seem to have been the first observers who believed hypertrophy of the left ventricle, or other disease of the heart, to be closely associated with apoplexy in the relation of cause and effect. Malpighi, and, at a later period, Cabanis, died of apoplexy, while labouring under hypertrophy of the heart. The deaths of these distinguished men recalled on each occasion attention to the subject; and the close relation of the two affections has since been received by many as established. Amongst the advocates of this opinion we find Legallois, Corvisart, Richerand, Lallemand, Bricheteau, Andral, Cruveilhier, Boilleau, Adams, Hope, and lastly, Burrows, who has with great ability investigated the true conditions of the cerebral circulation, and removed a mass of error by which the subject was oppressed.[1] He has shewn a very close relation between apoplexy and disease of the heart, having ascertained their coexistence in three-fifths of a number of cases, partly his own, and partly from other sources. In fifty-nine cases of apoplexy he found, in reference to the nature of the disease of the heart present, that in nineteen there existed hypertrophy with valvular disease, in ten simple hypertrophy, in eight valvular disease alone, and in one, simple dilatation—being thirty-eight cases of heart disease in fifty-nine cases of apoplexy. This association, as already stated, has been denied by many persons; in this country by Drs. Kellie and Abercrombie: and in France, by M. Rochoux and others. The latter has stated, that in taking two series of cases, the one consisting of persons who had died of apoplexy, the other of those who had died from other and different causes, he finds the proportion of heart disease to be rather less in the one series (apoplexy), than in the other; consequently that the relation between apoplexy and disease of the heart is a mere coincidence. For example, he found that in thirty persons who died from various acute diseases, the heart was hypertrophied in twenty-six. In thirty persons of about the same age, who died from apoplexy, he found the heart enlarged in only twenty-four. Louis, without expressing any opinion on the subject, states that in forty-five cases of hypertrophy, he did not meet with a case of apoplexy. Other observers, admitting the influenee of hypertrophy, believe that there are several equally powerful influences which cooperate in the production of apoplexy. Thus Dr. Williams includes as a cause, disease of the heart, when coincident, as it often is, with disease of the kidneys. Dr. Watson believes that the left side of the heart is rarely diseased without the right side participating in the affection; hence arises an obstruction to the venous circulation, and an accumulation of blood in the brain. When the vessels are diseased at the same time, rupture is the consequence. It is right to state that Dr. Burrows takes all these conditions into consideration when assigning the chief place to the influence of hypertrophy of the left ventricle, by the action of which an augmented quantity of blood is sent with increased force into the vessels of the brain. The present case seems strongly to corroborate this opinion.

A careful consideration, however, of all the views which have been expressed on this subject, leads me to conclude that though the action

[1] On Disorders of the Cerebral Circulation. London, 1848.

of the left ventricle in a state of hypertrophy may, in the first degree, and perhaps *per se* in some cases, conduce to the causation of cerebral hæmorrhage, there are many not less efficient causes which contribute to the same result. Among these may be mentioned, on the one hand, plethora, with excess of blood in the system; on the other, impaired nutrition, atony and weakness of the walls of the blood vessels, with perhaps a diseased state of blood, as in Bright's disease and purpura; also atheromatous disease of the vessels, and softening of the brain; venous congestion from disease of the lungs, as emphysema, as well as from dilatation of the right side of the heart; from the pressure of tumours, and from many other causes. Several of these sources of mischief often coexist; and in connection with any of them, the excited or increased action of the left ventricle, though healthy, becomes a powerful cooperating agent. Thus, if venous congestion occurs from any of the causes named, and the left ventricle is actively engaged at the same time in pressing the blood forward through the arteries, the brain is necessarily subjected to an increased pressure; coma is the result, and if death follows, we find all the vessels gorged with blood—*congestive apoplexy.* If one or several of the congested vessels give way, hæmorrhage occurs, and we find *hæmorrhagic apoplexy.* If serum has accumulated as the result of venous congestion, or from any other cause, it will offer an obstruction to the free flow of blood; the brain is again subjected to the pressure of the heart's action, and death may be the result. We here find not an excess of blood, though it has been the agent through which in reality the fatal pressure has been applied, but an excess of serum—*serous apoplexy.*[1] Any of these agencies may operate individually: thus in this child the action of the left ventricle sufficed to cause hæmorrhage from vessels, which were, so far as could be ascertained, perfectly healthy; whilst, on the other hand, atheromatous deposits in the arteries of aged persons are sufficient to permit of rupture of their coats, independently of any morbid action of the heart.

Case II.—*Meningeal Apoplexy in an unhealthy child aged two years and seven months. Insensibility and convulsions. Death in four hours. Large coagulum in the cavity of the arachnoid. Purpura—Anasarca—Disease of the kidneys.*

Mary Ann May, aged two years and seven months, had been a healthy baby until she was four months old. Her parents were healthy, but one of her sisters, when 2½ years old, "had a fit"; on recovering from which, her left arm was paralysed. She gradually recovered the use of this arm, but now, in her sixth year, has frequent epileptic seizures. When the subject of the present case was four months old, her mother was compelled to wean her, and she has since then been a delicate child, and has neither been able to walk or speak. Her food has been poor and deficient. She has suffered much in dentition, and at the

[1] When the serum contained in the arachnoid, the cerebro spinal fluid, as it is called, exists in its normal proportions it can pass with facility into the vertebral canal, and thus the contents of the skull can adapt themselves to the capacity of the space which contains them. But when the fluid is in excess, or when it is infiltrated beneath the pia mater, this adaptation cannot take place, and an injurious pressure is the result of any increase in the quantity of blood sent to the head.

time of her death, was cutting one of her eye-teeth. Her mother had for five or six months observed that her feet were swollen, and that in cold weather they became remarkably cold and livid. The child has had a good appetite, and has slept well. Her bowels have been in general relaxed; she frequently cried as if in pain when making water. She has not been known to indicate any pain or suffering in the head. Being unable to walk, she generally lay or sat on the floor, and was so weak, that the gown of her mother brushing past was sufficient to throw her down. Her mother recollects that she had one or two severe falls, but none within the last six months. The marks of bruises which appeared on her body were subsequently attributed by the mother to slight falls, or to the child having been pinched or pulled about by some of the other children. She had never taken particular notice of them.

It appears that her appetite had been bad for the two days preceding her death; and that, at about half-past three o'clock on the day of its occurrence, one of her sisters, who was holding her, noticed a jerking or catching of the jaw, that she was about to fall, and that she suddenly went into a fit and became insensible. Her body became rigid, her hands clenched, her jaws fixed, and she foamed at the mouth. This fit lasted a quarter of an hour. She got better for a few minutes, when another fit came on, lasting as long as the first; there was a similar appearance of relief, and a third fit, shorter in duration, succeeded. Some domestic remedies were applied; the child seemed better, and swallowed a little tea, went to sleep, and awoke about half-past seven, when she had another fit and died immediately. An order had been procured for one of the parish surgeons, but not in sufficient time to enable him to see her during life.

*Post-Mortem Appearances.*—I was not present at the opening of the body, but subsequently examined it and the different organs. The following description of the morbid appearances is founded on this examination, and on notes kindly given me by my friend, Mr. Greenhalgh, who was present at the previous inspection.

The body was emaciated; the feet and legs were œdematous, and numerous small spots of purpura were visible, particularly on the legs. There also appeared to be ten or twelve bruises on the scalp, face, arms, and legs; but they were mere brownish discolourations, arising from slight extravasation of blood under the skin, extending also, in some of the spots, into the subcutaneous cellular tissue. There was no external abrasion, nor any swelling. Each of the clavicles presented a remarkable bend at right angles to its length, on its own plane; and one of these bones had the appearance of having been, at some antecedent period, partially fractured. In the cavity of the arachnoid, particularly over the anterior portion of the right hemisphere, there was a coagulum of blood: there was also effusion of blood beneath the arachnoid, in the pia mater. These effusions, which extended to the left side, and also to the base of the brain, were supposed to amount in all to at least four ounces. The lungs and heart appeared healthy; the latter contained no blood. The liver was enlarged, remarkably pale, soft, and mottled. The kidneys were enlarged, very soft and pale, and evidently diseased. There were "some elevated, and apparently ulcerated, patches in the large intestine"; the mesenteric glands were enlarged.

Remarks.—The history of this case closely corresponds with that of some others, to be referred to presently. The attack occurred in an ill-nourished, unhealthy child, and was the result of a diseased state of blood (as shewn by the presence of purpura). The vessels, in such cases, participating in the general impairment of nutrition, lose their tonicity, admit readily, as from the reflected irritation of teething, of congestion and finally rupture, giving rise, as in the present instance, to the form of apoplectic effusion, known as *asthenic*, *cachectic*, or *passive* hæmorrhage. This case contrasts well with the preceding one, in which the powerfully-contracting left ventricle of the heart gave rise to hæmorrhage in an otherwise healthy subject.

The appearance of bruises on the body of this child led to a medico-legal investigation, which compromised the liberties—and it might have been, the lives—of its mother and aunt. One of the medical gentlemen who examined the body, having been informed by some persons that the relatives had treated the child badly, came to the conclusion, on seeing the bruises and the bent clavicles, that the effusion was the result of direct violence, and that it could not have been produced even by a fall. On this evidence the mother and aunt were sent to prison by the magistrate. On subsequently examining the body, by direction of the coroner, and seeing the anasarca, purpura, and diseased kidneys, I concluded that the bruises (which, though numerous, were slight), did not indicate the application of violence—(in one of Dr. West's cases, subsequently quoted, the knot of a night-cap sufficed to cause a similar appearance); and that the hæmorrhage, though a fall might have contributed to its production, was, in all probability, the effect of natural causes. In this opinion the jury coincided. It was further shewn in evidence, that the child had been kindly treated, and that the persons who reported to the contrary had been at enmity with the family. After a week's detention, and much consequent mental suffering, the relatives were again brought before the magistrate, and discharged. The chief arguments used, independently of the appearances described, in support of the notion of external violence, were: 1st, that apoplexy did not occur at this early age without some special cause, such as a violent blow—(the cases quoted in another part of this communication will shew such an opinion to be quite untenable); and 2nd, that if the hæmorrhage had been the result of purpura, the blood would not have been coagulated. That this, too, is a mistaken opinion, has been shewn by Dr. Williams,[1] and very recently by Dr Parkes.[2] Indeed, the fact has been long familiar to myself and others, that the blood is not prevented from coagulating by the existence of purpura.

## THE RELATIVE FREQUENCY OF CEREBRAL APOPLEXY AT DIFFERENT AGES.

Passing from these special points in the history and pathology of these interesting cases, it will be useful to view them connectedly, in reference to the early age at which the fatal lesions occurred, and to consider their relation, in this respect, to similar cases. There are

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[1] Principles of Medicine, 2nd edit.

[2] Medical Gazette, Nov. 17, 1848.

three phases or periods of life marked by such wide distinctions in their attributes and relations, that it will be useful to place our remarks under heads thus respectively indicated. They are:—

I. That of the infant at, or immediately after, birth.

II. That which extends from a few hours after birth to the age of 20, —that of full puberty or adolescence.

III. From the age of 20 to extreme old age or decrepitude.

No difference of opinion can exist, that it is this latter period or division of life, which includes the great majority of the cases of apoplexy which occur; but considerable doubt, and some errors, exist as to the periods of this lengthened interval, during which the predisposition to apoplexy is strongest. Some carefully-observed facts will, I hope, throw light on this subject, and afford an interesting and striking result. I shall first briefly consider the occurrence of apoplexy in early life, illustrating the remarks on the second period by reference to several cases. It is a remarkable fact,—rendered still more so by the observations which will presently be made,—that many writers, with extended means of observation, have not met with, or at least have not recorded, any cases of apoplexy before twenty years of age. We need not go further for an illustration than to the table of cases collected by Dr. Burrows[1] from his case books, and from the writings of others, amounting to 215 cases, and in all of which the individuals were over 20. It is probable (certain, in Dr. Abercrombie's cases, here included) that some few examples of the disease, occurring anterior to the twentieth year, have been omitted; yet sufficient evidence remains to show, at least, that such cases are considered very rare. Rochoux, indeed, goes further than this, and states his doubts as to the occurrence of true apoplexy before the age of puberty.[2] I have referred to the reports of the Registrar-General for information on this point; and find the following results of an examination of the tables of mortality for fifty-two weeks, ending the first week of November 1848.

*Ages of persons returned as having died of apoplexy in fifty-two successive weeks of the years* 1847-48.

| Age | 0 to 15. | 15 to 60. | 60 and upwards | |
|---|---|---|---|---|
| Number | 134 | 495 | 572 | Total 1201.[3] |

This return cannot, of course, be received as perfectly correct, the cause of death being too often returned without sufficient evidence of its accuracy; yet there must still be a large number of cases under the assigned age. The number 135 probably includes many infants who died at birth. These are the cases comprised in our first division, to which I shall now briefly allude.

1. HÆMORRHAGIC EFFUSION, OR APOPLEXY, IN INFANTS AT BIRTH.—In a very large proportion of the children who die during birth, or immediately afterwards, in a state resembling asphyxia, if the head be examined, blood will be found effused in the cavity of the arachnoid, and beneath this membrane. Such cases have been described by several writers on the diseases of children, including Barthez and Rilliet, Dr.

[1] Op. cit. p. 130. [2] Dictionnaire de Médecine.

[3] This number gives the proportion of about one case to 1782 persons actually living in the metropolis.

West and Dr. Kennedy,—the latter of whom has recorded a number of these cases in the *Dublin Journal*.[1] The locality of the hæmorrhage has given rise to the designation, *meningeal apoplexy;* this form is often accompanied by apoplexy of the cerebral substance in points, *capillary apoplexy*. The peculiar circumstances, however, in which the infant is placed during birth, the compression to which the skull is subjected, the change of shape which it undergoes, the impediment to respiration which frequently occurs, as well as the succeeding events (including the change of position, the altered circumstances of the functions, not only of the heart and lungs, but also of the skin, mucous membranes, and other organs and tissues), become predisposing and exciting causes of cephalic congestions and effusions much more frequently at this early period of life than in the second period. These peculiarities place so broad a line of demarcation between this class of cases, and those to which our inquiry more particularly belongs, that it will be unnecessary to make further allusion to them.

II. HÆMORRHAGIC EFFUSION, OR APOPLEXY, OCCURRING FROM THE PERIOD OF BIRTH TO THAT OF PUBERTY.—When we contrast the condition of the brain, and its circulation, in the adult, with that in the child or young subject, we can have little difficulty in arriving at the conclusion, that apoplexy will be less likely to occur in the one case than in the other. A brief reference to the causes of the disease, already mentioned in this paper, will suffice. In early life we find the skull less resisting, and the blood-vessels more elastic and yielding, than in the adult,—where the one becomes dense and rigid, and the others frequently the seat of deposit or degeneration. In the young subject, the energies of the system are directed to nutrition and growth; in the adult, the chief call is on the brain and nervous system, which then has to bear the wear and tear of life. The heart, in the progress of life, becomes larger and more powerful, it is subjected to greater excitement, and becomes oftener the seat of disease,—all interfering with the circulation through the brain. These, and other causes, make a wide difference in the comparative frequency of apoplectic disease in the second and third periods of life. There is, however, another condition on which apoplectic effusion sometimes depends: viz., that in which diseased blood escapes from equally diseased blood-vessels, as already described in the remarks on the second of the two preceding cases. I allude to purpura hæmorrhagica and analogous conditions. Here the function of nutrition, active in childhood, is interfered with; and hence the form of apoplexy resulting is of the asthenic or cachectic character. This form of apoplexy is found to occur more frequently in children than in adults,—a fact set forth in other parts of the present essay.

The number of cases of cerebral hæmorrhage and apoplexy, occurring between birth and puberty, which I have collected from different sources, amounts to twenty-three, making in all, with the two preceding cases, twenty five. Of these, however, there were three cases of hæmorrhage without coma, and in another case the symptoms were not very characteristic. This will reduce the number to twenty-one,—quite sufficient to form a basis for future observations.

[1] March 1837.

I have thought it desirable rather to give a short outline of each case, than to mention merely the age, and I have divided the cases into two classes: one, in which the hæmorrhage occupied the substance of the brain itself (fourteen cases); the other, in which blood was effused into the membranes (nine cases). The latter or meningeal form of the disease is generally believed to be the most frequent in children; though, on the present occasion, it will be found in the minority,—a fact which will be partially explained by my having placed, in the first class, three cases of which the *post mortem* appearances are not given: viz., a case by Dr. Kennedy, one by Andral, and another by Serres, and which might have belonged to the meningeal form.

A. *Cerebral Apoplexy—Hæmorrhage in the Substance of the Brain.*

1. An infant, three days old, apparently quite healthy, presented the ordinary symptoms of well marked apoplexy, and died suddenly. After death a clot of blood was found in the substance of the left hemisphere of the brain, immediately outside the corpus striatum. The tissue was "a little softened around it."[1]

2. An infant, five days old, presented characteristic symptoms of apoplexy, and died suddenly. A *post-mortem* examination was not permitted.[2]

3. A little girl, eleven months old, had suffered from head symptoms for a considerable time. She had an attack of extreme faintness forty-eight hours before death. After death several apoplectic clots were found in the brain, with much venous congestion. There was thickening and obliteration of several of the veins, the result of previous inflammation.[3]

4. A girl, two years old, had measles, and subsequently suffered from general illness. She was seized with convulsions, and the right leg became paralysed; the left pupil was contracted. Death occurred in a few hours after she came under observation. After death, a quantity of blood was found in the optic thalamus and corpus striatum, but on which side is not stated.[4]

5. A case (subject three years old) recorded by Lallemand[5] is very similar to that of Dr. West, already referred to.

6. M. Serres states that he has seen apoplexy in a child three years old. I have been unable to find the particulars in his essay.[6]

7. A child, five years old, laboured under an apoplectic torpor rather than coma, and died in a state of great prostration. After death there were found "capillary apoplexy and a clot of blood."[7]

8. The same authors record the case of a delicate girl, aged seven, who died after having suffered head symptoms for several days. After death, a clot of blood was found in the left optic thalamus.[8]

9. Mr. Worthington refers to Dr. Abercrombie as recording the case of a child, aged 9, in which both lateral ventricles contained clots of blood; that in the right ventricle being of the size of a large walnut. The

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[1] Billard, "Traité des Maladies des Enfans," p. 600.
[2] Kennedy, "Dublin Journal of Medical Science," 1837.
[3] West, "Lectures on Diseases of Infancy," etc. Lect. 4.
[4] Barthez et Rilliet, "Maladies des Enfans," tom. ii, p. 65.
[5] Récherches Anat. Path. sur l'encephale. Lettre 3ieme. [6] Annuaire des Hôpitaux, p. 284
[7] Barthez et Rilliet, Op. cit. tom. ii, p. 54. [8] Op. cit. p. 56.

symptoms, and the state of the substance of the brain itself, are not given. I have been unable to find the original case.[1]

10. Barthez and Rilliet refer to a case in the *Edinburgh Medical and Surgical Journal*, in which, without any disease of the brain, clots of blood were found in both lateral ventricles, and at the base. The subject was a boy, aged nine. The symptoms preceding death, were, headache, convulsions, constant movements of one side of the body, contraction of the fingers, followed by relief: on the sixteenth day, profound coma, and death in a few hours.[2]

11. A girl, when young, had suffered from necrosis of the jaw, and of the bones of one of her feet. Her health became re-established, and she continued well for some time, until head symptoms came on. She had convulsions of the right side, succeeded by paralysis of the left. From this she was relieved, as also from a subsequent attack of a similar character; but died after an attack of coma, of eighteen hours' duration, in her eleventh year. After death there was found a clot of blood, larger than a hen's egg, external to the right ventricle. The brain was softened. Blood was also found in the subarachnoid space over the surface of the right hemisphere.[3]

12. A boy of the same age, in good health, was found shortly after his breakfast in a state of insensibility and collapse, with stertorous breathing, convulsions, the right pupil dilated, and the left contracted. He died in about twelve hours from the commencement of the fit. After death, a large clot of blood was discovered in the right ventricle and adjoining portion of the brain. In other respects, this organ was healthy. His stomach and intestines contained a large quantity of raw undigested turnips.[4]

13. A case is referred to by Andral, in a child twelve years of age. The history is not narrated. He alludes to Guersent, as having seen similar cases.[5]

14. A boy, fourteen years of age, had been destitute during the winter. In a state of great debility in the spring, he complained of headache, restlessness, general feverishness, and slight delirium. On the following day he was brought to the hospital, in a state of insensibility, and died in an hour afterwards. A clot of blood was found in the posterior portion of the right hemisphere.[6]

It is unnecessary to extend this epitome of cases by referring to those which are recorded as having occurred between the ages of fourteen and twenty, such cases are not very unusual. Several may be found in Mr. Copeman's collection of cases of apoplexy.[7]

### B. *Hæmorrhage in the Membranes of the Brain—Meningeal Apoplexy.*

Serres[8] appears to have been the first writer who distinguished the effects of apoplectic effusions in the substance of the brain from those in which the effused fluid is contained in or beneath one of the membranes

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[1] Provincial Medical and Surgical Journal. New series. Vol. iii.
[2] Op. cit. p. 56.
[3] West, Op. cit.
[4] Mr. Worthington in "Provincial Journal," new series, vol. iii, p. 180.
[5] Pathological Anatomy, vol. ii, p. 723.
[6] Guibert, "Archives Générales, 1827," and Barthez and Rilliet, Op. cit. p. 52.
[7] A Collection of Cases of Apoplexy, by Edward Copeman, surgeon. London, 1845. Annuaire des Hôpitaux, 1819.

enveloping this organ. He attributed much importance to this distinction, believing that the lesion of the cerebral substance was always accompanied by palsy, whilst that confined to the membranes did not produce this result. On this distinction he founded a diagnosis as to the seat of the extravasation. He shewed also that meningeal apoplexy occurs chiefly in children under fifteen years of age, and in men above sixty.[1] He found that it occurred more frequently in women, and at a much earlier age than that last mentioned. In forty-one cases there were thirty-three females and only eight males.[2] It is almost invariably this form of apoplectic effusion which occurs in infants during birth, as already stated, and also in those children who are rendered cachectic by bad nursing, bad air, and bad food. These latter are the cases which are described by Barthez and Rilliet, Dr. West, and others, under the name of *cachectic, or passive hæmorrhage.* The following are examples of the meningeal form of apoplexy in children.

15. A male infant had suffered from drowsiness, sickness, and slight jaundice when a fortnight old. When five weeks old, he was seized with hurried respiration, great depression, and convulsions, during which he screamed aloud. Spots of purpura appeared on various parts of the body, and a dark mark under the chin, where the knot of the night-cap pressed. The fits recurred frequently, and he died in a state of exhaustion sixty hours after the first fit. After death, there was found to be much congestion of the sinuses of the skull, with large coagula over both hemispheres in the cavity of the arachnoid. The body was generally anæmic; the heart was healthy.[3]

16. A girl thirteen months old (in the practice of Drs. Lombard and Panchaud, of Geneva), after recovery from bronchitis, was seized with an attack of convulsions, which ceased, and she slept for a short time. The convulsions reappeared, and the child died suddenly in nineteen hours from the first attack. Hæmorrhage was found to have occurred beneath the arachnoid, in the pia mater, over both hemispheres of the brain. There was slight softening of some portions of the cerebral substance, and a tendency to hæmorrhage in points.

17. A boy, two years old, had convulsions during five weeks antecedent to his death. They became more frequent, he complained of his head, and bled much from the nose. After death, a large clot of blood, which had evidently been some time effused, was found in the cavity of the arachnoid.

18. A boy, two years and a half old, had suffered from some obscure illness for several months. There were many purpuric spots on the body. The immediate cause of death was lobular pneumonia: there were no cerebral symptoms. Clots were found over both hemispheres in the arachnoid cavity.

19. A girl, two years and a half old, had been delicate from the age of six months, in consequence of difficult dentition. On the day preceding her death, she exhibited signs of a convulsive attack, which became more marked, and she died. After death, a clot of blood was found in the cavity of the arachnoid.[4]

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[1] See also a Memoir, with a number of cases which occurred in old men, by Prus, in Mém. de l'Acad. de Médecine, vol. xl.

[2] Op. cit. p. 284.

[3] West, Loc. cit.

[4] These four cases are recorded by Barthez and Rilliet, Op. cit.

20. A child, two years and a half old, of scrofulous constitution, badly nourished, had disorder of the abdominal viscera, and symptoms of abdominal irritation. The child's mother was several times alarmed by its face becoming livid, and by its gasping for breath; a condition which generally lasted one or two minutes. It died in a similar fit. After death, the vessels of the brain were found congested; blood had been poured out in considerable quantity on the surface; the cerebral substance was healthy. The heart was small, the lungs were gorged, the liver and kidneys were enlarged.[1]

21. A boy, three years old, had suffered from the age of two years and five months from passing blood by vomiting and stool. His food was bad; and when three years old, he was puny and delicate. While he was in a state of exhaustion from diarrhœa, coma came on, and he died in twenty-four hours. After death, a large coagulum, amounting to six ounces, was found on the surface of the right hemisphere, and a small clot in the substance.[2]

22. A girl, twelve years of age, having suffered from some obscure illness for several days, was suddenly seized with loss of consciousness, and her left side was paralysed. Her consciousness was restored, and she regained some power in the side; but she became gradually weaker, and died in fifteen weeks from the date of the attack. After death, the remains of an old clot of blood were found over the right hemisphere, the substance of which was also slightly softened.

23. A girl, fourteen years old, had induration of the liver, and scrofulous glands. She died without any cerebral symptoms. After death, a clot of blood was found in the cavity of the arachnoid, over the anterior portion of the right hemisphere of the brain.[3]

It would be interesting to enter at greater length into the histories of the preceding cases, but it is not possible to do so on this occasion. The facts, thus briefly set forth, exhibit a great variety in the symptoms, and shew that in many respects they resemble those of very different cerebral lesions. The observation of this fact led Barthez and Rilliet to remark that they believed *cerebral hæmorrhage occurring in childhood was often confounded with other diseases, and that this confusion caused its frequency and importance to be overlooked.* The reader will observe that there was considerable effusion of blood, without head symptoms, in three cases. Such cases are not strictly apoplectic; yet I have felt that their mention could not be uninstructive.

In reference to the seat and frequency of cerebral hæmorrhage in children, the authors just named say, that in their own practice they have met with seventeen cases of meningeal, and eight of cerebral hæmorrhage. From these sources, and from the cases recorded by others, they have drawn up the annexed table,[4] shewing the age, sex, and seat of hœmorrhage in thirty-eight cases of apoplexy in subjects under fourteen years of age.

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[1] Mr. Greenhow, "London Medical and Physical Journal," vol. xlvii.

[2] Dr. West, Op. cit.

[3] Both the latter cases are from Barthez and Rilliet. Op. cit.

[4] Op cit. p. 64.

MENINGEAL HÆMORRHAGE.

| Years. | Cases. | Males. | Females. |
|---|---|---|---|
| 1 and 1½ | 4 | 1 | 3 |
| 2 and 2½ | 8 | 6 | 2 |
| 4 | 3 | 0 | 3 |
| 5 to 7 | 4 | 4 | 0 |
| 11 to 14 | 4 | 1 | 3 |
| Total, | 23 | 12 | 11 |

CEREBRAL AND VENTRICULAR HÆMORRHAGE.

| Years. | Cases. | Males. | Females. |
|---|---|---|---|
| 2 | 2 | 1 | 1 |
| 3 | 2 | 2 | 0 |
| 4 and 5 | 3 | 0 | 3 |
| 7 | 2 | 2 | 0 |
| 9 and 10 | 3 | 3 | 0 |
| 12 to 14 | 3 | 1 | 2 |
| Total, | 15 | 9 | 6 |

The preceding facts will show that the earlier periods of life are not as exempt from these lesions as has been generally supposed; and they will serve as a nucleus for future observations. Their present number, however, is scarcely sufficient to justify any conclusion as to the precise age, in children, most liable to apoplexy, except that it appears to occur frequently between the second and third years of life.

### III. RELATIVE FREQUENCY OF CEREBRAL APOPLEXY AT DIFFERENT PERIODS OF LIFE SUBSEQUENT TO THE AGE OF PUBERTY.

In this portion of our inquiry I propose to avail myself of the statistics of forty-nine cases of apoplexy which came under my notice during the time (above five years) that I was house-physician at University College Hospital.[1] The age was ascertained as accurately as possible in every case; and in every instance there was either a post-mortem examination, or a fit in which insensibility, followed by paralysis, occurred, which justified the case being considered as one of apoplexy. The record has this peculiarity, that the date of the first fit (if there has been more than one) is that which has been retained. The conclusions thence derived are therefore necessarily as to the *period of life most liable to apoplectic seizures*, and not as to *the age at which death by apoplexy occurs*. I believe this to be a distinction of some importance; for, as it must be always a matter of doubt whether a fit will be fatal or not, our inquiries should tend to illustrate the date of its probable occurrence rather than that of its probable fatality. It is this date (that of the probability of the apoplectic seizure) which is of importance in our prophylactic, as well as in all other, considerations.

Table I shews, that of the forty-nine cases, twenty-seven occurred in males and twenty-two in females. The relative frequency of the disease in the sexes, thus shewn, corresponds with the generally-received opinion, that apoplexy more frequently attacks males than females. As the number of females living is greater than that of males, the difference is in reality somewhat greater in favour of the latter. The entire number of cases of every description received into the wards of the hospital cotemporaneously with those of apoplexy, just referred to, amounted to 3665; of these, 1834 were males, and 1831 females. When we thus take all diseases, and find the proportion of the sexes nearly identical, we may safely conclude that the disproportion shewn in the cases of apoplexy is not due to any accidental or special disturbing causes, but that *the liability to the disease is in reality greater in males than in females.*

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[1] I have pleasure in acknowledging the assistance of my friend Mr. Wilkes (late physician's assistant at the hospital) in preparing the table of ages.

This table shews also in other respects a marked distinction, still connected with the influence of sex. The age of the oldest male attacked, of these twenty-seven cases, was sixty-two years; that of the youngest, twenty-two. The oldest female was aged seventy-five; the youngest thirty. Hence, so far as the number of facts justifies a conclusion, *the disease occurs earlier in life in men than in women;* and further, *the liability to attacks ceases in the like order.* The uniformity of progression, as shewn in the table, seems to support the probability of the facts being sufficient to justify the conclusion. The mean age was, of males, 34.19 years, of females, 50.9.

The Table numbered II, shews the frequency of attacks during decennial periods of life, from twenty to eighty.[1] From it we learn that the *greatest number of cases of apoplexy occurs in males between the ages of* 40 *and* 50; *in females, between those of* 50 *and* 60, in the proportion of ten to seven. The former fall beneath the pressing influences which at this period of life surround them, whether these influences derive their origin from the incessant demands made on the functions of the vascular and cerebro-spinal systems, or from the irregularities to which the digestive and secerning systems are too often then subjected. In females we observe the frequency of the disease increase after the period when menstruation has for some time ceased. This result is not quite consistent with that which might have been *à priori* anticipated. The feminine attributes, however, become less marked at this period, and many women then evince and put into operation mental and physical powers which had previously been undeveloped. Can such a change have aught to do with the result before us?

This table shews further a reduced scale of the population alive of both sexes, according to the census of 1841, arranged in decennial periods. When we compare the relation between the number of apoplectic cases in each period and the number of persons living, we get the only accurate estimate which can possibly be had, of the relative frequency of the disease. On doing so, we find that relation to be (without reference to sex) as 1 to 66 between the ages of 20 and 30,[2] omitting fractions; 1 to 25 between 30 and 40; 1 to 16 between 40 and 50; *and* 1 *to* 10 *between* 50 *and* 60, *at which point the disease reaches its acme of frequency, and then again diminishes;* being 1 to 18 between 60 and 70; and 1 to 16 between 70 and 80. After 80 no case came under observation. It is a singular and confirmatory fact that in 35 cases Bricheteau did not find one above 60 years of age. The steady uniformity of these numbers in the ascending, and again in the descending scale, seems to confirm the probability of their being an accurate representation of the real state of the question. It will be found, on referring to the Tables III and IV, of cases recorded by other observers, that this uniformity does not there exist. The nearest

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[1] It will be seen that I have not included the two cases in young subjects, of which the histories have been given here. I have strictly confined myself to the regular admissions to the medical wards of the hospital, and have avoided the introduction of cases from other sources of observation. I might have added several, but they would interfere with what I believe fairly represents the actual amount of disease in a given district of considerable extent.

[2] It is hoped it will be understood that the relation is shewn to a reduced scale of population, and not to the numbers *actually* living in the metropolis. It has been stated already, that there is about 1 death by apoplexy for every 1800 persons of all ages living. (Note p. 7.)

approach to it is in the cases observed by Dr. Burrows; the others ascend and descend with great irregularity. It is also a remarkable fact, that, with one exception, the proportion in my cases finds, in each period, a nearly similar proportion in the same period of one of the other observers, though each may differ from the others. For example, I find the proportion of cases to the population at the period between 20 and 30 to be as 1 to 66; Mr. Copeman as 1 to 53; Dr. Burrow's collected cases 1 to 80; his own cases 1 to 135; and M. Rochoux 1 to 285. Again, between 50 and 60, I find it as 1 to 10; Dr. Burrows 1 to 10; Mr. Copeman 1 to 20. Additional confirmation of the correctness of my figures may be derived, as already stated, from comparing the proportions which I have found with those of some one of the other observers; thus, 1st, 1 to 66 between 20 and 30 years of age is between 1 to 53 of Mr. Copeman and 1 to 80 of Dr. Burrows; 2ndly, 1 to 25 between 1 to 22 of Dr. Burrows and 1 to 38 of Mr. Copeman; 3rdly, 1 to 16 is not far removed from 1 to 18 of Dr. Burrows; 4thly, 1 to 10 corresponds with 1 to 10 of Dr. Burrows (fractions being omitted); 5thly, the 1 to 18 between 60 and 70 finds its nearest approach in the 1 to 10 by Mr. Copeman; 6thly, 1 to 16 between 70 and 80 finds a relation in Dr. Burrow's cases of 1 to 21, while that of M. Rochoux is as high as 1 to 3. Thus the facts collected by these observers seem to give strength to the results which are here developed, viz., *that apoplexy is not, as many suppose, chiefly a disease of old age; neither does it uniformly increase in the frequency of its occurrence as life advances, but that the liability to its attacks, considerable between* 30 *and* 40, *is greatest between the ages of* 40 *and* 60—a conclusion which is exactly in the words of Hippocrates, and which is, I believe, as near an approach to accuracy as the proof of it is an interesting fact. The opinion to the contrary (that apoplexy is a disease of advanced old age), is in a great measure due to the records put forward by Rochoux, whose cases were derived from the wards of the Bicêtre—an hospital for aged men. Hence the value of a series of cases collected like those here presented, in which there has been nothing particular or special to cause them to be recorded or remembered, but which, so far as they go, represent the ordinary types of the disease. I believe, then, it may be safely concluded, that the liability to apoplexy, which is greatest between 40 and 50 in the male sex, and between 50 and 60 in the female, diminishes gradually after the last-named age in each sex. The source of error, as in Rochoux's case, by which apoplexy is shewn to be a disease of extreme old age, has been already indicated; but there is another and still greater source of error. Very many sudden deaths are attributed to apoplexy without reference to the state of the heart. Morgagni was accustomed to decapitate persons supposed to have died of apoplexy, for the purpose of removing the parts for demonstration, without any reference to the state of the other organs. I hope to be able to shew, on a future occasion, that many sudden deaths in advanced life are connected with a morbid condition of the heart's tissue. The length to which this communication has already extended, prevents any further allusion to this affection at the present time.

[TABLES.

TABLE I. Showing the number of cases of apoplexy observed, with the maximum, minimum, and mean ages of the individuals, distinguishing the sexes.

| Sex. | Totals. | Max. ages. | Min. ages. | Mean ages. |
|---|---|---|---|---|
| Males ..... | 27 ..... | 62 ..... | 22 ..... | 34.2 |
| Females. .... . | 22 ..... | 75 ..... | 30 ..... | 50.9 |

TABLE II. Showing the number of cases of apoplexy, observed in each decennial period of life from twenty to eighty years, distinguishing the sexes; also a reduced scale of the numbers of the population living in each of these periods, and the proportion of the cases to this scale of population.[1]

| Age. | Males. | Females. | Total. | Reduced scale of population. | Proportion of cases to this scale of population. |
|---|---|---|---|---|---|
| 20 to 30 | 5 | 1 | 6 | 400 | 1 to 66.4 |
| 30 to 40 | 6 | 6 | 12 | 300 | 1 to 25 |
| 40 to 50 | 10 | 3 | 13 | 210 | 1 to 16.2 |
| 50 to 60 | 5 | 7 | 12 | 125 | 1 to 10.5 |
| 60 to 70 | 1 | 3 | 4 | 75 | 1 to 18.3 |
| 70 to 80 | 0 | 2 | 2 | 32 | 1 to 16 |

TABLE III. Showing the number of cases of apoplexy from the tables of different authors, arranged according to the ages in decennial periods.

| | Under 20 | 20 to 30 | 30 to 40 | 40 to 50 | 50 to 60 | 60 to 70 | 70 to 80 | 80 upwards | Total |
|---|---|---|---|---|---|---|---|---|---|
| Dr. Burrows[2] | 0 | 2 | 9 | 6 | 8 | 7 | 1 | 1 | 34 |
| Dr. Burrows[3] | 0 | 12 | 13 | 27 | 23 | 24 | 17 | 2 | 118 |
| Mr. Copeman[4] | 20 | 30 | 31 | 31 | 25 | 30 | 25 | 5 | 197 |
| M. Rochoux[5] | 0 | 2 | 10 | 7 | 13 | 24 | 12 | 1 | 69 |
| Dr. R. Quain | 0 | 6 | 12 | 13 | 12 | 4 | 2 | 0 | 49 |
| Total | 20 | 52 | 75 | 84 | 81 | 89 | 57 | 9 | 467 |

TABLE IV. Showing the proportions of the preceding cases to the scale of population living in each decennial period.[6]

| | 20 to 30 | 30 to 40 | 40 to 50 | 50 to 60 | 60 to 70 | 70 to 80 | 80 upward |
|---|---|---|---|---|---|---|---|
| Dr. Burrows | 1 to 135 | 1 to 22 | 1 to 23.2 | 1 to 10.3 | 1 to 7.1 | 1 to 21 | 1 to 4.1 |
| Dr. Burrows (collected cases) | 1 to 80.6 | 1 to 49.9 | 1 to 18.2 | 1 to 13.1 | 1 to 7.2 | 1 to 4.8 | 1 to 8.1 |
| Mr. Copeman | 1 to 53.1 | 1 to 38.3 | 1 to 27.3 | 1 to 20.3 | 1 to 10 | 1 to 5.3 | 1 to 5.3 |
| M. Rochoux | 1 to 285.1 | 1 to 42 | 1 to 42.6 | 1 to 13.9 | 1 to 4.9 | 1 to 3.9 | 1 to 10 |
| Dr. R. Quain | 1 to 66.4 | 1 to 25 | 1 to 16.2 | 1 to 10.5 | 1 to 18.3 | 1 to 16 | |

[1] This and the two following tables are not intended to be fractionally correct.

[2] On Disorders of the Cerebral Circulation. London, 1846.

[3] Collected from Abercrombie, Andral, Bright, and Hope.

[4] A Collection of Cases of Apoplexy, by Edward Copeman, surgeon. London, 1845.

[5] Dictionnaire de Médecine.

[6] The scale is of course proportioned to the whole number of cases referred to by each writer.

23, Harley Street, London, November 1848.