Material and Methods

In two patients with Pickwickian syndromes we have determined both during wakefulness and sleep, P02, PC02, pH, buffer capacity, excess salts, bicarbonate and total CO2 in the arterial and venous blood (internal jugular vein). The urinary metabolites of serotonin (5-HIAA) [6] and of catecholamines [8] were also determined. Polygraphic recordings were made during 24 h. Wakefulness, slow-wave sleep (SWS) and rapid eye movement (REM) sleep were diagnosed for periods of 10 min and on this basis diagrams were made of the time course of these three phases during the entire 24-hour period [4, 5]. Finally, the relationship between the nasal pneumogram and the abdominal pneumogram was established.
Results

The functional-respiratory investigations demonstrated ventilatory dysfunction of an obstructive type. In the first case there also was indication of a 4 diencephalic syndrome. From table I, it is evident that there was a decrease of PO2 in the arterial blood; this was the case particularly during sleep and especially during the apnoic episodes of sleep. PCO2 was increased in the arterial and venous blood during wakefulness and, particularly in the arterial blood, during sleep. The increase was still more pronounced during the episodes of apnea. The amount of total CO2 showed a considerable increase in the arterial blood during sleep. The amounts of 5-HIAA were found to be elevated (10.4 mg%/00, respectively 10.8 mg%/00).

The polygraphic recordings (fig. 1) revealed marked hypersomnia, increased proportion of SWS, and particularly of 'intricated' sleep (IS); there was, however, a notable decrease in the percentages of the REM sleep. The episodes of IS consisted of rapidly changing sequences of SWS, sometimes also of REM, interspersed with short periods of wakefulness (fig. 2a). The long periods of apnea were always accompanied by muscular atony and bursts of 8-activity (fig. 2b). Resumption of respiration was preceded by the reappearance of the muscular tonus for 2-6 sec, by the appearance of a-waves of the awakening for 1-2 sec (fig. 2c). In one of the cases we noted a marked ocular myoclonic activity during a C phase, which started with an episode of apnea and lasted for 17 min.

During waking, a few minutes before dozing off, we noted a decrease in the amplitude of the 'upper' (nasal) respiration, while the amplitude of the 'lower' (abdominal) respiration decreased. Evidently, with intensification of hypnic apnea (recorded through the nasal pneumogram) abdominal respiratory movements increase. At the same time there was a flattening of the EEG recordings, which were interspersed with slow activities; these patterns were accompanied by bradycardia and EKG changes (prolonged QRS interval towards the end of the apnea; reversal of the T wave) (fig. 2d, 2c, 2f, 2g).

Discussion and Conclusions

Our analysis of two patients with Pickwickian syndromes has revealed the marked disorganization of the wakefulness-sleep cycles and the massive

Table I. Study of the venous and arterial gasometry, of the pH value and of the alkaline reserve

Popoviciu Investigations of Wakefulness-Sleep
Fig. 1. Diagrams illustrating the 24-hour cycle of wakefulness-sleep in patients I.I., aged 53, and M.D., aged 40. MS = Mixed (indicated) sleep, REM = rapid eye movement sleep, TS = total sleep.

Hypersomnia with an increase in SWS and in the phases of IS. We also noted numerous phases of REM, which is in agreement with the data of other authors [2, 3]. Of particular interest are the massive respiratory disorders, manifested by the decrease of the amplitude of the nasal respiration and an increase of the amplitude of the abdominal respiration.

Popoviciu Investigations of Wakefulness-Sleep

Fig. 2a. EEG recording of ‘indicated sleep’ (wakefulness, phases B, REM and C). b Apnea in light SWS, with the appearance of bursts of slow waves during an episode of apnea, c The transition from a B phase into the REM phase of sleep, with apnea, d Wakefulness. Both the nasal (Pneumo.) and the abdominal pneumograms (AP) are normal.

In our opinion, the Pickwickian syndrome should be classified among the functional hypersomnias of central origin, and that it is not a type of narcolepsy related to obesity [1, 3]. There is a number of other indications supporting the central nervous origin of this condition, viz. the EEG changes, the bradycardia, the modifications of the EKG, the signs of a ‘diencephalic syndrome’ present in our first case, and the ocular myoclonic episodes.

Evidently we are confronted with a competitive dysfunction of the three systems: wakefulness, SWS and PS as suggested by Passouant et al. [4]. The deficiency of the systems of wakefulness and the exceptionally high reactivity of the systems triggering SWS constitute the main factors that lead to crises of sleep. These systems and, probably, the coming into play of certain descending inhibitory influences of the structures organizing PS lead to marked muscular hypotonia of the facio-bucco-respiratory areas [1, 7] responsible for the appearance of the obstructive apnea. The ensuing hypoxia and hypercapnia activate, then the respiratory centers (with the setting-in-motion of the ‘lower’ diaphragmatic and
thoraco-abdominal respiratory mechanisms) as well as the arousal system thus bring about awakening and the subsequent resumption of respiration.

References


Fig. 2e. The same patient: B phase of sleep. Apnea (nasal pneumogram). One may note a slow EEG activity, bradycardia. / The same patient: C phase of sleep. Apnea on the nasal pneumogram; the abdominal respiratory movements are becoming more and more intense, while the bradycardia is very manifest, g The same patient: REM phase of sleep. Apnea in the nasal pneumogram, good abdominal respiration, muscular atony, EEG with a ‘ saw-tooth ’ aspect. Upon resumption of the nasal respiration: appearance of the muscular tonus and of flat EEG activity of wakefulness.

AP = abdominal pneumogram.

Free Communications: I. Clinical Investigations 550


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Clinical and Polygraphic Study of Cataplectic Attacks

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Materials and Methods

By means of clinical, electroencephalographic, and polygraphic evaluation we have examined 8 narcoleptic patients (7 males and 1 female), who, in addition to the attacks of sleep, also showed cataplectic episodes. We have investigated these crises, their duration and frequency, as well as the conditions of their appearance. EEG recordings were made during spontaneous activity, as well as after hyperpnea and following activation with Ahypon, and Baytinal photocardiazol stimulation. Polygraphic recordings of the pneumogram, of the eye movements (EM), of the EMG and EEG were done around the clock. In three patients, acoustic and photic stimuli were applied during the various phases of sleep.

Results

The frequency of the attacks varied between 1 and 10 per year. In none of the cases did the duration of crises exceed 12-30 sec; none of the patients lost consciousness during these attacks. The crises involving the loss of postural tonus were usually set off by pleasant or unpleasant emotions; occasionally they occurred spontaneously. All our patients have shown total cataplectic episodes.