PAROXYSMAL NOCTURNAL HYPERTENSIVE EPISODES

A CASE REPORT

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The concept of paroxysmal hypertension is usually associated with phaeochromocytoma, hypertensive encephalopathy and the paroxysms of hypertension occurring in patients with a labile vasomotor system. Diencephalic hypertensive attacks and bouts of hypertension in patients suffering from lead poisoning, tabes dorsalis and spinal cord injury may present a similar picture.[1]

CASE REPORT

Late on 29 April 1962 I was called to attend to a European male, D.B., aged 49, who since that evening had developed weakness of the right upper and lower limbs. On examination, a right-sided hemiplegia was found. The blood pressure was 200/120 mm.Hg. The patient was subsequently referred to hospital. The diagnosis was confirmed and the hypertension was treated with 'rautrax' tablets.

During the night of 20 July I was asked to see the patient again. He stated that after eating a light supper he retired to bed and fell asleep. That day had been spent quietly and he had not had any emotional stress. At approximately 11 p.m. he awoke, his heart pounding and his abdomen feeling distended. He was anxious and weak but had no headache. His most important symptom was the feeling of abdominal distension and flatulence, unrelieved by flatus. On examination the blood pressure was 260/120 mm.Hg, and the pulse rate was 122 per minute. Phenobarbitone sodium, 5 gr., was administered by intramuscular injection. The next morning the blood pressure had dropped to 200/118 mm.Hg. No antihypertensive drugs had been given to the patient that night.

In the early hours of 19 August I saw the patient again for a similar episode. He woke at 2 a.m. with a feeling of intense abdominal distension and flatulence, palpitations, and the inability to keep his hands from trembling. He felt weak and anxious, but had no headache. His blood pressure was 260/180 mm.Hg, and the pulse rate was 106 per minute. Phenobarbitone sodium, 5 gr., was again administered and the next morning his blood pressure had dropped to 170/98 mm.Hg and he felt his normal self. The pulse rate was 84 per minute.

Daily blood pressure readings were then recorded as follows:

25 August: 10.30 a.m. 152/106 mm.Hg. Pulse: 86 per minute.
27 August: 6.30 p.m. 150/100 mm.Hg. Pulse: 84 per minute.
28 August: 7.40 p.m. 168/110 mm.Hg. Pulse: 87 per minute.
29 August: 7.35 p.m. 164/110 mm.Hg. Pulse: 86 per minute.
30 August: 7.20 p.m. 160/110 mm.Hg. Pulse: 84 per minute.

On 2 September I was called again, and found that after sleeping for several hours, the patient woke up complaining of abdominal distension, palpitations and trembling of his hands. His mouth felt dry and he called for a drink of sugar water. There had been no antecedent emotional upsets. The blood pressure was 230/120 mm.Hg and the pulse rate was 120 per minute. Phenobarbitone sodium, 5 gr., was given and the next morning the blood pressure had dropped to 188/116 mm.Hg. The pulse rate was now 96 per minute.

On closer questioning the patient stated that he had experienced similar attacks about once a year, ever since he had been stung by bees 13 years before. Now, however, the attacks had increased in frequency. He stated that the first sign of an impending attack was often the feeling of abdominal distension. After a period, varying from hours to days, the attack subsided, often without any treatment. The shortest attack had been approximately two hours. These episodes tended to occur almost invariably at night and after he had fallen asleep.

DISCUSSION

In reviewing this case, the following features are of note:

1. The paroxysmal hypertensive episodes invariably occur at night and after the patient has been asleep for several hours. The patient is awakened by the onset of the symptoms.

2. No history of emotional stress immediately before each attack can be obtained.

3. Abdominal and anxiety-state symptoms are prominent, i.e. abdominal distension, palpitations, trembling hands and weakness. No headaches, disorientation, fits or loss of consciousness are associated with these attacks, as is typically found in hypertensive encephalopathy.

4. During the intervals between attacks the blood pressure is of a considerably lower order; also, the patient is asymptomatic.

It is difficult to account for the predilection of these attacks to occur when the patient is asleep. A suggestion is made that the vasomotor centre is subjected to an increased carbon-dioxide concentration, as might well occur during sleep, when the respiratory centre becomes inhibited owing to the elimination of carbon dioxide. A period of apnoea results, allowing the level of carbon dioxide reaching the respiratory centre to rise and thus to stimulate it. It has been found that the intensity of sympathetic activity of the vasomotor centre increases almost directly in proportion to the concentration of carbon dioxide in the extracellular fluids. Therefore, carbon dioxide is one of the most powerful of all stimuli affecting the activity of the vasomotor centre — a very high carbon-dioxide concentration can increase the mean arterial pressure from a normal of 100 mm. Hg up to as high as 200 - 270 mm. Hg.[2] A labile vasomotor system would then be very sensitive to small changes in the carbon-dioxide level of the blood reaching it.

In a patient presenting the symptoms of paroxysmal hypertension, one's thoughts automatically turn to the possibility of a phaeochromocytoma. A vanillyl mandelic acid estimation was done on a 24-hour urine specimen (volume 1,200 ml.). The value obtained was 2·5 mg./24 hr. The normal range for the method used was 1 - 8 mg./24 hr. This test showed that the levels of adrenaline and noradrenaline in the urine were within normal limits. A rogitine blocking test was not done. Harrison stated: 'In spite of such diagnostic technics as perirenal air injection and retroperitoneal insufflations with oxygen, the newer pharmacologic agents for provocative and blocking tests and the assay of urinary amines, the diagnosis remains extremely difficult and may be established only by exploration'.[3]

Thus if the patient refuses an operation a definitive diagnosis may never be made.

The occurrence of the attacks of paroxysmal hypertension in this patient could not be correlated clearly with the actions of the antihypertensive drug he was regularly taking, i.e. rautrax. The latter is a combination of hydroflumethiazide, rauwolfia serpentina whole root, and potassium chloride. The patient was taking this drug morning and evening, and without exception his attacks subsided before he had taken his next usual dose. He often omitted to take this drug for days and he still experienced these nocturnal attacks.

Another suggestion is that, following the bee sting 13 years before, his vasomotor centre became unduly sensitive, operating on a possible histamine-release mechanism.

SUMMARY

1. A case of paroxysmal nocturnal hypertension of unknown aetiology is presented.

2. Suggestions as to the mechanism of the process are made.

3. The prominence of the abdominal and anxiety state symptoms, as opposed to the classic symptomatology of hypertensive encephalopathy, is stressed.

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REFERENCES

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3. Harrison, T. R. (1953): *Op. cit.*[1], p. 1421.