PSYCHOLOGICAL IMPLICATIONS OF FUNCTIONAL CIRCULATORY DISORDER.*

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I HAVE been asked to speak to you about the psychological implications of functional circulatory disorder. Unfortunately there is no generally recognized system of the functional disturbances of the circulation. For this reason, and also for want of time, I shall be able to deal only with a few sections of the problem.

The functional disorders of circulation are generally classified according to the organ which they concern; their correlations within the total organism have hardly ever been studied in full. Vasomotor neuroses are known, but the vasomotor neurosis, as an entity, is only spoken of if its constitutional basis is discussed, e.g., in genetics or in work concerned with morphological anomalies of the capillaries (capillary microscopy). The inter-correlation of abnormalities of function between various vascular provinces has been investigated very little. Therefore it is practically impossible to draw any definite conclusions from observations of vascular function at the periphery as to what happens, for instance, in the brain vessels. If, therefore, the vasomotor neuroses with psychological symptoms are to be discussed, it can be assumed only with all possible reserve that something similar in kind to what can be seen in the peripheral vessels may occur in the brain, viz., vasomotor disturbances of some description. This, however, seems justified as a working hypothesis, considering the general medical tendency to attribute co-existing symptoms to one cause as long as there is no evidence to the contrary.

It is proposed to take as an example of the vasomotor neuroses Raynaud's disease, or better Raynaud's syndrome, i.e., paroxysmal local syncope, which may, or may not, lead to gangrene. There are a number of cases on record in which this illness was accompanied by mental symptoms. Heuck has calculated that 7½% of all cases show mental disorder. On looking through the cases published, with a view to finding out if, and how far, the cause or the mechanism of Raynaud's syndrome may be made use of to explain the pathology of the mental symptoms, it will be found that the psychiatric findings are well in keeping with Krapf's views. There have been found confusional states (Bland, Pechkranz) and depressive phases (Ritti, Macpherson,

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Shaw and Targowla, Nedopil, Heuck) coinciding in time with the vasomotor attacks ; in the case of which we have the best description (Heuck) there were also hysterical fits.

The fits accompanying vasomotor disturbances point to another aspect of the problem. Theoretically there are three ways of explaining this coincidence : (1) The hysterical symptoms are psychologically connected with the vasomotor crises, that is to say, the patient is aware of the physical symptoms and responds to this awareness in an emotional or tendentious way ; (2) an emotion produces a hysterical reaction, and, at the same time, a vasomotor attack in the *locus minoris resistentiæ* ; (3) similar or identical vascular crises co-occur in the extremities and in the brain, the latter producing fits. These are either (*a*) cerebral attacks *sensu strictiori* which only appear similar to hysterical ones, or (*b*) they disturb consciousness and lower the mental level to one on which hysteriform primitive reactions occur. Instead of testing the three possible modes of origin in Heuck's case, which has not been examined and described from this angle, it is proposed to demonstrate in other examples that all the possibilities outlined theoretically occur in reality.

(1) The first variant, the psychological explanation, appears to be the rarest one. In severe cases of Raynaud's and similar syndromes, the examiner is struck by the patient's remarkable capacity for putting up with the disease. It is true that the awareness of the mutilations often produces the emotions which one would expect, but gross emotional symptoms are rarely met with. Only occasionally neurotic mechanisms make use of these physical symptoms, and claims for compensation or other demands of similar psychological structure are based on them.

(2) The classical, one may call it the experimental example, of the co-occurrence of hysterical dissociation and vasomotor reaction are those cases in which the clinical picture of Raynaud's syndrome, or an intermittent local syncope, may be produced in hypnosis by suggesting emotional experiences. Such cases of so-called hysterical pseudo-Raynaud are numerous in the literature of some decades ago, when definition and differential diagnosis of this disease entity were discussed. It is interesting to note how this problem was settled by shifting the view-point : the patho-physiological (instead of the nosological) approach reveals the identical mechanism, which may be set going in various ways.

(3*a*) In order to discuss this group, organic discharges of cerebral vascular origin, it must be kept in mind, that there are cases of epilepsy in which the attacks start not only with paræsthesiæ, but even with true vasoneurotic pictures, such as " doigts morts ". Furthermore, one should not lightly discard the possibility of mistaking epileptic fits for hysterical ones and *vice versa*. The recently growing interest in the epileptic fit as such has enabled investigators to see how frequently atypical motor discharges can be observed in definitely organic epileptic states, which, therefore, may be regarded mistakenly

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as hysterical. Fits with rotation around the logitudinal axis, running movements, grasping and similar automatisms, alterations of typical discharges in the various stages of dissolution of the normal motor functions under the influence of posture and righting reflexes may only be mentioned here.

The question, however, arises—particularly from the patho-physiological standpoint—how far psychogenic discharges may use the same mechanism as organic ones, and how far they really and necessarily do so ; in other words, how far eventually the differences disappear owing to a final common pathway. In a particular group of the so-called affect epilepsies I have been able to demonstrate that this may be so.

(3*b*) Disturbances of consciousness produced by vascular disturbances in the brain, with consecutive hysterical reactions, can be discussed best in the light of psychoses accompanying migraine. Migraine is sometimes combined with Raynaud's syndrome. I saw recently at the Maudsley Hospital a woman who had been suffering from severe attacks of migraine for many years. She had all the typical symptoms, such as scintillating scotoma, fortification figures, unilateral headache, nausea or vomiting and post-paroxysmal euphoria. Besides, she described most clearly how these symptoms were preceded by tingling and numbness in the tips of her fingers, then blanching of the fingers followed without pain. This patient had one unusual symptom ; she complained that she lost her eyesight during these attacks. Since she apparently did not behave as if she were blind, she was regarded as hysterical. No attack was observed, therefore nothing can be said about type and intensity of the alteration of her consciousness during the attacks. That there was some can hardly be doubted from her vague and patchy recollection. Considering the whole setting of the case, this alteration of consciousness must be regarded as the basis of the hysteriform reaction.

Most impressive examples of mental disorder in connection with ***migraine*** have been collected by Mingazzini and his school. Apart from depressive states and attacks of catatoniform excitement, they observed psychoses of short duration characterized by obtrusive, complex, scene-like, visual and auditory hallucinations. The patients saw first the usual flashes, dots and zigzag lines ; from these developed figures of dead persons, black men ; wild animals, barking dogs leapt on them ; men armed with knives attacked them and threatened to cut their legs, etc. During these states consciousness was definitely disturbed ; recollection was vague and patchy. It would be superfluous to suggest any other explanation of the confusional-delirious state than its common causation with the migrainous attacks. This must be so, however hysterical these hallucinatory episodes appear to be, with their scene-like character, their obtrusive symbolism, etc. Their close relation to the physical mechanism is further underlined by their abrupt cessation with the end of the migrainous attack.

Fainting may be discussed as a different type of vasomotor disturbance,

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without going into the question of which particular type of vascular dysfunction underlies either of them. The psychological implications are of little interest, as there is a rapid onset of unconsciousness with loss of muscle tone; occasionally if the beginning is not quite so acute, something about the dissolution of sensation, e.g., of visual perception, may be learned from self-description. Instead I should like to discuss another state in the origin of which a reflex change of the tone of the cerebral vessels plays a part, viz., the *knock-out in boxing*. It hardly needs emphasizing that this technical term does not imply any medical uniformity. I shall not speak about the psychological accompaniments of the rare reflexes from the carotid sinus or the solar plexus, but I will only refer to the typical knock-out. By analysis of observation and self-description of knock-out after punches to the jaw, various mechanisms can be demonstrated as being of importance. Either singly or by interaction they bring about the state of unconsciousness called "knock-out". Direct mechanical action on medullary and di- or mesencephalic centres and the labyrinthine organ and reflexes from the vestibular apparatus seem to be the most important factors. The latter act either on the muscle tone or the vascular system, producing a fall of the blood-pressure. Furthermore, direct damage to the vessels may occur, as hardly needs mentioning. But it seems worth emphasizing that late effects can be observed which can only be explained by functional vascular disturbances due to a disturbance in the vessel tone following the trauma. I saw lately a boxer who developed a progressive hemiplegia a few days after severe blows to his temple, whilst in the interval only headaches and dizziness were present. This point is mentioned here only because one of the numerous psychological features of the knock-out seems to be closely related to functional-vascular disturbances, viz., the amnesia without fall or unconsciousness. This is what happens: the pugilist is hit at some moment; he does not fall nor go down, but keeps on boxing almost or apparently quite normally. Retrospectively, however, he reports a shorter or longer gap in his memory which often extends from the injury backwards in time (retrograde amnesia), and sometimes forwards to cover the end of the fight or even a period after it (anterograde amnesia).

There is evidence that this is not a hysterical repression of an unpleasant experience, since the same occurs after victories as well as after defeats. Furthermore, onset and end of, behaviour during and retrospective attitude towards the amnesia are entirely unhysterical. What really exists is a post-traumatic state of altered consciousness, very similar to an epileptic twilight state. If you remember what Dr. Krapf has said about the pathology of the twilight states, you will recognize this similarity as an additional argument in favour of its vascular origin. This assumption is furthermore supported by the occurrence of similar states of changed consciousness in patients with arterial hypertension, in whom the organic nature of the condition is sometimes proved by residual focal symptoms.

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J. Ch. D—, a bus conductor, aged 53, was sent to the Maudsley Hospital because he was suffering from attacks in which his legs became stiff. He also noticed that sometimes he lost control of his right hand and so was unable to punch the tickets correctly. He had always been rather quiet, but easy-going, generally happy, steady in mood, energetic, and had always plenty of friends. For the last two years he had gradually become moody and depressed and had lost interest. He had also shown increasing difficulty in understanding what was said to him.

He had " brain fever " when at school, and an accident twenty years ago. He has been married happily for thirty-six years and has three children.

On admission he had a blood-pressure of 150/100, the deep reflexes were very weak, the peripheral arteries (including those of the fundi) were thickened, otherwise he showed nothing abnormal physically.

He was co-operative and cheerful, and behaved normally ; in performing the usual intelligence tests he was somewhat slow, but showed no marked impairment of memory (apart from the amnesia after the attacks), nor any other gross signs of dementia.

From his description it transpired that he had an amnesia for a period of some length after what he called his attacks. He could state, for example, if questioned, at what point on his route he felt the disturbance in his head, and afterwards his memory was blank until he found himself at the terminus during the usual rest period. His wife also described another type of attack in which he behaved in an extraordinary manner, banging doors and rushing up and down stairs.

While he was in hospital several attacks were observed in which he jumped about, flung out his arms and legs periodically, and rolled on the floor. He did not lose consciousness, but was not quite clear when seen immediately afterwards. His attention was difficult to arouse; questions had to be asked repeatedly, and he fiddled with objects in an absent-minded way.

The following patient was admitted because of a hysterical outburst. Several attacks of undoubted hysterical character were observed in hospital. They could be induced by talking of the onset of her illness, or by over-breathing, or by any minor examination or test. In these attacks she shook all over and screamed. She was not unconscious, did not bite her tongue nor wet her clothing.

Miss A. F—, aged 42. Nothing noteworthy in the family history. No early neurotic traits. Standard VI in school. In domestic service since 15 years of age. She had been suffering from occasional diffuse headaches ever since she was 16. During the last three years she had had attacks of " bilious " bilateral headaches, which were strong enough to wake her up during the night.

She was described as a reserved, rather self-conscious, obstinate, cheerful, active and entirely undemonstrative personality.

She had the first attack when she was at work in the kitchen ; she suddenly felt a stabbing pain on the top of her head. She lost consciousness, and was found lying on the floor. She was not paralysed, but very weak all over.

Physically : Her blood-pressure was 230/155 mm. Hg., and rose during the attacks by 10–30 mm.

The attacks were observed to come on while the patient was asleep.

Again, it seems hardly conceivable that these particular attacks can have been of purely psychogenic origin, if one considers the previous history, the absence of emotional causes, the occurrence during sleep and the character

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of the first attack. One will have to assume that the primary cause is an impairment of the brain function due to a circulatory disturbance leading to alteration of consciousness, which provides the background for a primitive hysteriform reaction.

The main elements in these attacks in both cases were probably, in the first place, the disturbance of consciousness brought about by a fluctuation in the blood-pressure, secondly a motor discharge on an intermediate level consisting of repetitive pseudo-spontaneous movements, and thirdly, possibly an elaboration or continuation of these movements in the stage of transition to normal consciousness.

Mentioning these observations I have trespassed to some extent on Dr. Mayer-Gross's topic, since he is going to deal with hyperpiesia. However, the conception of hypertension, as brought forward by Dr. Krapf, puts it into close relationship to the vasomotor neuroses. It is, so to speak, a vasomotor neurosis on a high blood-pressure level.

It is only a short step now to raise the question whether similar clinical pictures, observed in neurotics, correspond to similar happenings in the vascular system, only on a normal level. There is very little exact knowledge which would enable one to answer this question. We know, however, that there are constitutional relationships between the vaso-labile and the hypertensive patient. Furthermore, there is material to show that vaso-neurotics have a tendency to develop hypertension, and finally, experiences of the R.A.F. in recruiting appear to demonstrate that by excluding vaso-neurotics from the service, neurotics generally can be excluded. There still remains a wide field for further research. Only a certain percentage of neurotics exhibit a high blood-pressure. There is some evidence of correlation between the blood-pressure level and the type of neurosis (S. Barton Hall). Besides, a certain percentage of patients and normal persons show a transient rise of the blood-pressure, generally interpreted as an emotional rise due to the tension during the medical examination or other extraneous stimuli, but it may be asked why all subjects do not show this vasomotor reaction. What is the particular constitution of this selected group? Of what particular kind is the interaction of the vascular system and the mental symptoms in the particular case, considering the various possible ways outlined before?

A special instance of organ-disposition can be found in neurotics with low and labile blood-pressure—a group which may be mentioned here as a contrast and a supplement to that showing vaso-labile hypertension. Very little attention has been paid to this group from the psychological standpoint. As I see it, two types may be distinguished: one consists of asthenic, fatigable, permanently tired and easily exhausted persons, inclined to faint and to break down in neurosis; the other group inclines to create tension artificially, to breed conflicts and intrigues. A well-known type of hysteric seems to belong to this group. Also the inclination for some blood-pressure-raising drugs,

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such as caffeine or benzedrine, may be rooted in this constitutional predisposition, whereas the vasodilator alcohol is the favourite drug of the pyknic who tends to hypertension.

A few words should be said about the psychological symptoms in functional heart disease. The general trend of investigation should be again to study the psychological accompaniments of well-known disturbances of the heart function, in order to learn finally what anomalies of function may be expected to be found as the basis of the neurotic symptoms of which the patients complain. There is little reliable material available on which to base our views. Fahrenkamp has systematically collected interesting material which demonstrates a remarkable discrepancy between patients with organic and functional heart trouble concerning their subjective symptoms and their attitude towards illnesses, or work, or pleasure. Roughly it seems that those patients who complain verbosely about sensations in the heart, about palpitations and anomalies of rhythm, who feel largely interfered with in their capacity for working, and who live more or less the life of an invalid, show no clinical signs of structural change or incompetence of the heart. On the other hand, it is striking how little patients with heart failure and decompensation complain. These patients often only come to see a doctor when urged by their relatives, and it is often hard to make them adapt their work and pleasure to their state. " The psychological problem is not solved," says Fahrenkamp, " as to why the self-confidence of patients with severe heart disease is so little impaired and why these patients have such a non-neurotic attitude."

This problem indicates one line of psychiatric research in *organic* heart conditions, whereas in cardiac neurosis " the choice of organ " is the point of conspicuous interest, in other words, the question, How far is it the symbolic expressive significance of the heart, how far is it some alteration of its function that determines the neurotic to be a *cardiac* neurotic ?

A promising mode of procedure seems to me to find out whether, and if so, which, anomalies of function are met with in these neuroses, how they may be influenced, and how their alteration changes the picture of the neurosis or possibly of the neurotic personality.

The most impressive psychological symptom of a disturbance of heart function is the " vital " anxiety, the fear of death or " impending dissolution " in angina pectoris. Many hypotheses on the physiology of anxiety generally are based upon cardiac anxiety in angina or the so-called pseudo-angina. There is a serious objection, however, to this view, in the fact that *pain*, not anxiety, is regarded as the cardinal symptom of angina by the most experienced writers. Paul D. White, for example, says, " There may or may not be fear of death ; in my experience such fear is uncommon ; certainly it is absent in the majority of cases ". Sir James Mackenzie's view was similar. He stated : " Apart from pain, other symptoms may be present, such as constriction across

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the chest, a sense of great depression." On the other hand, the same author said: "There are a number of patients who have sudden attacks of great oppression across the chest, a sensation of sinking, and of impending dissolution, in whom there is no pain. . . . I am disposed to think that in some the attacks are brought on by the sudden onset of an abnormal rhythm, such as auricular fibrillation or flutter."

Furthermore, theories which take cardiac anxiety as the starting-point for the explanation of anxiety in general have often not taken into account that identical or similar sensations may be elicited from other sources as well, and that even in heart conditions there is often not a localized sensation in the heart, or in the chest, but a general feeling of suffocation. Studies of physiologists (Haldane) and clinicians (Wassermann) have shown that probably the oxygen need of the tissues produces those sensations which are in the centre of the experience "anxiety", and it is interesting to note in this connection that attacks of angina pectoris can be induced in favourable subjects by rebreathing low oxygen mixtures (Carl J. Wiggers).

Dattner has tried to explain neurotic anxiety as due to insufficient oxygen supply to the tissues; but experimental proof of this working hypothesis is still scanty. In patients with a particular neurotic picture, the so-called psychogenic hyperventilation tetany, I have been able to show some relationship between tetanoid hyperexcitability (demonstrated by measuring the galvanic threshold of the peripheral nerves) and the predisposition for anxiety attacks, consecutive overbreathing and fits. Considering the influence of alkalosis on the capacity of hæmoglobin to supply oxygen, one can interpret these findings in the light of Dattner's views. Such analyses of small clinical groups appear to me more promising than premature generalization and theorizing.

Many problems which arise in studies of "anxiety" in its psycho-physiological aspect seem to be due to poor definition of the terms used. For many psychological facts are mixed up and summarized under the clinical term "anxiety". Fear, the recognition of and reaction to danger, has often been contrasted with anxiety as objectless fear. This antinomy, however, does not exhaust the problem in question. The objective incapacity for handling a situation and the reaction to this incapacity—called the "catastrophe reaction"—are, according to Goldstein, the deepest roots of "anxiety". On the other hand, according to Jaspers, asking oneself questions about the fundamental metaphysical problems (such as Why am I? Why is something? Why is not nothing?) will cause an experience of "anxiety". For emotions of this kind one can hardly expect the same physiological substratum as for the sensation which one experiences after running up a staircase too quickly.

Therefore the first demand is for a detailed analysis of the psychopathology of anxiety. Only this will furnish the basis for investigation into the pathology and patho-physiology of the various types.

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