The Cerebral Associations of Raynaud's Disease.[1]

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IT was in 1862 that Maurice Raynaud first made public his description of the condition with which his name has since been associated, and his Thesis for the Doctorate in Medicine has now become a classic in medical literature (1). Although there may be meticulous critics who will dispute his claim to priority in the delineation of the disease-complex known as Raynaud's disease, there can be no doubt that it was he who first succinctly and clearly described the condition. To use a phrase which is apposite in dealing with such a subject—it was Raynaud who first drew a "line of demarcation" between the symptoms characteristic of his syndrome and those typical of gangrenes in general. In his own words, he was dealing with "a very limited corner of the general history of gangrenes." In the present paper it is proposed to remain within still stricter limitations, yet even there the amount of clinical material is not inconsiderable. There is an additional interest, too, from the fact that Raynaud, especially in his later researches (2), became more and more convinced that the explanation of the peripheral symptoms should be sought in some change in the central nervous system.

It may be of interest briefly to summarise Raynaud's description of the peripheral changes which are to be seen in this disease. In the mildest degree there is local syncope in which the toes, fingers, nose, or ears are yellowish-white, cold, numb, insensitive to touch; next there is local asphyxia, in which the fingers, for example, are cyanosed; and thirdly, the condition may become more marked and gangrene may result. According to Raynaud these are degrees of one condition. The point upon which he laid most stress is the symmetrical nature of the disorder, there is, he said, "une remarquable tendance à la symétrie" (3).

This tendency to symmetrical involvement is the most characteristic feature of the condition differentiated by Raynaud. It is almost needless to say that not all degrees of the disorder occur in a particular patient, although cases have been recorded in which they have all been observed.

Such an obvious and intractable condition as gangrene was

certain to attract the attention of observers. From Hippocrates onwards it has formed the subject of much discussion and speculation.(3) Raynaud realised this fully, and he devoted some time to the consideration of the work of his predecessors, but he came to the conclusion that little work had been done before his time in regard to the question of gangrene resulting from disorder in the nervous system. One observer, Zambaco, he considered, however, as going too far in the opposite direction (4). Zambaco, whose material was drawn chiefly from among the insane, believed that if the function of the nervous system is interfered with, it alone can bring about gangrene without any lesion in the vessels. Racle, writing in 1849, described a form of gangrene with nervous phenomena (5). He did not draw attention to the symmetrical nature of the affection, but he did not complete the writings of the papers in gangrene. His brother, however, informed Raynaud that he had been much impressed by the symmetry of the lesions (6). An interesting case, which Raynaud appears to have overlooked, was published in the same Journal as Racle's memoir. It is that of a Mulatto in Brazil, recorded by Rapillaud (7). Monro has given a historical sketch of cases prior to the publication of Raynaud's thesis which approximated to Raynaud's disease or were actual examples of it (*7a*). While Raynaud himself quotes in his thesis cases described by Lachmund (1676), Hertius (1685), Bocquet and Molin (1808), Rognetta (1834), Godin and Portal (1836), Topinard (1855), and Bernard Henry (1856). It is clear, therefore, that even the minor degrees of this condition had been noted before Raynaud wrote, but it was left for him to make the generalisation and to marshal the facts in an orderly way.

In enumerating the predisposing causes, Raynaud notes that those chiefly affected are persons of a "nervous or lymphatic" temperament, and in the history of the patient he lays stress upon the influence of neuroses, especially hysteria and epilepsy. Several cases which I have had the opportunity of observing—among them, three soldiers who have been in active service—and a brief survey of the literature of the subject, have served to strengthen the impression of the importance of the nervous element in the production of this disorder. To deal with the condition from all points of view, even in a cursory manner, would entail the expenditure of much time and energy; it may

not, however, be without interest to gather together a group of symptoms illustrating one aspect of Raynaud's disease, and to note the cerebral disorders which may be associated with it.

"*Hysterical*" *Symptoms.*

Among Raynaud's cases there are some which exhibited markedly the "hysterical" element. One (Observation 2), a young woman, æt. 25, who suffered from local syncope, had "quelques attaques de nerfs, avec perte de connaissance, sensation de boule, pleurs et rires involontaires." A young soldier (Observation 3), also with local syncope, when electrical treatment was tried became hysterical, "Larmes, cris, désespoir, sans perte de connaissance." Raynaud notes the predominance of the nervous element in this instance. In another case (Observation 8) which he quotes from Landry (8), that of a young woman, æt. 22, the symptoms were still more marked. With the local asphyxia there was such intense pain that the slightest touch produced convulsions. Later in the attack the pains recurred, and the convulsions became so marked that it was necessary to restrain her "on était forcé d'attacher la malade." He notes that there was no *globus hystericus*. The fits, which continued for nearly two months, were not apparently epileptic but hysterical. Nine months later, when she was pregnant and had improved in health, her husband died, and she had violent convulsions followed by "délire nerveux" which passed in three days. Another interesting symptom—which will be referred to again—was noticed at this time, namely, "complete aphonia." At times there was analgesia of almost the whole of the body, without anæsthesia. Later in the attack she attempted suicide by taking laudanum; she became comatose but rallied and recovered from this. She is stated to have had no recollection of this incident. She improved for a time, but died later of phthisis. Raynaud, commenting on this case, remarks that "the hysterical convulsions always accompanied the paroxysms of pain in such a way that it is legitimate to place them to the account of the latter . . . in proportion as the malady developed and became established, the nervous symptoms became generalised and aggravated, aided by moral emotions of which the influence was undoubted" (9).

Observation 9 deals with a female, æt. 30, who had, when a

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child, lived in bad hygienic conditions and been maltreated by her parents. She gave a history of hysterical symptoms at about the time of puberty, and since that time she had had hysterical attacks at times of emotional stress. At the age of 27, as a result of a fright, she had amenorrhœa, dead fingers, and local asphyxia. Her condition varied, but generally became worse; in addition to the Raynaud there was profound cachexia. As a result of hearing of her father's death she had a violent "nervous attack," and was insensible during a whole night. There was slow but progressive mental enfeeblement, cerebration slow, and she was easily fatigued mentally; there was steadily increasing difficulty in hearing. This case ended fatally.

Observation 15, a female, æt. 27, had local asphyxia in hands, feet, and nose: dry gangrene of extremities. After weeks of acute pain and loss of sleep there were three days in which she was subject to disorientation for about ten minutes on waking. This was followed by hysteriform attacks, throbbing in the head, and a feeling as if she would fall.

In case three of Raynaud's "New Researches" he notes a sudden loss of consciousness associated with left hemiplegia.

Hale White considered a case of Raynaud in a girl, æt. 16, an orphan who had been much neglected, as being "decidedly hysterical" (10).

The term "hysteria" is generally a rather unsatisfactory one. Frequently it appears that symptoms which do not fall into more definite categories are heaped together under the name of hysteria. Rheumatism and hysteria have more offspring fathered upon them than they can reasonably claim. Raynaud may have been right in calling the seizures in, for instance, Observation 8 hysterical: but it is not unreasonable to suggest that they might have been to a considerable extent spasmodic, and secondary to the vasomotor condition. Monro appears to be of this opinion in regard to Observation 15. The "hysterical phenomena," he says, "seem to have been due to the exhaustion and pain caused by the vasomotor disorder in one who had previously been neurotic" (11). It has been said that "hysteria may mimic all other maladies": but the same writers remark that the greatest danger arises from failing to appreciate the limitations of hysteria and allowing its presence to interrupt careful search for organic disease of which it

may be a secondary expression" (12). It is more necessary to heed this warning in such a condition as Raynaud's disease, where there undoubtedly are in certain cases changes in the cerebral circulation. Until, however, we know the etiological factors in hysteria and epilepsy, discussion of details is likely to be barren of results. Nor is knowledge much further advanced when we come to consider the "congestive attacks" and the seizures of general paralysis of the insane, infantile convulsions, or the fits of chronic alcoholics. Are the fits primarily associated with the nerve-cells or are they secondary to changes in the circulatory system? To this we have no satisfactory explanation.(3)

The convulsions of an hysterical nature already mentioned shade almost indefinably into seizures of a more obviously epileptic nature associated with Raynaud's disease. Nor is this surprising when we realise how difficult it is to draw any distinct line between the various forms of convulsions. Nature does not proceed by leaps and bounds. The hiatuses described are blanks in our knowledge rather than definite intervals between one series of facts and another; and, however convenient they may be for purposes of description, they are inimical to a proper perception of the uninterrupted processes of Nature. So it is difficult to decide where in the present instance hysterical phenomena end and those of epilepsy begin. In Raynaud's nineteenth case, that of a female, æt. 32, in whom were observed local asphyxia and gangrene, there was a history of "epileptiform" attacks; and Raynaud's justifiable comment is that, "les antécédents epileptiques de la malade doivent ici entrer en ligne de compte." In this case also there was apparently moral defect, for he tells us that she was arrested in several cases for vagabondage. Southey has recorded a case of a child æt. 2½, who died thirty-two hours after the onset of the disease. Convulsive attacks occurred two hours before death, increasing in frequency up to the time of death (13). Thomas had a patient, a man æt. 26, who had local asphyxia of the ears and dead fingers; at this time he had the first convulsion. During the following summer he was better. In the winter there was local asphyxia of the ears and syncope of the hands and feet. There were then convulsions every day and as many as three in the day. "Whenever he went out in the cold weather, a fit was certain

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to come on. The attacks always began with a chilly feeling, as if cold water were being poured down his back; this sensation lasts long enough for him to find a place upon which to lie down; he then loses consciousness and becomes convulsed. The attack lasts about half an hour, and after it he feels weak and has headache." The local syncope occurred at times in this case without fits being associated with it. The fits did also take place during cold wet weather in summer. After three winters the convulsions ceased but the other symptoms continued; while after another three years, when there was asphyxia of nose, ears and fingers, there were no further convulsions, but abdominal pain (14). Osler describes an anomalous case, that of a German girl, æt. $13\frac{1}{2}$, who came of a neurotic family. At ten years of age she had (?) chorea: "This was followed immediately by three groups of symptoms, *viz.*: painful swelling of the legs, painful swelling behind the left ear, and falling attacks. . . . At first, and for a year, she would fall forward, two or three times a day, on her hands and head, and, unless caught, would roll on the floor. There was no sound, no convulsion, and probably no loss of consciousness, but of this she is not sure. For the last two years she has always had time to get to a chair or lounge, never loses consciousness, and it never lasts more than five minutes, and often only a few seconds. During these attacks she feels faint and powerless" (15). There is here possibly a condition less marked than in the definitely convulsive cases already mentioned: and one analogous to minor epilepsy. In another case, a little girl who had a condition allied to Raynaud's disease—cyanosis of both lower limbs nearly up to the knees—there were "some ill-defined epileptoid attacks followed by some paresis of the lower limbs" (16). Colcott Fox narrates a case, a woman, æt. 41, "of spare habit and with an anxious face and intensely nervous temperament." He said that she was a "dreadful sleeper," especially after the slighest worry or excitement, and she had had several severe hysterical attacks on similar provocation. He describes her as "excessively nervous, hysterical and emotional" (16*a*). Levi and Raymond describe the case of a woman, æt. 43, who had Raynaud's disease of hands, feet and forearms. She was "undoubtedly hysterical," and she had convulsions, polyuria, etc. Another case they describe as hysterical and easily

hypnotisable (16*b*). Solis-Cohen, dealing with an analogous—if not a similar—condition, acroasphyxia, describes the case of a woman, æt. 25, who suffered from constant headache for three or four months and who had occasional attacks of dizziness. In another case by the same author, a woman, æt. 19, who was "easily excited," there were mild epileptic attacks. In a third case, in addition to other symptoms there was on one occasion a "transient partial obscuration of consciousness" (16*c*).

In a case of Osler's he records occasional attacks of "dizziness and transient obscuration of consciousness," paresis and aphasia.

Epilepsy.

Raynaud's disease has been noted in several cases in association with definite epilepsy, and with epileptic insanity. Here the local symptoms have been subsequent to more or less prolonged epileptic trouble. Case 19 of Raynaud's thesis, which has already been referred to, would probably fail to be included in this category. In a case of Féré's (17) he was unable to obtain a definite history as to which condition preceded the other. It is that of a man, æt. 48, who had his first epileptic fit at 41, and who had had twelve up to the time when Féré saw him. The patient stated that they all occurred during the winter; but more frequently he had had attacks of vertigo with loss of consciousness. He also exhibited a curious condition, a disseminated asphyxia over the body. (In a case of symmetrical congestive mottling of the skin reported by Cavafy there were no cerebral symptoms) (18). Féré came to the conclusion that as the epileptic manifestations and the circulatory disorders seemed to have developed about the same time, and as they both showed marked predominance during the winter, it was permissible to establish a relationship between them. If this were so it would, as Féré points out, lend support to the angio-neurotic or sympathetic theory of epilepsy, a theory attributed to Schneevogt and also to Charles Bell.

Bland recorded a case, a man, æt. 23, who had epileptiform seizures for ten years previously and who, at the time when he developed the symptoms of Raynaud, was acutely maniacal (19). I am able to add to this the case of a man æt. 35, who had had epileptic fits for at least seven years. He had

recurring attacks of epileptic furor; he was at these times violent, intractable, and destructive; and, also, during the phase of excitement, he was much influenced by auditory hallucinations. During a maniacal phase he developed an inflammatory condition of the forehead which was suggestive of erysipelas, but which was not definitely so: this descended downwards to the face, and passed off in the course of a few days. After it had lasted two days local asphyxia of both feet was noticed which subsided, except from the toes, which were gradually becoming more gangrenous when death resulted. The feet had been exposed to cold about this time, as the patient tore all his clothing off and would not allow cotton wool and bandages to remain on his feet.

Bernstein has reported a case of Raynaud's disease associated with epilepsy (20). In Wiglesworth's case, an epileptic and insane woman who suffered from chronic Bright's disease with secondary hypertrophy of the heart, there was gangrene of fingers and one great toe. In addition she had peripheral neuritis in all four limbs (21).

Mania and Melancholia.

The occurrence of the symptoms of Raynaud's disease have been noted in association with epilepsy, and with the maniacal phase in epileptic insanity. They have also occurred fairly frequently in mania not dependent upon epileptic excitement, in melancholia, and in the depressed phase of manic-depressive insanity. In a case of Southey's, a boy, æt. 9, in whom gangrene of the right index finger developed, there were maniacal symptoms. He was nervous, excited, cried constantly when examined or spoken to; and he was noisy, especially in the evenings (22).

Barlow says that Southey informed him that "since the publication of this case he had seen several examples of Raynaud's disease in asylum cases": the type of case is not specified (23). Edgerley gives a case, a woman who had been in an asylum "for some years," and who during the early part of her stay had long periods of comparative sanity. During the maniacal attacks she was noisy, incoherent, destructive, and covered herself with dirt. Whenever the mental relapses occurred, her hands showed characteristic signs of Raynaud's

disease. At first local syncope and then local asphyxia. During remissions of mental symptoms her hands resumed their normal appearance. (The same author records a case which may be compared with Féré's. A woman, æt. 37, who had had three previous attacks, developed ecchymoses over her body and limbs at the height of the excited period. Such a case, though not one which Raynaud would have included in his category, is interesting from the point of analogy. It might be more properly associated with the cases of "stigmata" upon which much stress is laid by many of the devout: more especially as she had delusions that she was being crucified and stoned) (24). Macpherson relates the case of a girl in whom the vasomotor symptoms set in four days after her admission with acute mania. At the outset, however, a week before admission, there had been depression with suicidal impulses instead of exaltation (25).

In manic-depressive insanity the symptoms of Raynaud's disease may appear either during the maniacal or during the depressed phase. Ritti noted in his cases that it was during the stage of depression. Esquirol (26), among others, has called attention to the alteration in the peripheral circulation which occurs so frequently among the insane. In Ritti's first case, a woman, æt. 28 on admission, there is a history of mania for a number of years with occasional remissions. Later, definite alternations are noted, each phase lasting from ten to fifteen days, and at this time there were lucid intervals between the attacks. After a time, however, the attacks of mania and melancholia followed one another without intermission. Frequently during the depressed phase she had local syncope and local asphyxia of the fingers of both hands, but always one hand at a time and only certain fingers. In his second case, a woman æt. 41, there had been an attack of acute mania, at the age of 27. This lasted for three months, and she then became depressed. After this she was normal for a time, then for several years there were periodical attacks of boulimia lasting about eight days. From the age of 35 and onwards excitement and depression followed one another without any intervals. When the symptoms of Raynaud appeared it was in the form of local syncope and local asphyxia of the hands and feet. Ritti remarks that he never observed them during the maniacal phase (27). In contradistinction to this I have observed a case of

manic-depressive insanity in which there was local asphyxia, and later symmetrical gangrene of the toes and of the tissues for about an inch and a half above them. This was in a woman, æt. 37, who had six months prior to admission been depressed and had attempted suicide. On admission she was maniacal, noisy, restless, incoherent in speech, sleepless, unclean in habits. The symptoms of Raynaud's disease appeared after about three weeks of this intense excitement. In another case there was a history of two previous attacks of mania, but no record of any pronounced periods of depression. She remained acutely maniacal for two months after admission. Her left forearm and hand were first affected with swelling and then discoloration; this disappeared and then the right leg and foot were attacked. Next the left lower limb and side up to the axilla were affected. Later, symptoms of gangrene appeared in the feet.

Urquhart recorded a case of Raynaud's disease in association with melancholia. The patient was a woman, æt. 50. There was a history of one sister having had puerperal mania, and another sister was said to have committed suicide. The melancholic symptoms had lasted for three years: she was restless, deluded, had ideas of impending ruin and of her unworthiness; she was also suicidal. Two years prior to the symptoms of Raynaud's disease—local asphyxia in both feet—she developed left hæmatoma auris (28). In a case reported by Shaw there were melancholic symptoms, delusions of poisoning, and suicidal tendencies (29).

Targowla tells of a man, who, at the age of thirty-six, began to suffer from melancholia with suicidal impulses and insomnia. Raynaud's phenomena began about a year earlier than the mental symptoms. According to Targowla the attacks of melancholia and of local asphyxia did not appear to exert any influence on one another (30).

Another of Urquhart's cases exhibited symptoms more suggestive of dementia præcox. It was that of a man, æt. 33, who had always been "nervous and flighty." Seven years prior to admission he had yellow fever. He became mentally confused, made mistakes, and was unable to work. He was obstinate and resistive; he was depressed and had hypochondriacal ideas, such as, that he could not straighten his legs, that he had no feet or stomach. He was at times excited and

restless. He had a habit of crawling on the floor, squeezing into corners or underneath furniture, and when thus placed lay stark and still. He was persistently wet. He developed hæmatoma auris on both sides, and later local asphyxia of both feet. Later there was gradual exhaustion and death (31). I have observed a case of dementia præcox with marked negativism, restlessness, apathy, and progressive mental enfeeblement, who developed local asphyxia in both feet with patches of desquamation. He was at that time æt. 25, and his mental symptoms had lasted for several years. Another case, a female, æt. 23, was stated to have begun to show mental symptoms at 21. She gradually became mentally enfeebled; she was confused, unable to converse, and childish. She was at times excited, restless, violent, occasionally thought that she was going to be burnt. She developed recurring local asphyxia of both feet.

The following cases I have had the opportunity of observing at the County of Middlesex War Hospital among soldiers who have been on active service: Pte. A. B—, æt. 19, went to France in April, 1915, and was in the trenches and under fire. In October he was sent into hospital with "bad circulation." It was reported that on parade he did not appear to understand the orders given, and sometimes wandered from the ranks. He complained that things were stolen from him. He rambled about at night, and was much influenced by auditory hallucinations. He was depressed and wept. He was deluded and said that "chloride of lime was being sprinkled over blood in the next room." He was dull, stupid, and confused, cerebration slow, memory defective. He had typical recurring attacks of local syncope and local asphyxia in the fingers and toes. He improved steadily and became bright and cheerful. After the mental improvement had become pronounced the circulatory troubles still continued, and later he had an attack of aphasia to which reference will again be made.

Private C. D—, æt. 21, went to France in January, 1916; he was in the trenches for five days. Whilst there he was heavily shelled but was not struck or buried. On the last day which he spent in the trenches his officer was shot beside him. He was much shaken, and later was not able to recall events about this time very accurately. He became depressed and was sent into hospital. He was at that time nervous, emotional,

and depressed. He stated that when alone he felt as though someone were following him. He did not hear voices but he felt as if "things were put into his mind." He said that on the day before his admission to hospital in France, he felt that he wanted to jump into the water. On his return to England he was brighter, but still nervous and rather tremulous. The noises in his head he compared to the ringing of bells. For some time he had suffered from headaches, at nights he had "visions which were not quite dreams."

He had local syncope and local asphyxia of the first and second fingers of both hands, and these symptoms recurred from time to time. He had noticed this condition for years. He believed that it came on after he had acute rheumatism and chorea. He said that his mother suffered from a similar condition. Vision impaired; he had observed a progressive weakening of his sight for some years. The right pupil was slightly larger than the left.

This patient gave an extensive history of nervous instability in his family, and of a tendency to vascular degeneration. On the paternal side, his great-grandfather had hemiplegia, grandfather very excitable, became "mad if he took drink," eventually had a seizure and died, grandmother suffered from violent headaches, was of a very nervous temperament, markedly hypochondriacal; a cousin is mentally deficient. His mother is nervous and hysterical. His brother is very excitable, and at times becomes confused and forgetful. This patient himself is sallow-complexioned and nervous. He says that he was troubled by "voices" when he was about 17 years old, and that about the same time he attempted suicide. He says that he was subject to convulsions as a baby.

Private E. F—, æt. 40, exhibited a local condition which approximated to Raynaud's disease, but which might be more accurately described in the category of acroasphyxia. Both hands were from time to time cold and almost syncopal, and this was succeeded by the asphyxial state; in the latter phase the hands looked as if they had been dipped in a solution of indigo. He gave an interesting history. He had had fifteen years' service. In 1906 he was operated on for "tumour" of the left testicle, and the testicle was removed. Just after this he had a "fit," and another one a year later. During these fits he lost consciousness. In 1908, while he was in India, he

had malaria, and it was after this that he first noticed the change in his hands. In May, 1915, he was in France, and was buried by a shell explosion. He had a similar experience later in the year, in September, and he gave a history of gas-poisoning about the same time. In November he went to Salonika. Whilst walking with a friend there he fell down suddenly and was unconscious for about twenty-four hours. He was sent back to England, and on his return was depressed, nervous, and tremulous. At times he was incoherent in speech. On two occasions he attempted suicide in what is described as "an ostentatious manner," and he is reported to have had some fits of an hysterical nature. Since I have seen him there have been no further fits or attempts at suicide. He has, however, been depressed and plaintive. He complained very much about his digestion, and said that he had been unable to retain any food, but this was not noticed after his admission. Gradual improvement took place mentally and physically, but at the time of writing there is apparently some mental reduction, and he is facile and more satisfied with his capabilities than his state warrants.

Mental Enfeeblement.

In certain cases the symptoms of Raynaud's disease in insane patients have been noted after mental enfeeblement had become pronounced, although it does not occur in demented cases to the extent that one might, *à priori*, imagine when the sluggishness of the circulation in such patients is considered. There is apparently some other factor, it may be the involvement of certain parts of the nervous system. Iscovesco has noted the occurrence of local asphyxia in three cases (females) with confirmed general paralysis of the insane (32); Hutchinson in a case of congenital syphilis with defective mental development (33).

The majority of Zambaco's cases which exhibited symptoms of Raynaud's disease were general paralytics (34). A case of Barlow's, a middle-aged woman, during a slight remission of her attacks of local asphyxia, became the subject of delusions which were always worse in the evening (34*a*). Ibotson relates a case, a woman, æt. 40, who suffered from phthisis, and exhibited progressive mental enfeeblement and who developed Raynaud's disease. The mental symptoms

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had lasted six years. She had the delusion that she would find a fortune in a water-closet, and she was continually putting both her hands down to pull it up. She was also influenced by auditory hallucinations (35). Case 16 of the Thesis, though not a case of dementia, was one of profound physical and mental enfeeblement. It related to a man, æt. 34, who had gangrene of fingers and toes. "Le visage, ainsi que tout le corps, était d'une paleur mortelle: les yeux étaient fixés ou roulaient languissamment dans leurs orbites, puis s'arrêtaient comme ceux d'un idiot, ou comme si l'esprit affaibli du malade avait été frappé par quelque objet effrayant" (36). Pitres and Vaillard record a case, a young woman of feeble intelligence from childhood. At 18 years of age she began to suffer from tremors and stiffness of the limbs until at length walking became impossible; the lower limbs passed into a state of extreme contracture, and the patient was bed-ridden and demented. Gangrene of the feet developed, and eschars on the body. *Post-mortem* there were discovered chronic hydrocephalus of the lateral ventricles, undue adhesion of the pia mater to the cortex of the hemispheres, and great thickening of the skull. There was also diffuse sclerosis of the dorso-lumbar part of the cord (36*a*).

Aphasia.

The occurrence of aphasia in connection with Raynaud's disease is rare. He drew attention to it in his eighth case, to which reference has already been made. From time to time there was a complete loss of speech with inflammation, laryngeal pain, cough, or expectoration. "Cela lui arrive souvent, presque tout à coup, et dure quelques heures ou quelques jours." He noted that no other nervous symptoms accompanied this loss of speech: and he appears to have thought it a part of the hysterical condition: "La malade prétend que c'est par faiblesse qu'elle ne peut pas parler, bien que rien ne dénote une faiblesse générale plus considerable en ces moments" (37). One of the most interesting cases with this symptom is recorded by Weiss. It occurred in a woman, æt. 35, of neurotic tendency both personally and by heredity. The disturbance of speech began suddenly. The patient became pale at the same time: her lips were pale, and

the retinal arteries narrowed to a striking degree. She had difficulty in pronouncing familiar words, and she transposed words or syllables and used wrong ones. Voluntary movement not affected. The attack was at an end in fifteen to twenty minutes, speech being quite restored. A similar attack occurred four weeks later (38). In Osler's case, already referred to, a woman, æt. 47, there were occasional attacks of numbness and mottling of the fingers for five or six years. Then she began to have dizziness and transient obscuration of consciousness. This occurred on three occasions. About a month after the third attack there was aphasia, paralysis of right hand, and paresis of right foot. From all these she soon recovered. Four weeks later she developed complete motor aphasia and spasm of the right hand. In less than a day these passed off. Two months after this there was headache, left hemiparesis, discoloration and tenderness of right hand. This was in February. In July of the same year she had a third attack of aphasia with right hemiplegia. At this time there was local syncope and asphyxia of right hand and fingers, and the tip of the nose was blue. In July of the following year she had again the giddiness and vomiting. This was followed by intense pain in the right hand: the fingers blue and the hand anæsthetic. Speech was on this occasion retained. She gradually became comatose and died (39). Stockman has described a case of a woman, æt. 26, who had local asphyxia of both feet and of the left forefinger, and of the nose and ears. On three occasions she lost the power of the whole of her left side. She could not move her arms or legs and could not speak: on one occasion she felt as if her tongue were fixed and immovable. This lasted about five minutes each time, during which she remained perfectly conscious. On several occasions she had temporary loss of memory during conversation. She expressed it as forgetting what she intended to say. "It was not inability to express her thoughts in words, but a lapse of memory as to what she was going to say. On each occasion it only lasted a few minutes" (40). In Simpson's case, a woman, æt. 60, in whom the symptoms of Raynaud's disease had appeared at the age of 48, there were giddiness and faintness, some paresis of the left arm and of the left leg, and slight aphasia. She was strange and dazed: she had left-sided headache and her sight was dim. The speech defect

lasted till the following day. There was also hyperæsthesia for the taste of sugar (41).

In the case of Private A. B—, to which I have already adverted, aphasia occurred after the symptoms of depression, the delusions, and the auditory hallucinations had passed. He was a bright, intelligent youth, and did not seem in the least hysterical. The aphasic state began on the Saturday morning. He said that he was trying to say something and could not. He had "felt a bit strange in the morning but could speak": and on the day before it seemed to him "as if things were muddled up. He felt limp, and as if he was going to be ill." He could understand what was being said to him but could not reply. He played the piano during the aphasic period, and there were no hemiplegic symptoms. Speech returned suddenly during the following Wednesday night. He said that "he sat up in bed and spoke": and he continued able to speak thereafter. He stated that the muscles of his throat felt stiff and painful. He thought this was because of the efforts he had made to speak.

The association of aphasia with Raynaud's disease is an interesting and rare one. To look upon it as an hysterical aphonia is unsatisfactory. We do not know what hysteria is in spite of elegant explanations—and to explain one incomprehensibility by another advances knowledge little. There may be a figurative and ironical meaning even in the term itself: perchance he who coined the word intended to convey that the true explanation of the condition was hidden in the womb of time! If so, parturition does not yet appear to have taken place.

The explanation given by Weiss seems plausible. He thought that the aphasia was due to a "spastic ischæmia in the region of the third left frontal convolution." A similar condition in other regions of the brain might account for other symptoms. Simpson suggested the varying symptoms in his case could "only be accounted for by corresponding attacks of cerebral anæmia or congestion affecting different areas of the brain." "They are," he adds, "compatible with no single lesion." A case recently recorded of injury in the neck which necessitated ligature of the common carotid artery on the left side is interesting in this connection. The patient "lay in a dull, heavy, stupid condition for twenty-four hours.

After that he gradually seemed to understand what was said to him, but was quite unable to put any of his thoughts into words for several days. . . . The mental dulness rapidly improved, but recovery from the aphasia was a much more gradual process." He was not able to articulate for fourteen days, and then only a few words. Slow recovery of the power of speech followed. There was paresis of the right side for two or three days.(4)

Ocular Symptoms..

In his later researches Raynaud paid much attention to the ocular symptoms. One case, a man, æt. 59, became subject to local asphyxia of the extremities, and a few weeks later to paroxysmal impairment of vision. During the period of asphyxia he could see quite well, but as the digits were recovering their normal colour the sight, especially of the left eye, became dim. Vision was restored at the moment when a new attack of asphyxia supervened. Ophthalmoscopic examination in the period when the discoloration of the extremities was at a minimum revealed narrowing of the arteries and pulsation of the veins. During the period of cyanosis the arteries did not regain their normal calibre, as might have been expected, and the venous pulsations persisted. Yet the cyanosis of the extremities and the visual troubles alternated so regularly that the diminution of one "infallibly announced the appearance of the other, and this many times in the same day." The second case, a young man, æt. 22, was admitted for boulimia and polydipsia, and had cyanosis of hands and face. "At the same moment when a paroxysm commenced, he experienced a notable obscuration of sight, but when the cyanosis passed off, vision was restored." There was narrowing of the arteries during the cyanotic period, with restoration of calibre when reaction set in. The retinal veins were not observed to pulsate (42). Bland's case had for a time dimness of vision and inability to read, with unusual pallor of the fundus. Morgan noticed a narrowing of the retinal arteries though vision was good. After recovery from the Raynaud's disease and after three months' good health, the patient suffered for a time from severe headaches with simultaneous dimness of vision (43). Stevenson has reported a case in which there was on one occasion complete loss of sight for some minutes and at several

other times dimness of vision (44). In a case of Hutchinson's there was iridoplegia. Both pupils were quite immobile, the left was larger than the right. There was gangrene of the nose and left ear. Simpson's patient, it has already been noted, suffered from dimness of sight. Calmette states that in three malarious patients who had local asphyxia, there were also ocular troubles (45). In one of Solis-Cohen's cases, vision was at times misty, in another there was sudden dimness of vision, progressing in the course of a few minutes to total blindness which lasted about a second. This transient blindness recurred, but affecting only one eye.

Private C. D—, whose case has already been referred to, noticed that, in association with the local syncope and local asphyxia, there was increased dimness of vision, particularly in the right eye.

Headache.

Headache is a not infrequent concomitant. It has already been referred to in the cases quoted from Osler and Simpson (where it was left-sided). Wood speaks of a case, a man, where there were attacks of localised pain similar in character to the pain felt in the fingers. It sometimes accompanied and sometimes alternated with the pain in the extremities (46). In the case of Private C. D— there were troublesome generalised headaches. Solis-Cohen noticed in one of his cases of acroasphyxia that there was, associated with visual trouble, intense headache lasting about ten minutes, and in another constant headache for three or four months. In a case of Vulpian's there was occipital headache.

Paretic (Hemiplegic) Symptoms.

These have been noted in several cases. In Raynaud's third case (Thesis) there was apparent paresis of the right arm. In Case 3 of the New Researches there was hemiplegia of the left side lasting for two hours.

In Simpson's case, associated with the aphasia, there was left-sided hemiplegia; a similar condition was recorded by Stockman. Osler noted at one time affection of the right side with aphasia; on the second occasion, of the right hand again with aphasia; thirdly of the left side but without aphasia; and

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at the fourth attack right hemiplegia again associated with aphasia. In the case recorded by Weiss there was diminution of motor power.

Raynaud believed that the motor symptoms were due to defect in the afferent impulses rather than to muscular weakness.

With the hemiplegic symptoms one may associate the condition of the intermittent limp where the patient "after a few steps becomes unable to walk farther, owing to intolerable pain in the muscles of the leg" (47).

Erythromelalgia and Raynaud's Disease.

There is, at times, some confusion in regard to the two conditions. Yet Weir Mitchell, who first described erythromelalgia (1872), remarks that it is inconceivable that these two disorders should ever have been confused, and a glance at his categories of symptoms certainly supports this statement. In erythromelalgia, or "red neuralgia," there is flushing and local fever; in Raynaud's disease the part is either bloodless or dusky and congested, and there is lowering of temperature. In the two cases with which he illustrates and contrasts these conditions there were noticeable nervous symptoms. The case of Raynaud's disease was a woman, æt. 30, pale, nervous, and excitable, who, after a long strain and much work, developed local syncope and local asphyxia in the fingers. The erythromelalgia was in a man. In the course of the disease he became silent and morose; there was paresis of leg and arm; he had nine attacks of convulsions—"or rather of rigidity." He became "more or less hysterical," gradually bed-ridden, had "queer, indescribable feelings in the head," and he disliked all mental effort (48). Although there is dissimilarity in the symptoms there may be some relationship in pathological bases. Weir Mitchell at first inclined to the view that erythromelalgia was due to "some form of spinal disorder," later he considered peripheral neuritis as a possible cause. In Wiglesworth's case of Raynaud's disease there was neuritis in all four limbs, while Pitres and Vaillard held that most of Raynaud's cases of gangrene were caused by peripheral neuritis. Bramann recorded some interesting cases which were possibly related to both Raynaud's disease and to erythromelalgia. In three brothers,

æt. 7, 10, and 13 respectively, there began in each at the fourth year of life a condition characterised by violent pains, great redness and swelling. It attacked almost symmetrical spots on the extremities, and most of these spots proceeded to the state of gangrene. Bramann considered that the symptoms pointed not to vascular but to spinal disease, and he suggested syringomyelia as the most likely cause (49).

Conclusion.

The time has not yet arrived when it is possible to say what is the exact condition in the nervous or vascular systems which give rise to the varied symptoms of Raynaud's disease. This being so it is not surprising that various theories have been promulgated to account for them. Some of the suggestions are rather of the nature of explaining one symptom by another. For example, when the localised headache in a case of Raynaud's disease is said to be due to "localised meningeal congestions," one is still left to discover the cause of these congestions. Raynaud inclined to the view of the central nervous origin of the symptoms. "The marked symmetry of the lesions," he wrote, "ought to suggest that they originate in a discharge either spontaneous or reflex, starting from the cord and radiating thence to the vascular nerves of the extremities" (50). Barlow sums up as follows: "The last development of Raynaud's doctrine . . . is that there is a peripheral excitation, most commonly consisting in an impression produced by change of temperature in the cutaneous nerves, and that whilst in a normal state either very low temperature or exposure for a long period are necessary for the production of more or less analogous effects, in these individuals an insignificant difference is sufficient; further, that the peripheral stimulus affects that part of the grey matter of the cord which presides over the vasomotor innervation, and that a great exaggeration of the irritability of that part of the cord must be assumed . . . Now given the initial slight peripheral stimulus there seems no reason why the central disturbance should not radiate and become manifest in several different regions successively instead of simultaneously" (51). Monro, whose admirable monograph on this condition has rendered all students of it his debtors, thinks that "the phenomena of Raynaud's disease must be brought

about through the agency of the nervous system." As to the particular part involved he says: "Accepting then the theory of an increased excitability of the vasomotor centres in the cerebro-spinal axis, allusion must be made to the situation of the unduly sensitive centres. Raynaud is undoubtedly correct in saying that the part of the cord varies in different cases. The varied distribution and the occasional unilateral character of the symptoms suggest that the disturbance is in the subordinate vasomotor centres of the cerebro-spinal axis. This is doubtless specially true of cases that originate in consequence of severe exposure. On the other hand, cases that are due to emotion have their source in cortical disturbances, and these will, no doubt, operate through the principal centre in the medulla. If a subordinate centre in a given limited area has once been rendered over-excitable, through exposure or otherwise, cortical discharges, such as those connected with emotion, may at any time call forth paroxysmal overaction limited to the over-sensitive region. The theory of a cortical starting-point for the vasomotor discharge is favoured by the frequent association with such functional disorders of the cortex as insanity, epilepsy, etc." (52). Purves Stewart suggests that "profound molecular changes exist . . . in the sympathetic system" (53). Lévi and Raymond lay stress upon the emotional factor. They think that the vasomotor phenomena in Raynaud's disease and in erythromelalgia have their origin in certain emotions which give rise to subconscious fixed ideas. Among their general conclusions are the following: (1) There is a form of Raynaud's disease which is purely hysterical. It may originate or reappear under the influence of a moral emotion or shock, it may disappear or be improved by hypnotism, but there remains a vasomotor system easily affected. (2) Acute rheumatism is frequently found in the antecedents of patients, and may determine the localisation of hysterical manifestations. (3) The onset is sudden, the origin emotional. The disease is psychical. (4) The central theory must be accepted, that is of a neurosis with localisation in the cerebro-spinal centres (54).

Those who incline to the so-called "psychic" agency in the production of such symptoms as are seen in Raynaud's disease would do well to remember that he looked upon those differing symptoms as degrees in one condition. So with the exciting

factors it is a question of the strength of the stimulus, but there is at the same time the response of the organism. Where the instability of the nervous system is so marked that it responds to minimal stimuli the tendency is among certain people to place the results in a category which is marked off from all others. Thereafter it is only a question of personal predilection as to whether they are labelled psychic, spiritualistic, or miraculous, or by those who, admitting in their very phraseology their incompetence to see, designate the facts as hidden or "occult," and then quarrel with anyone who endeavours to illuminate the dark places—and they are many—of their scheme of things.

Emotion as at least an exciting factor has been frequently noted. Noyes records a case, a woman of nervous temperament, in whom attacks were produced by cold and emotion. "Three or four separate attacks have been observed to occur in rapid succession whilst the patient was under examination . . . owing to emotional excitation" (55). In Stockman's case, "cold, mental excitement, worry, and slight traumatism" brought on the attacks (56). Colcott Fox notes regarding his case that "the extremities were affected in a second if she was startled by a sudden knock at her door or any unusual occurrence" (57). Solis-Cohen thinks that in certain individuals there is a congenital want of balance in the circulatory apparatus. "Mental or even physical shock in a subject of congenital vasomotor ataxia might cause the sudden development of exophthalmic goitre, and an exposure to cold from which a normal individual would quickly react may cause local asphyxia, chilblains, frost-bite, or even gangrene" (58).

In certain cases the cerebral symptoms are apparently secondary to the vasomotor changes. The ætiology of manic-depressive insanity is obscure, and the possibility of periodic vascular changes cannot be lost sight of. Ritti was strongly of this opinion. He thought it allowable to surmise that a similar condition might be taking place in the brain, that there is spasm of the cerebral capillaries, that the depression may only be the result of cerebral anæmia consecutive to this spasmodic contraction, and that finally the mania is due to re-establishment of the cerebral circulation, which in the phase of reaction may even be exaggerated (59). This was the opinion of Luys, who, speaking of this form of insanity, said that the phenomena

of alternating depression and excitement succeeded one another by imperceptible degrees; this is brought about by "la fatalité des lois de la circulation capillaire." According to him, excitement and despression are only "des variations dynamiques apparentes de l'état d'ischémie ou d'hyperhémie successives par lesquelles passe la trame nerveuse intéressée" (60). It is probable, however, that the explanation is not quite so simple as this, and other additional factors will doubtless be found to underlie these changes. Nevertheless, the mental symptoms which have been observed in association with Raynaud's disease may eventually help to elucidate the subject of the causation of mental disorder.(4)

In other cases the symptoms of Raynaud's disease have been subsequent to long-continued mental disorder. Edgerley remarks, "While in certain cases disorder of the circulatory system is a cause of insanity, much more often mental disorder produces circulatory disorder" (61). It seems probable from a survey of certain of the cases already mentioned that the gradual spread of disorder in the cerebral cells gradually involved those areas of the nervous system which preside over the vasomotor and other mechanisms. Cases of general paralysis of the insane frequently provide a dramatic illustration of the gradual spread from one area to another of the nervous system, and one may readily agree with Urquhart when he remarks of the cases described by him that they may be "correlated with cases of general paralysis where intractable bedsores occur in similar symmetrical disposition." It is not possible, however, to come to any decision on the matter at the present time. The mechanism of cerebral processes has yet to so great an extent to be discovered that the relative value of various factors, physiological or pathological, in bringing about disorder in the human economy cannot be decided. We do know that gross interference with the cerebral circulation can bring about rapid changes in brain function. Pressure on the common carotid arteries, such as is practised by Japanese wrestlers, speedily produces unconsciousness, and Lauder Brunton gives an example of how the same result used to be brought about before the introduction of chloroform as an anæsthetic by means of raising a person rapidly from the recumbent to the standing position (62). In these cases there is produced almost instantaneously the condition to which all insanity tends—abolition of cerebral func-

tion. If in such a condition as Raynaud's disease there is a more localised interference with cerebral circulation, it is legitimate to infer that function may be inhibited *pari passu* with the vascular involvement. A further inference would be that cerebral disorder involving those areas whose function is more specifically described as mental, may arise from the vaso-motor changes. On the other hand, it is necessary to remember that the cerebral and the vascular changes may be dependent on other factors such as a toxæmia or a deficiency in glandular secretions. Nothing but a patient study of all the factors involved is likely to lead to that stage at which it will be allowable to pass from hypothesis to explanation; it must suffice us for the most part to endeavour to gather the materials wherewith others shall build.

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(1) Read at the Spring Meeting of the South-Western Division, April, 1916, and awarded a Divisional prize.—(2) Hippocrates, describing the effects of what is described as epidemic erysipelas, notes that "in many cases both forearm and arm dropped off" (*Epidemics*, Bk. iii). Thucydides, in his description of the plague at Athens, says: "For the mischief, being first seated in the head, spread over the whole body, and if one survived the most formidable symptoms, an attack in the extremities manifested itself; for it was determined to the genital organs and to the hands and feet, and many escaped with losing them, and some with the loss of their eyes" (ii, 49). Lucretius, following Thucydides, describes those who were afflicted with the plague in the following terms: "The powers of the whole mind and the whole body grew languid, as if on the very threshold of death . . . the mind was distracted with anguish and dread; the brow was gloomy; the look wild and fierce; the ears disturbed and filled with noises." He tells us how some of the sufferers lost their hands and feet, the eyes, and the genital organs. "Upon some, too, came forgetfulness of all things, so that they knew not even themselves" (Bk. vi, 1159 *et seq.*).—(3) Vulpian records a case in which there were symptoms

apparently of an hysterical nature. Discussing the nervous symptoms he says that he does not consider them to be hysterical, but due rather to constriction of the vessels in the heart, the brain, and the medulla. "L'affaissement général, l'obnubilation de la vue, les vertiges, l'impossibilité de parler, etc., s'expliquent par un trouble cérébral" (*Gazette des Hôpitaux*, 1884, vol. lvii, pp. 65–66). Paget noted a case where, following cold bathing, there were local syncope and subsequently flushing and heat. Commenting on the probable condition of parts which are the seat of pain or other morbid sensations, in cases of spinal irritation or so-called hysteria, he remarks: "When such parts are out of sight, we are apt to think of them as changed in nothing but their nerve-relations. They are spoken of as only functionally disturbed, this implying that if we could see them they would appear in a perfectly normal state. It is more probable that their vasomotor, as well as their cerebro-spinal, nervous systems are, as in this case, affected; and that through the vasomotor influence they are in some cases anæmic and in some hyperæmic, or in both of these conditions at different times" (Sir J. Paget, *St. Bartholomew's Hospital Reports*, vol. vii, pp. 67–69).—(4) Targowla hazards the opinion that the melancholia and the local asphyxia in the case described by him may be due to the same cause—vasomotor disorder. "Lorsque ce trouble survient dans la circulation encéphalique, il se manifeste par un accès de lypémanie; le même trouble, localisé au extrémités, produit l'asphyxie locale intermittente dont souffre le malade" (*Annales Médico-Psychologiques*, 1892, vol. xv, p. 403).

On Shame. By JAMES RAE, M.A., M.D.

HERE we shall consider the effect of shame; then try to discover why we feel it at certain times, and whether it can be represented as the development of any other emotion.

I.

In the first place we must establish the physical signs and accompaniments of shame: The attitude changes slightly; there is a movement of withdrawal, a shrinking from notice. The eyes are averted or downcast, and the head droops. The face flushes, and the dilatation of the vessels may extend over the chest or even further; the pulse-rate is quickened. A tingling of the skin is next perceived as the vessels contract and the face pales. At the same time there is—though perhaps only momentarily—a confusion of thought.

Here is a physiological state. The vasomotor centre is actuated by the emotion, and the fibres must therefore have a connection with the frontal lobe. We know that the vasomotor centre lies in the grey matter of the floor of the fourth ventricle, and the fibres are believed to pass down the lateral tracts. The vasomotor fibres to the face are mixed up with the fibres of the seventh and ninth cranial nerves. It is, however,