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# The Psychiatric Aspects of Paroxysmal Tachycardia

By FRANK FISH

The nervous origin of paroxysmal tachycardia was stressed by many German-speaking authors (Hering, 1911; Rothberger and Winterberg, 1911a; Rothberger and Winterberg, 1911b; Schlesinger, 1906) at the beginning of the century. Thus Schlesinger (1906) pointed out that many patients who suffered from this disorder had neurasthenic or hysterical personalities. He postulated that there was a mechanism responsible for the attacks of tachycardia which could be set in motion by a stimulus in the nervous system. Somewhat later a French cardiologist (Gallavardin, 1922) described a type of paroxysmal tachycardia which he believed was of nervous origin. In more recent times Stokvis (1947) has given an account of a Jewish patient who was taught to think himself into an attack in order to avoid deportation by the German occupation authorities in Holland. There is some pharmacological support for the central origin of paroxysmal tachycardia. Thus Klein (1940) described a schizophrenic patient who when treated with pentamethylene tetrazol developed supraventricular paroxysmal tachycardia four minutes after the injection of the drug. He suggested that the delay between the injection and the onset of the attack indicated that the attack was due to an effect of the drug on the central nervous system. Several different workers (Evans, 1940; Peters and Penner, 1946) have claimed that intravenous injection of ergotamine tartrate will relieve an attack of supraventricular paroxysmal tachycardia within a few minutes. Rothlin and Cerletti (1949) have shown that the effect of adrenaline on the heart is not altered by ergot alkaloids, so that the effect of ergotamine in paroxysmal tachycardia cannot be due to an unopposed vagal effect and must therefore be due to a direct action on the brain centres which regulate cardiac activity.

It is strange that on the whole English-speaking cardiologists do not pay much attention to the nervous factors in this condition (Bramwell, 1953; Campbell and Elliot, 1939). Thus in one authoritative survey of paroxysmal tachycardia (Campbell and Elliot, 1939) *in the absence of organic heart disease*, it is admitted that anxiety may produce attacks, but the authors go on to write "We are speaking of the effect of anxiety on normal people. There is no special association with pathological anxiety states". However the role of the nervous system in the causation of paroxysmal tachycardia has been discussed more recently by English-speaking authors, Friedman (1947) studied a series of twenty-eight patients with neurocirculatory asthenia and found that they suffered from attacks which were cardiac, respiratory, peripheral neurogenic, or cerebral in type. Sixteen of these patients had attacks of cardiac arrhythmia, of which nine consisted of ventricular extrasystoles, four of paroxysmal auricular tachycardia, two of a wandering auricular pacemaker and one of paroxysmal auricular flutter. The other twelve patients had attacks of regular forceful rapid contractions of the heart. Friedman considered that the cardiac and other attacks in neurocirculatory asthenia were due to hypothalamic overactivity. However, since neurocirculatory asthenia is basically an anxiety *state prolonged by secondary gain*, it is reasonable to consider these attacks as acute exacerbations of anxiety. Thus Friedman has shown that severe anxiety not only causes simple tachycardia, but also may produce other cardiac arrythmias including paroxysmal tachycardia and auricular flutter. Duncan *et al.* (1950) studied a series of fifteen patients with paroxysmal tachycardia, seven of whom also suffered from organic heart disease. They claimed that attacks were often related to environmental stress and that many of these patients had marked obsessional traits. From

the case histories quoted by these authors it would appear that some of their patients suffered from mild agitated depressions.

Subbotnik *et al.* (1955) have pointed out that the concept of the nervous origin of paroxysmal tachycardia is widely held by cardiologists in the Soviet Union. They investigated a series of sixteen patients with this disorder. In thirteen of these patients the EEG showed absence of, or irregularity in shape and distribution of, the alpha rhythm. These patients were given diphenylhydantoin sodium because the EEG record was held to show a decrease in cortical inhibition. It was claimed that this treatment reduced the number of attacks. However in three patients in whom the drug had no effect, the EEG showed an excess of slow waves or slow and fast waves. In these patients it was assumed that there was increased cortical inhibition and they were, therefore, treated with caffeine with good effect. Since these workers treated all their patients with fresh air, exercise, psychotherapy and hypnosis it is difficult to decide what part was played by the drugs in the alleviation of these patients. These authors explained the disorder in Pavlovian terms and claimed that the lowering of the tone of the cerebral cortex leads to a pathological eruptiveness in the region of the "invalid point"* which is at the cortical end of the cardiac analyser.

The psychological aspects of paroxysmal tachycardia were impressed on the present author by the occurrence of this disorder in a severely disturbed patient with an hysterical personality. A pilot study of patients suffering from supraventricular paroxysmal tachycardia in the absence of organic heart disease was carried out in the Cardiac Department of the Newcastle General Hospital by the kind permission of Dr. W. G. A. Swan. This revealed that a substantial number of patients with this disorder suffered from a neurotic anxiety state or a depression. One patient with a Wolff-Parkinson-White syndrome had her first attack of tachycardia after the onset of a depressive illness in middle life. Unfortunately these patients could not be investigated in detail before the author moved to London.

Fortunately, by kind permission of Dr. Evan Jones, the author was able to study a series of out-patients in the Cardiac Department of St. Thomas's Hospital, London. The investigation was originally intended to include all patients suffering from paroxysmal tachycardia, in the absence of organic heart disease, who attended the Cardiac Department between 1 January, 1948 and 1 January, 1953. Thirty-eight patients fell into this group, but for various reasons only seventeen were interviewed. Most of these patients were interviewed for an hour on two occasions and a close relative was also interviewed separately.

A personal and family history was elicited in each case, using the Maudsley Hospital case-taking scheme (Mayer-Gross, Slater and Roth, 1954). A questionnaire consisting mainly of questions concerned with anxiety, depression and obsessional character traits was given to each patient. The mental state was not formally assessed, but was estimated during the interviews. Standard questions were asked about the attack of tachycardia (see Appendix) and an attempt was made to get a clear description of the attack from a relative. The diagnosis of paroxysmal tachycardia was considered to be definite when there was independent evidence of a pulse rate over 140 per minute or electrocardiographic evidence of paroxysmal tachycardia. Twelve cases conformed to these criteria. The diagnosis was considered to be probable when there was a good description of a sudden onset and offset of rapid beating of the heart. This applied to the remaining five cases.

All patients were asked if any event was likely to bring on an attack and were asked to describe the events leading up to the first attack and the most recent attack. Only two patients said that there was no event which was likely to produce an attack. The commonest provoking causes were excitement and emotion in eight cases, exercise in six cases and sleep in a further five cases. The frequency of the provoking causes is shown in Table I. Attacks provoked by sleep occurred before dropping off to sleep, during sleep or on waking. As one patient early on in the investigation complained of well-marked

* This is usually designated as a "sick point" in Pavlovian literature, but the term "morbid focus" is a more satisfactory term.

TABLE I

*Provoking Causes of Paroxysmal Tachycardia in 17 Cases*

| | |
|---|---|
| Excitement and emotion | 8 |
| Exercise | 6 |
| Sleep | 5 |
| Fatigue | 2 |
| Sudden movements | 2 |
| Bending over | 2 |
| No cause known | 2 |

hypnagogic experiences, these phenomena were looked for in the fifteen subsequent cases and were found in seven, although in two of these the hypnagogic experiences had only occurred once. Of the five cases in which attacks had occurred in connection with sleep, four had had hypnagogic experiences, although these had only occurred once in one of these patients.

Moderate or severe obsessional traits were found in all seventeen patients. The significance of this finding is doubtful, because in England many diligent "normal" people have obsessional traits to some degree and the examiner could have influenced the replies to questions about personality traits by his attitude. If in fact obsessional traits are more common in patients with paroxysmal tachycardia attending cardiac clinics, this would suggest that the more anxious patients with this disorder tend to see cardiologists, since it is generally accepted that persons with marked obsessional traits are more prone to anxiety and depression than the general population.

The patient's current psychiatric condition and the state when he first reported to the cardiology department were both estimated. In one case the patient denied any psychiatric symptoms at the onset of paroxysmal tachycardia which led to his first attendance, and at the time of the psychiatric interview he had no obvious psychiatric disorder. However this patient did not allow his wife to attend for interview.

In the other sixteen cases some psychiatric disorder was present at the first attendance at the clinic. Two patients had suffered from acute anxiety states and had developed paroxysmal tachycardia shortly after the beginning of their psychiatric illnesses. One of these was a man with an abnormal personality showing evidence of latent homosexuality. He had been training as a male nurse and developed an anxiety state because of difficulty in working with a ward sister. The other patient had developed an anxiety state as a result of the behaviour of an unpleasant superior at his work. Both these patients had no more attacks after their difficulties had been resolved and the reactive anxiety state had died away.

One patient was a passive inadequate man who had been very dependent on his mother. His attacks of paroxysmal tachycardia became more frequent after his marriage to a widow. This seemed to be related to the development of a mild chronic anxiety state, which had subsided when he was seen during the investigation. Two patients were suffering from chronic anxiety states. One of these had paroxysmal tachycardia proven by electrocardiography. This disorder seemed to have developed *pari passu* with his anxiety state, which was a reaction to an insoluble difficult domestic situation. The other patient had a chronic anxiety state, which originally occurred after an air crash while he was serving in the R.A.F. and was kept going by his resentment about his job as a policeman. In this case the paroxysmal tachycardia developed some months after the onset of the nervous illness.

One patient had had travelling phobias for fourteen years and had had an anxiety state for eight years, which had slowly improved, so that when seen it was very mild. In this case paroxysmal tachycardia and the anxiety state developed at approximately the same time.

One patient was a chronically depressed anxious woman, who had had two clear-cut depressive illnesses. Paroxysmal tachycardia had begun while she was recovering from the second illness.

One patient was suffering from an anxiety state, the duration and causation of which could not be estimated, as she failed to attend for a second interview because of repeated attacks of paroxysmal tachycardia.

Eight patients were considered to have been suffering from agitated depressions when they first attended. Four of these had suffered from definite paroxysmal tachycardia for periods of twelve, twenty, thirty and forty-three years

and had developed depression which had aggravated the tachycardia and brought them to the clinic. The other four patients had developed depression and paroxysmal tachycardia about the same time. Three of these patients had definite and one probable paroxysmal tachycardia.

## Discussion

The provoking causes of paroxysmal supraventricular tachycardia are all states in which lack of co-ordination of different parts of the central nervous system might occur. Thus, sudden movements, excitement and exercise may produce sudden changes in the ergotropic (Hess, 1962) part of the central nervous system. In falling asleep, sleeping and awaking lack of balance between different parts of the nervous system is quite common and may express itself in hypnagogic or hypnopompic experiences. Since anxiety produces overactivity of the ergotropic part of the nervous system, it is likely that anxiety would exacerbate any lack of balance of different functional parts of the nervous system. It is therefore understandable that attacks of paroxysmal tachycardia would be more likely to occur in states of morbid anxiety, so that the high incidence of morbid anxiety in the present series is to be expected. It is interesting that this finding is in direct contradiction to the findings of Campbell and Elliot (1939). However this difference can easily be explained. The present author often spent as much as two hours talking to a patient and gave each patient every opportunity to discuss his problems and difficulties. Apart from this a close relative was seen in most cases. This was because many depressives, particularly if they have obsessional personalities, tend to minimize their symptoms, so that it is only by obtaining an account of the patient's behaviour from a close relative that one can assess the nature and extent of the nervous illness. Obviously it is not easy to carry out such a detailed anamnesis in a busy out-patient clinic, so that Campbell and Elliot could easily have overlooked psychiatrically ill patients.

The relationship between psychiatric symptoms and the onset of paroxysmal tachycardia was very variable and this is clearly shown in Table II. It is often suggested that a psychosomatic illness may become chronic as a result of the anxiety created by the unpleasant symptoms. Since paroxysmal tachycardia is a frightening experience and most lay people associate heart disease with death or severe invalidism, it would be reasonable to expect that the illness might produce an anxiety state which would lead to repeated attacks of tachycardia. However, the two patients who suffered from acute anxiety states made complete recoveries and had no further attacks of tachycardia after their situational difficulties were resolved. Only one patient with a chronic anxiety state admitted that he was worried about his heart, and he had a very difficult and demanding wife so that his "bad heart" was useful in controlling his wife's behaviour. Of the remaining five patients with chronic anxiety states one had had tachycardia long before the onset of his neurosis, one was an active man who was not worried about his health, but very unhappy about his occupation, two had had neurotic symptoms long before the onset of tachycardia and in one patient the relation between the anxiety state and the tachycardia could not be determined. Four of the eight depressives had had tachycardia for many years before the onset of the depression, and not one of these patients had developed an anxiety state after the paroxysmal tachycardia had been diagnosed and the nature of the condition had been explained to them. Taking all these facts into consideration there is no

Table II

*The Relationship Between Psychiatric Symptoms and the Occurrence of Paroxysmal Tachycardia*

| Onset of Psychiatric Symptoms | No. of Patients |
|---|---|
| No psychiatric symptoms .. .. .. | 1 |
| Long after onset of tachycardia .. .. | 5 |
| Shortly before onset of tachycardia .. | 1 |
| Simultaneously with tachycardia .. .. | 7 |
| Long before onset of tachycardia .. .. | 2 |
| Relationship with tachycardia not established .. .. .. .. | 1 |

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evidence to support the suggestion that paroxysmal tachycardia may produce an anxiety state which leads to frequent attacks of tachycardia. It could be argued that the tachycardia had provoked the depressive illness in those four patients in whom the depression and tachycardia developed at approximately the same time. This certainly does not apply to the patient in the pilot series with a Wolff-Parkinson-White syndrome who developed paroxysmal tachycardia for the first time after the onset of a depressive illness. It therefore seems probable that the patients who develop paroxysmal tachycardia and depression simultaneously are predisposed to the heart condition, which is activated by the morbid anxiety resulting from the depression.

The excess of morbid anxiety in the present series does not indicate the immediate cause of paroxysmal tachycardia. Since only five of Friedman's twenty-eight anxious patients (1947) had attacks of paroxysmal auricular tachycardia or flutter, some other factor apart from anxiety is necessary to produce a paroxysmal auricular arrhythmia. This could be some property of the heart itself or some imbalance within the nervous system.

It is interesting that four out of the five patients who had attacks in relation to sleep also had hypnagogic experiences, whereas among the remaining ten patients, who were asked about these experiences, only two had had them. In a group of normal university students McKellar (1957) found that 63·18 per cent. had had hypnagogic experiences. Thus the incidence of hypnagogic experience in those patients with attacks related to sleep was the same as in McKellar's normals, but the incidence in the remaining group of patients was well below that of McKellar's series.

## Summary

A series of seventeen out-patients suffering from paroxysmal supraventricular tachycardia, in the absence of organic heart disease were investigated psychiatrically. The commonest provoking causes of an attack were excitement and emotion, exercise, and sleep. Hypnagogic experiences were more common in patients who had attacks in relation to sleep. All patients showed marked obsessional personality traits, but since these characteristics are frequently found among tense anxious individuals and depressives, the significance of this finding is doubtful. Only one patient was apparently psychiatrically normal. Two had acute anxiety states, six had chronic anxiety states and eight had depressions at the time of reporting to the clinic with the complaint of tachycardia.

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## References

Bramwell, C. (1953). *Brit. Med. J.*, *i*, 500.
Campbell, M., and Elliot, G. A. (1939). *Brit. Heart J.*, **1**, 123.
Duncan, C. H., Stevenson, I. P., and Ripley, H. S. (1950). *Psychosom. Med.*, **12**, 23.
Evans, E. I. (1940). *Mod. Med. (Battle Creek)*, **8**, 40.
Friedman, M. (1947). *Psychosom. Med.*, **9**, 242.
Gallavardin, A. (1922). *Arch. Mal. Coeur.*, **15**, 1.
Hering, H. E. (1911): *Münch. med. Wschr.*, **58**, 1945.
Hess, W. R. (1962). *Psychologie in Biologischer Sicht.* Stuttgart: Thieme.
Klein, F. (1940). *Brit. Heart J.*, **2**, 213.
McKellar, P. (1957). *Imagination and Thinking.* London: Cohen and West.
Mayer-Gross, W., Slater, E., and Roth, M. (1954). *Clinical Psychiatry.* London: Cassell.
Peters, M., and Penner, S. L. (1946). *Amer. Heart J.*, **32**, 615.
Rothberger, C. J., and Winterberg, H. (1911a). *Pflügers Arch. ges. Physiol.*, **41**, 343.
—— —— (1911b). *Ibid.*, **42**, 461.
Rothlin, E., and Cerletti, A. (1949). *Helv. physiol. pharmacol. Acta.*, **7**, 333.
Schlesinger, H. (1906). *Samml. klin. Vortr. (N.F.)*, **15**, 737.
Stokvis, B. (1947). *Arztl. Mh. berufl. Forth.*, **4**, 259.
Subbotnik, S. I., Feinberg, Y. S., and Spielberg, P. I. (1955). *EEG Clin. Neurophysiol.*, **7**, 527.

## Appendix

*Questionnaire on Attack of Paroxysmal Tachycardia*

1. How does the attack begin?
2. What is it like when it is on?
3. How fast does the heart beat?
4. How long does it usually last?

5. How does it finish?
5a. Slowly or suddenly?
6. How do you feel when the attack is over?
7. Do you have any special feeling before the attack begins?
8. How frequently do attacks occur?
9. Is there anything which brings attacks on? (Take down what patient says and then ask following if necessary):
9a. Do they come on after exercise, if so how often?
9b. Do they occur during sleep, if so how often?
9c. Do they come on at rest, if so how often?
9d. Do they come on if you are upset by worries and troubles?
9e. Can you think yourself into an attack?
10. Can you describe the first attack? (If necessary Q.: Where were you, what were you doing and so on?)
11. Can you describe the last attack which you have had?

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