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CLINICAL NOTE

MICTURITION SYNCOPE

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Since little is known of the pathogenesis of syncope in general the study of special types, in which contributing factors can be manipulated, may be of great value for its understanding.

By micturition syncope we mean syncope following nocturnal micturition in the standing position. Probably for this reason the syndrome has been encountered exclusively in men.

Gastaut and Gastaut (1956) were the first to describe cases (40) studied electroencephalographically. They postulated that the syncope is caused by orthostatic hypotension combined with a considerable decrease of the heart rate in constitutionally hypersensitive subjects. The cardiac slowing was thought to be caused by increased nocturnal parasympathetic action enhanced during micturition.

Dermksian and Lamb (1958) found syncopal episodes in 82 pilots, 3 of whom had fainted during micturition. These 3 cases are also included in a series of 23 cases mentioned by Lyle *et al.* in 1961. In both studies the investigators concluded that the aetiology of the syndrome was not yet understood, but that the Valsalva manoeuvre was probably not important.

Proudfit and Forteza (1959) were of the opinion that the influence upon the circulation of carrying out the Valsalva manoeuvre, at a moment when the venous return to the heart and the peripheral resistance were low, might be the cause of the syndrome.

A patient described by Eberhart and Morgan (1960) gave ground for presuming the same cause. He suffered from functional obstruction of the vesical neck, a circumstance forcing him to strain strongly before and during micturition. After surgical removal of this obstruction the syndrome disappeared.

Prozan and Litwin (1961) also emphasized the influence of the Valsalva manoeuvre, but they believed that secondary reactions of the circulation following the test are instrumental in causing the syncope. Therefore they called the syndrome post-micturition syncope.

Lukash *et al.* (1964) succeeded in provoking syncope twice in the same patient by means of the tilt-table. They pointed out that in their patient orthostatic hypotension alone was not sufficient to cause unconsciousness, but that the combination with cardio-inhibitory reflexes from micturition was necessary.

Zivin and Rowley (1964) provoked syncope in a patient with post-micturition psychomotor epilepsy by instilling into the bladder 150 ml isotonic saline and instructing the patient to void. Because of the clinical symptoms accompanying the attack and because of the fact that anticonvulsive therapy (Primidone) relieved the patient of his attacks, it was concluded that their origin was epileptic. The EEG recorded during the attack, however, did not show any abnormalities characteristic of epilepsy.

Conversely, Coggins and Gray (1964) produced, in a patient with akinetic epilepsy whose syncopal attacks did not respond to anticonvulsive therapy, a typical attack by using the tilt-table. From their description it is not clear whether there was any connection between the syncope elicited during the experiment and the akinetic epilepsy.

From this survey of the literature it is clear that so far opinions differ regarding the aetiology of the syndrome. Because patients with micturition syncope are often diagnosed as having epilepsy a differentiation is necessary. One method of distinguishing between the two disorders is recording the EEG during an attack. We have not come across any mention of such a procedure in the literature. The only EEG recordings in patients with micturition attacks were made during those provoked under special circumstances, as described by Gastaut and Gastaut (1956) and by Zivin and Rowley (1964).

For this reason we present here the case of a pilot in whom three spontaneous micturition attacks occurred while the EEG and the electrocardiogram (ECG) were being recorded.

METHOD

The patient was asked to drink his usual quantity of beer (2 l) between 8.00 and 10.00 p.m. Around 10.00 p.m. the EEG electrodes were fixed to the scalp according to the 10–20 system. The ECG was simultaneously recorded from a bipolar precordial derivation. The patient went to bed in a dark and more or less soundproof room from which continuous recording could be performed. Recording started

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immediately after the patient went to bed and was done with a 16-channel electroencephalograph (Elema-Schönander) which was connected with a 14-channel tape recorder (Ampex).

Description of the patient

The patient was a 37-year-old pilot who was examined because he suffered from fainting attacks associated with the voiding of urine at night. He had his first attack in 1961 at the age of 28 and until April 1969 had been free from further attacks. Since then he suffered from fainting upon several occasions, always at night when, awakened by stimuli from his full bladder, he voided in the standing position. This occurred most often after having ingested beer the evening before. He usually developed weakness in his legs and dizziness, followed by a period of unconsciousness, at the end or just after termination of voiding, never at the beginning. He denied having to strain to empty his bladder and had never had syncopal attacks associated with defaecation, heavy lifting or other activities involving the Valsalva manoeuvre. He had never complained of tachycardia, palpitations or sensations of dizziness when getting up at night. After an attack he felt heavy and dull.

There is a past history of lymphocytic meningitis in 1955 and concussion in 1961, which he received in a football match. The patient had never had an affliction of the urogenital tract. The family history was unremarkable. The patient used no medicaments and no tobacco. His alcohol intake was not excessive.

He was admitted to the University Hospital of Utrecht for detailed cardiological and neurological investigations. On physical examination he appeared healthy. His blood pressure was 130/80 mm Hg in the recumbent and 115/75 mm Hg in the standing position. His pulse rate was about 60/min. The general examination was within normal limits. The neurological examination was completely normal. The routine laboratory tests gave normal results. The ECG showed a sinus rhythm with normal PR and QRS intervals and an intermediate electrical axis in the frontal plane. The record was within normal limits. Cystoscopy and the micturition cystogram showed a normal shape of the bladder without obstruction of the neck.

RESULTS

The resting EEG was normal. Neither the Valsalva manoeuvre nor ocular compression produced significant changes in the EEG and the ECG. Massage and compression alternately of the right and left carotid arteries did not produce any EEG or ECG abnormality. There was a regular heart rate of 72/min. After some time the EEG showed typical changes as the patient fell asleep and subsequently slept soundly. No rapid eye movement sleep was recorded. Between 1.00 and 2.00 o'clock in the morning the patient awoke spontaneously and signified that he had to urinate. He arose from bed carefully to avoid artifacts in the EEG and ECG records and started voiding in the standing position into a urinal. The EEG under these circumstances showed no abnormalities. The ECG, however, showed changes in the heart rate, which increased from 72 to 130/min (Fig. 1). The blood pressure was 130/80 mm Hg. Immediately after the

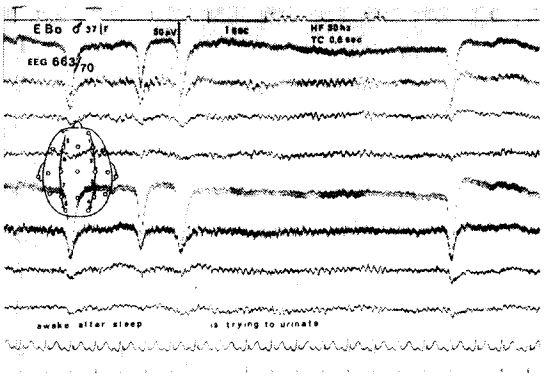

Fig. 1. Normal EEG; the patient is awake after sleep. He stands upright and tries to urinate. The ECG shows an increase of the heart rate to 130/min.

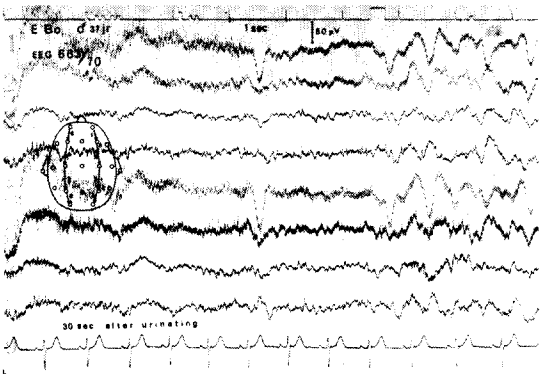

Fig. 2. EEG approximately 30 sec after the patient stopped voiding. Theta activity and later delta activity occur diffusely. The ECG shows a decrease of heart rate to 78/min.

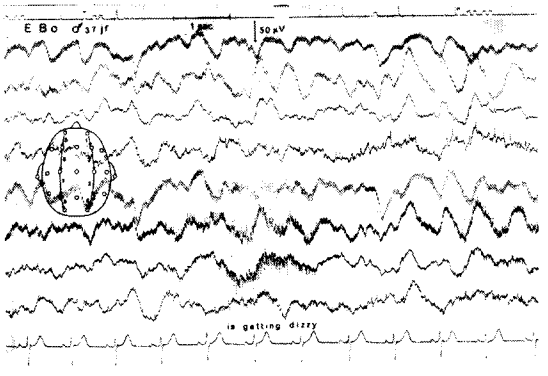

Fig. 3. 10 sec after Fig. 2: there is an increase of delta activity with low frequencies, diffuse in both hemispheres. Heart rate 72/min.

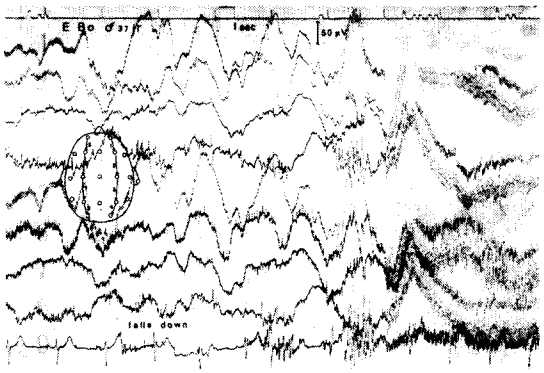

Fig. 4. 10 sec after Fig. 3: very slow delta waves of high voltage occur predominantly in the frontal regions. When the patient falls the record shows artifacts. The ECG shows a decrease of heart rate to 60/min.

beginning of micturition the heart rate quickly decreased to 60/min approximately 60 sec after the patient had stopped voiding. At that moment theta activity, and a few seconds later, delta activity with a frequency of 2–3 c/sec and an amplitude of 100 μV, appeared in the EEG (Fig. 2). The frequency of the delta waves diminished to 1–1.5 c/sec and the amplitude rose to 200 μV. The delta waves became diffuse and bilaterally synchronous and predominated in the frontal regions. The patient now complained of dizziness, staggered and fell unconscious. His blood pressure was 80/60 mm Hg (Fig. 3, 4) During the period of unconsciousness the EEG and ECG showed artifacts (Fig. 4). On one of the three occasions, just before the patient fell, the ECG showed sinus bradycardia followed by a period of 2:1 atrio-ventricular block with a ventricular rate of 28/min. The patient was laid on his bed immediately and regained consciousness within a few minutes. He was pale and was sweating, with a sensation of malaise, but could go back to sleep without difficulty. Some 10 sec after the patient had regained consciousness the delta and theta activities in the EEG disappeared in the reverse order from which they had appeared and the EEG pattern became normal again. We never saw any phenomenon in the EEG that could be interpreted as epileptic. The ECG showed a rather slowly increasing heart rate and the blood pressure also returned to normal slowly. We were never able to provoke attacks in our patient in the daytime. We treated our patient with Insidon, 50 mg, 3 times daily with little effect. Recently Mellander and Nordenfelt (1970) suggested the administration of dihydroergotamine; we have not checked their results in our patient.

DISCUSSION

The fact that it was impossible to provoke an attack during the day suggests that the parasympathetic nervous system, which dominates during the night, played an important role in the origin of the syncope. Our patient had no symptoms of excessive vagal activity. Ocular compression, the Valsalva manoeuvre and carotid sinus massage failed to produce significant slowing of the heart rate and to provoke syncope. The case demonstrates therefore that a constitutional sensitivity of the parasympathetic system is not a prerequisite, as was put forward by Gastaut and Gastaut (1956).

At the moment the patient awoke and assumed the upright position the heart rate increased from 72 to 130/min. This increase may be regarded as one of a series of compensatory mechanisms which normally aid in maintaining a constant arterial blood pressure on assuming the standing position. In our patient, immediately after starting to void at night, this mechanism is obviously disturbed. This may be due to the parasympathetic activity of the micturition and cardio-inhibitory reflexes from the bladder producing slowing of the heart rate (Fig. 2–4) and the increased vagal tone during the early morning hours causing vasodilatation and a low blood pressure. Thus in our patient at night, when the parasympathetic activity is predominant, the compensatory mechanisms fail to produce sufficient vasoconstriction. The combination of insufficient peripheral vasoconstriction and decreased heart rate causes hypotension, resulting in cerebral anoxia and fainting.

From this study it is clear that the micturition syndrome in our patient must be diagnosed as a vaso-vagal (vasodepressor) reaction. As the attacks only occur under the special circumstances described, we believe the syndrome will not incapacitate the pilot from performing his duty.

SUMMARY

The case history is presented of a 37-year-old pilot with micturition syncope. Three spontaneous nocturnal attacks were observed clinically and with EEG and ECG recording. These recordings started at the moment the patient went to bed. In this way the EEG and ECG on awaking during the night, rising from the bed and voiding while in the upright position were studied. Since fainting always occurred some 30 sec after the patient had finished voiding and since no obstruction in the urogenital tract and bladder was found, and the patient denied straining on micturition, a Valsalva effect cannot be regarded as an important aetiological factor. The syndrome is considered to be a vaso-vagal or vasodepressor reaction upon assuming the upright position under special circumstances, in which a low heart rate combined with lowered peripheral vascular resistance is presumably the most important factor. From the EEG and ECG records it is clear that the attacks are not of epileptic origin and that they cannot be explained by slowing of the heart rate alone. EEG recording during an attack is considered to be essential for differentiating between this syndrome and epilepsy because they may resemble each other clinically and because the resting EEG nearly always shows a normal pattern. The syndrome was not considered as a contraindication for licensing the pilot.

RESUME

SYNCOPE A LA MICTION

Les auteurs présentent l'observation d'un pilote âgé de 37 ans présentant des syncopes à la miction. Trois attaques spontanées nocturnes ont été observées cliniquement ainsi qu'à l'enregistrement EEG et ECG. Ces enregistrements ont débuté au moment où le malade est allé se coucher. De cette manière, les ECG et EEG au cours des réveils pendant la nuit,

lorsque le malade s'est levé et a vidé sa vessie en position debout, ont été étudiés. Etant donné que l'évanouissement survient toujours environ 30 sec après que le malade ait terminé de vider sa vessie, qu'aucune obstruction n'a été observée dans le tractus uro-génital ou la vessie, et que le malade nie toute tension au cours de la miction, un effet de Valsalva ne peut pas être considéré comme un facteur étiologique important. Ce syndrome est considéré comme une réaction vaso-vagale ou vaso-dépressive en position debout, ou dans certaines conditions dans lesquelles une vitesse cardiaque basse combinée à une résistance vasculaire périphérique abaissée sont probablement les facteurs les plus importants. Des enregistrements EEG et ECG, il ressort clairement que ces attaques ne sont pas d'origine épileptique et qu'elles ne peuvent pas être expliquées par un ralentissement de la vitesse cardiaque seule. L'enregistrement EEG au cours d'une attaque est considéré comme essentiel pour faire le diagnostic différentiel entre ce syndrome et l'épilepsie du fait qu'ils peuvent se ressembler cliniquement et du fait que l'EEG de repos montre presque toujours un pattern normal. Le syndrome étudié n'a pas été considéré comme une contre-indication à la licence de pilotage.

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