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ELECTROENCEPHALOGRAPHIC SYNCHRONIZATION INDUCED BY STIMULATION OF SMALL INTESTINE AND SPLANCHNIC NERVE IN CATS

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The visceral receptors, as parts of the homeostatic regulating system, may modify human and animal behavior in several ways (Ádám 1967). One possibility is the contribution of interoception to the control of the physiological sleep—wakefulness cycle. A great number of data indicate that both activating and hypnogenic influences originate in several internal organs. Synchronization and desynchronization of the EEG have been shown to be induced in conjunction with stimulation of visceral surfaces and vegetative nerves in acute preparations (see references in Ádám 1967; Puizillout and Ternaux 1974). After severing the baroceptive (Baust and Heinemann 1967) or gastrointestinal (Rubinstein and Sonnenschein 1971) afferents a marked sleep time reduction was observed. Very few data are available, however, on the receptors and functional states of internal organs responsible for synchronzing and hypnogenic effects.

The present study investigates the possibility of a synchronizing influence originating in the small intestine. It was observed in our previous experiments that cortical activation elicited by intestinal stimuli extinguished rapidly when stimulation was repeated (Ádám et al. 1965; Ádám 1967). The synchronizing and hypnogenic influences of habituated cutaneous stimuli are well known phenomena (Roitback 1960; Pompeiano and Swett 1962). It seemed possible that weak intestinal stimuli, below threshold for inducing cortical desynchronization, can also cause synchronization. Therefore, the electrographic effects of habituated and subliminal stimulation of the small intestine and the splanchnic nerve were studied in all stages of the sleep—wakefulness cycle. The dependence of the splanchnic, vagal and intercostal neurograms on the intensity of intestinal stimulation were analyzed in a series of acute experiments in order to shed some light on the visceral afferents mediating the synchronizing influence. In this paper we present evidence that the central synchronizing mechanism can be brought into action both by intestinal and by splanchnic stimulation.

Materials and method

1. Chronic experiments

These experiments were performed on 8 adult cats. The animals were surgically prepared under barbiturate (Nembutal) anesthesia. In 4 animals, a small intestinal fistula was made by Thiry—Vella's procedure and in the others, bipolar stimulating electrodes were fixed on the abdominal part of the left splanchnic nerve. All cats had stainless-steel screw electrodes implanted over the parieto-occipital cortex and the right orbit in the usual way. Steel wire electrodes were inserted into the cervical musculature.

After recovery the cats were placed in a

sound-proof cage and the ECoG, EMG and EOG were continuously monitored on an 8-channel EEG recorder. Wakefulness (W), drowsiness (D), slow wave sleep (SWS) and paradoxical sleep (PS) were distinguished according to the usual polygraphic and behavioral criteria (Delorme et al. 1964; Sterman et al. 1965). Mechanical or electrical stimulation of the intestine and electrical excitation of the splanchnic nerve were used to modify the EEG. Intestinal tension receptors were rhythmically stimulated by a balloon inserted into the fistula, and inflated (at 1/sec) by manual compression of a rubber ball. A manometer controlled the distension pressure. A plexiglass cylinder with gold-plated caps on its ends (diameter, 0.5 cm; length, 2.5 cm) served as an intestinal electrode. Square wave pulses (0.01—0.5 msec duration, 1—15 c/sec frequency, 1.5—5 V were delivered from a conventional stimulator. Both rhythmic distension and electrical stimuli were applied in 1 min trains. At first, the threshold of cortical activation was determined in D and SWS through increasing the distension pressure or stimulus voltage. Thereafter, stimulation was repeated until extinction of desynchronization, or the stimulus voltage was lowered by 0.1—0.4 V. Both habituated and subliminal stimuli were used to induce synchronization. The synchronization was considered to be a result of stimulation if: (a) it began within 10—15 sec after the onset of stimulation; (b) the length of the synchronized interval was at least 1 min; (c) a quantitative increase in amplitude of the spontaneously synchronized activity could be verified. Therefore the mean values, calculated from the peak-to-peak amplitudes of the 10 largest waves of each control and test record, were compared.

2. Experiments on acute preparations

These experiments were carried out on 14 adult cats under chloralose anesthesia. In the course of surgical preparation the upper part of the small intestine, the left splanchnic, cervical vagal and the 12th intercostal nerves were exposed. The electrode used in chronic experiments was inserted into the lumen of the small intestine. Bipolar recording electrodes were fixed on the exposed nerves. The intestinal mucosa was stimulated with the same stimulus parameters used in the chronic experiments and the corresponding neurograms were recorded and averaged on an NTA-512 B type averager. The averaged neurograms were compared to detect: (i) the splanchnic and vagal afferents activated by different stimulus parameters; (ii) those activated when a response developed in the intercostal neurogram, indicating a viscerosomatic reflex (Downman 1955).

Results

1. Synchronization induced by habituated intestinal stimuli in chronic experiments

In these experiments, repetitive distension and electric stimulation of the small intestine produced extinction of cortical activation. Subsequently, stimulation induced synchronization of the EEG both in D and SWS.

Desynchronization was induced by the initial inflations of the intraluminal balloon at 1 c/sec (Fig. 1, *A*). The threshold of cortical activation varied in different animals and from experiment to experiment, but generally it ranged between 15 and 40 mmHg. As the rhythmic inflations were repeated, the desynchonizing effect showed a gradual extinction (Fig. 1, *B* and *C*). The number of trains of successive distensions necessary for total habituation of the arousal reaction varied in different animals and in successive experiments; an average of 8—25 repetitions was required. After habituation the rhythmic distension induced synchronization in the explored cortical areas. The reversal of electrographic influence was more obvious when stimulation was performed during less synchronized periods of D and SWS (Fig. 1, *D*). In most cases synchronization continued after cessa-

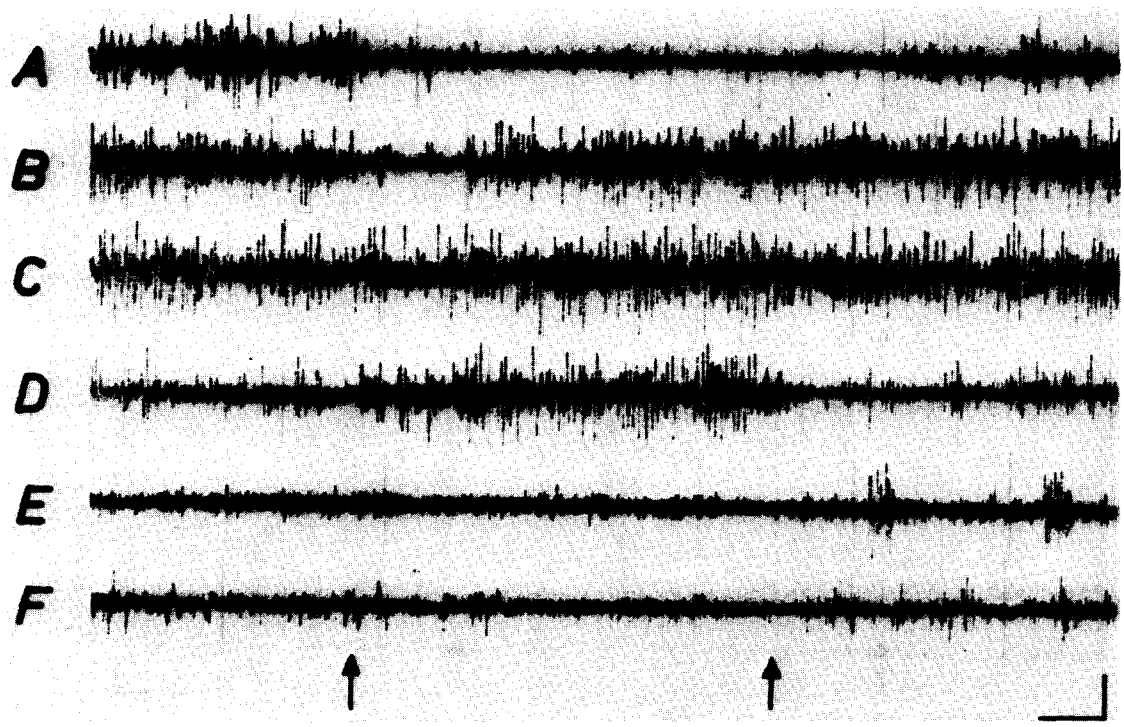

Fig. 1. Synchronization induced by rhythmic distension (30 mmHg, at 1 c/sec, between arrows) of small intestine after habituation of cortical activation. *A*: First stimuli induced cortical activation during drowsiness (threshold intensity). *B*: Reduction of desynchronizing influence after 8 repetitions of stimulus strain. *C*: Total habituation after 15 repetitions. *D*: Habituated stimulation induced synchronization in drowsy state. It had no effect in wakefulness (*E*) or in paradoxical sleep (*F*). Parieto-occipital leads. Calibration: 10 sec, 100 μV.

tion of distension. These types of reaction were observed with 87% of stimuli, whereas spontaneous activities, similar to the induced synchronization, were present in only 21% of the control periods preceding stimulation. When stimulation was performed a spontaneously synchronized background a decrease of frequency and an increase of amplitude were observed. As a result of intestinal distension there was a 16% increase in amplitude. In W or in PS stimulation had no effect (Fig. 1, *E*, *F*) and the intestinal stimuli even showed a tendency to regain their activating influence after every W and PS episode. The habituation procedure had to be restarted in every experimental session. However, considerably fewer repetitions were necessary to re-extinguish cortical activation in the last sessions than in the first ones. One min long steady distension caused desynchronization only at a very high pressure (over 40 mmHg). The desynchronizing effect could not be extinguished by repetition of this strong stimulation.

Similarly, electrical stimulation of the mucosal surface induced synchronization after habituation of the arousal reaction in D and SWS (Fig. 2). The longer the stimulus duration, or the higher the stimulus frequency, the more frequent were the repetitions required to habituate cortical activation and to elicit synchronization (Table I). The extinction of desynchronization and the induction

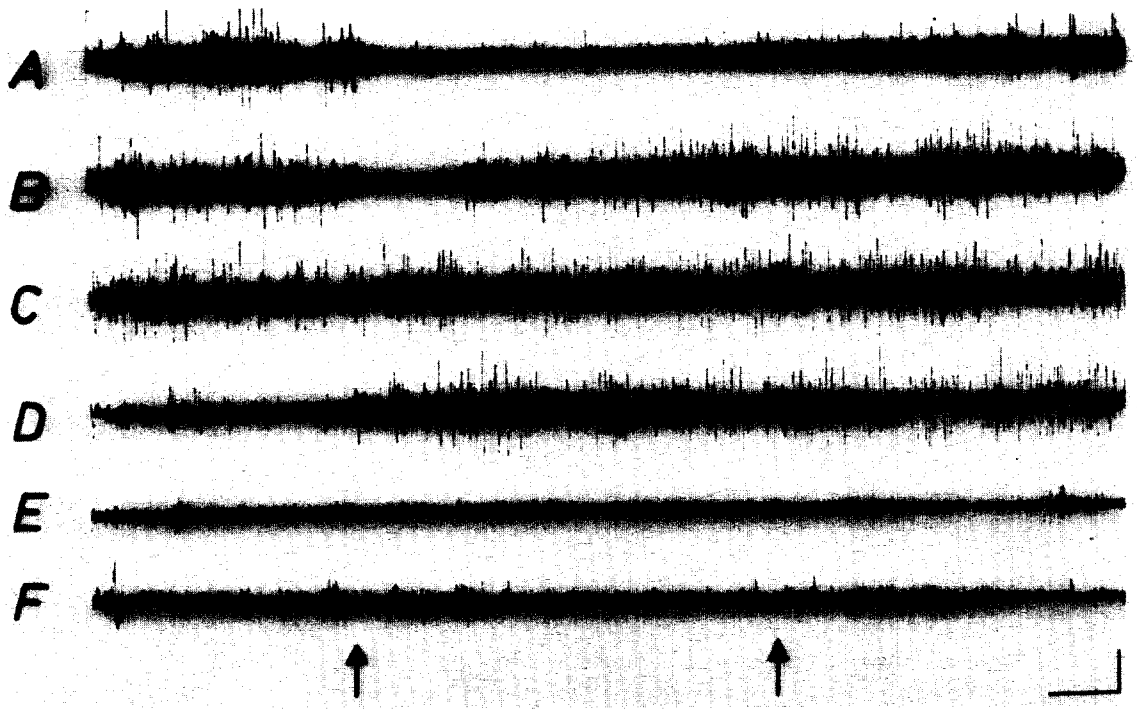

Fig. 2. Synchronization induced by electrical stimulation of small intestine after habituation of cortical activation (shocks of 0.5 msec, 4 c/sec, 3.5 V, between arrows). *A*: First stimulation induced desynchronization in drowsiness. *B*: Partial habituation of activating effect after 11 repetitions of stimulus train. *C*: Total habituation after 16 repetitions. *D*: Habituated stimuli induced synchronization in drowsy state. They had no effect in wakefulness (*E*) or in paradoxical sleep (*F*). Calibration: 10 sec, 100 μV.

TABLE I

Dependence of arousal habituation and of synchronizing effect on frequency and duration of intestinal stimulation in drowsiness. Each value is calculated in % of 200 control or test records.

Stimulus		Mean number of stimuli required for habituation	Occurrence of spontaneous synchronization in controls (%)	Effect of habituated stimuli on	
Frequency (c/sec)	Duration (msec)			Desynchronized background: occurrence of synchronization (%)	Synchronized background: increase of mean amplitude (%)
1.0	0.5	11	23	78	18
3.0		19	21	81	22
6.0		28	25	64	14
8.0		41	19	56	8
6.0	0.1	8	20	75	17
	0.2	13	22	69	19
	0.3	19	18	71	18
	0.5	28	25	64	14

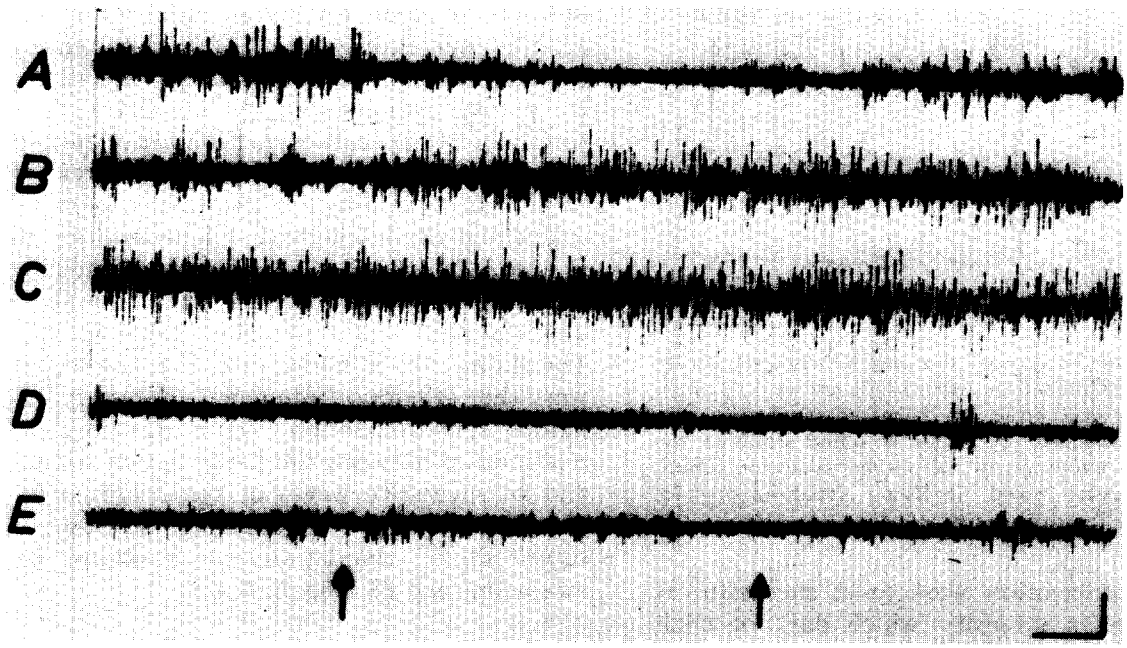

Fig. 3. Synchronization triggered by weak (subliminal) intestinal stimulation. Between arrows: stimulation with shocks of 0.10 msec duration and 5 c/sec at different voltages. *A*: Threshold stimulation for cortical activation during slow wave sleep at 4.8 V. *B*: Induced synchronization at 4.5 V during drowsiness. *C*: Induced amplitude increase at 4.5 V during slow wave sleep. Weak stimulation had no electrographic effect in wakefulness (*D*) or in paradoxical sleep (*E*). Calibration: 10 sec, 100 μV.

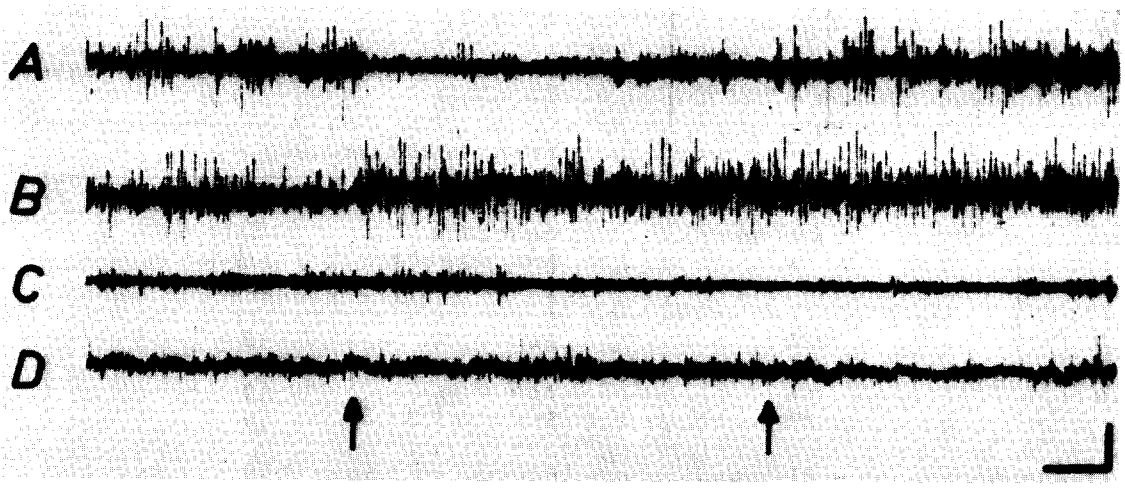

Fig. 4. Synchronization triggered by weak (subliminal) splanchnic stimulation. Between arrows: stimulation with shocks of 0.10 msec duration and 3 c/sec at different voltages. *A*: Cortical desynchronization at 2.6 V in drowsiness. *B*: Triggered synchronization at 2.4 V in drowsy state. Subliminal stimulation had no effect in wakefulness (*C*) or in paradoxical sleep (*D*). Calibration: 10 sec, 100 μV.

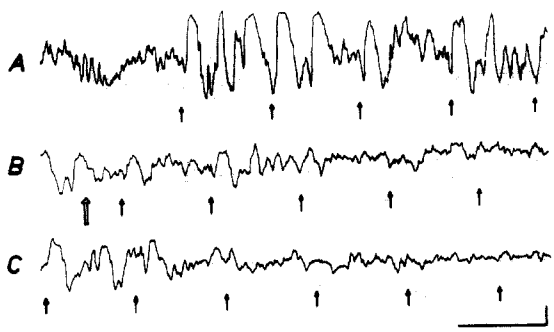

Fig. 5. Effect of arousal and paradoxical sleep on induced synchronization. Arrows indicate weak splanchnic stimuli at 1 c/sec. *A*: Triggered synchronization during drowsiness. *B*: Arousing sound stimulus, delivered at white arrow, abolished synchronization. *C*: Paradoxical sleep abolished synchronizing influence. Calibration: 1 sec, 50 μV.

of synchronization were most successful at the stimulus intensity which did not elicit the viscero-somatic reflex.

2. Synchronization induced by weak (subthreshold) intestinal and splanchnic stimulation in chronic experiments

In this series of chronic experiments a subthreshold stimulus (i.e., below the threshold of cortical desynchronization) directly induced synchronization. After determining the threshold of cortical activation, the stimulus voltage was reduced by 0.1—0.4 V. The threshold of cortical desynchronization ranged between 2 and 5 V. The delivery of a train of subliminal stimuli to the intestinal mucosa or splanchnic nerve triggered synchronization in D and SWS (Fig. 3, 4, 5). The synchronizing influence showed a correlation with the stimulus duration and frequency (Table II, III). The shorter the stimulus duration, the higher was the stimulus frequency necessary to induce synchronization. There was no close relationship between the frequency of stimuli and of successive waves of synchronized activity (Fig. 5, *A*). The synchronizing effect of subthreshold stimulation was abolished by awakening and PS (Fig. 5, *B*, *C*).

3. Neurographic correlates of intestinal stimulation in acute preparations

In acute preparations a close relationship was observed between the stimulus intensity and the neurograms of the 12th intercostal, splanchnic and vagal nerves (Fig. 6). When the intestinal mucosa was stimulated with shocks

TABLE II

Influence of subliminal stimulation of small intestine on the EEG in drowsy state (at different stimulus frequencies and durations). Each value is calculated in % of 200 control or test records.

Stimulus		Occurrence of spontaneous synchronization in controls (%)	Effect of subliminal stimuli on	
Frequency (c/sec)	Duration (msec)		Desynchronized background: occurrence of synchronization (%)	Synchronized background: increases of mean amplitude (%)
1.0	0.1	26	62	12
5.0		31	73	14
10.0		24	75	13
13.0		20	58	9
5.0	0.1	21	62	19
	0.2	32	70	21
	0.3	19	89	27
	0.5	23	73	16
	1.0	28	56	21

TABLE III

Influence of subliminal stimulation of splanchnic nerve on the EEG in drowsy state (at different frequencies and durations). Each value is calculated in % of 200 control or test records.

Stimulus		Occurrence of spontaneous synchronization in controls (%)	Effect of subliminal stimuli on	
Frequency (c/sec)	Duration (msec)		Desynchronized background: occurrence of synchronization (%)	Synchronized background: increase of mean amplitude (%)
1.0	0.1	23	81	23
5.0		26	93	26
10.0		22	58	19
13.0		17	47	13
5.0	0.05	26	93	26
	0.10	21	87	23
	0.20	25	79	24
	0.30	18	63	17
	0.50	22	52	15

of low voltage and short duration (i.e., which proved to be suitable for induction of synchronization in chronic experiments), only the largest splanchnic afferents (Aβ) were excited (Fig. 6, part I, SPL); the intercostal and vagal nerves were practically "silent" (Fig. 6, part I, IC, VAG). They were excited instead at a stimulus intensity reaching threshold for smaller (Aγδ) splanchnic fibers (Fig. 6, part II). Thus, the intercostal neurogram (i.e., the viscero-somatic reflex) showed a correlation with the activity of small (Aγδ, B, C) splanchnic and vagal afferents. It was not present when only the largest splanchnic fibers were excited.

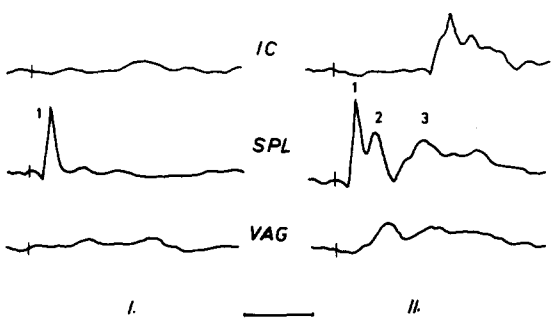

Fig. 6. Averaged neurograms elicited by intestinal stimulation in acute experiment. Recording from 12th intercostal (IC), left splanchnic (SPL) and left vagus (VAG) nerves to weak (I) and strong (II) stimulation of small intestine. Components of splanchnic neurogram: 1: Aβ; 2: Aγδ; 3: B and C. Calibration: 5 msec.

Discussion

It is necessary to consider the receptors and afferent pathways and their central effects which may be responsible for induction of cortical synchronization of intestinal origin.

1. Problem of receptors and afferent pathways

The wall of the small intestine contains mucosal and serosal mechanoreceptors, as well as tension receptors. The majority of mechanoreceptors are highly sensitive and give a rapidly adapting phasic response. The tension receptors are tonically active during intestinal distension and show slow adaptation (see references Iggo 1966; Mei 1970;

Ranieri et al. 1973). It may be supposed that the synchronizing influences belong mostly to the excitation of rapidly adapting intestinal receptors. This possibility is suggested by the following facts: (a) Exclusively non-painful rhythmic intestinal stimuli can induce synchronization. Such stimuli excite mostly the phasic receptors (Iggo 1966). (b) Phasic receptors are innervated by fibers of large diameter running partly in the splanchnic nerve (Gammon and Bronk 1935; Iggo 1966; Ranieri et al. 1973).

Synchronization elicited by splanchnic stimulation may be regarded as a result of selective excitation of the largest (Aβ) afferents since only those intestinal and splanchnic stimuli which do not cause a somatic response can induce synchronization. The largest (Aβ) splanchnic fibers are not included in the viscero-somatic reflex arc (Dowman 1955). In our experiments, when intestinal stimuli did not evoke the viscero-somatic reflex, only the Aβ component was present in the splanchnic nerve action potential.

On the other hand, a phasic excitation of tension receptors may contribute to the synchronizing influence since rhythmic distension can induce synchronization as well. Tension receptors are innervated by thin, non-myelinated fibers forming the major part of the abdominal vagal nerve (Paintal 1963; Iggo 1966; Mei 1970). Chase and Nakamura (1968) obtained exclusively desynchronization through abdominal vagal stimulation. However, extinction of activating effects and induction of synchronization through habituated abdominal vagal stimuli have not been attempted in chronic experiments.

Concerning the afferent pathways, our results suggest that the synchronizing afferent impulses are mostly conducted by the large splanchnic fibers. The synchronizing effect is most obvious when the stimulus intensity just reaches threshold for the Aβ splanchnic afferents. EEG synchronization can also be triggered by selective excitation of baroceptive vagal afferents of large diameter (Dell and Padel 1964; Chase et al. 1967). The synchronizing influence, however, seems to be the result of an adequate discharge pattern of large visceral afferents. This is supported by the following: (a) Induction of synchronization depends on the stimulus parameters. It can be seen only with weak intestinal or splanchnic stimulation. In *encéphale isolé* animals, adequate stimulus parameters are required to trigger synchronization by excitation of vago-aortic afferents (Dell and Padel 1964; Chase et al. 1967). Stimulation with any other parameters has resulted in desynchronization (Zanchetti et al. 1952). (b) In chronic experiments there is a correlation between the vagal neurogram and the EEG effects, the discharge rate of vagal afferents being reduced during cortical synchronization (Varbanova 1967; Leichnetz 1972).

In conclusion, it seems probable that rapidly adapting receptors and their large afferents can play an important role in the induction of synchronization of intestinal origin. However, in freely moving animals, the activating effect of phasic receptors and their large fibers, as well as certain synchronizing influences of slowly adapting receptors and thin afferents, are not excluded.

2. Problem of central influence

Both habituated and weak stimuli induce synchronization when the animals are drowsy. Thus, the afferent impulses of visceral origin trigger synchronization only against a background of reduced vigilance. However, an accumulation of their effect may be possible since the latency of falling asleep has been found to be shortened and the sleep duration to be increased by repetitive stimulation of the small intestine in hungry and satiated cats (Kukorelli et al. 1971). Under natural circumstances such patterns of afferent impulses, synchronizing the EEG, could be generated by the digestive activity of the intestine since the discharge rate of gastrointestinal afferents is increased in hungry, and decreased in satiated, animals (see references in Chase and Nakamura 1968). The afferent impulses

generated by digestive activity may contribute to the induction of drowsiness after eating. Thus, our present and other studies (Kukorelli et al. 1971), together with the experiments pointing to a marked shortening of sleep duration after severance of abdominal vegetative nerves (Rubinstein and Sonnenschein 1971), raise the possibility of the existence of a hypnogenic effect of gastrointestinal origin.

The mechanisms of this central influence of visceral afferent impulses are unknown. The bulbar synchronizing center and sleep-inducing mechanisms can be activated by stimulating the baroceptive vagal afferents, as was demonstrated in studies on "vago-aortic sleep" (see Puizillout and Ternaux 1974). Moreover, stimulation of vago-aortic afferents can trigger "phasic" SWS, PGO activity and PS (Foutz et al. 1974; Puizillout et al. 1974). Consequently, afferent impulses of baroceptive origin can induce or modify each sleep stage in *encéphale isolé* preparations. It seems probable that intestinal afferents can also trigger both SWS and PS. Our quantitative study shows that repetitive intestinal stimulation greatly increases the total sleep time and the number of PS episodes without disturbing the ratio of SWS and PS durations (Kukorelli et al. 1971). Nevertheless, a more detailed exploration of the effects of intestinal afferent volleys on sleep regulating system is required.

Summary

1. In freely moving cats, the cortical desynchronization elicited by painless rhythmic distension, or by low voltage electric stimulation, of the small intestine in drowsiness and slow wave sleep is extinguished following a few repetitions. After extinction of the arousal reaction, similar intestinal stimulation was systematically followed by the appearance of synchronized activity, or an increase of spontaneous synchronization, in the explored cortical areas (parieto-occipital).

2. Intestinal or splanchnic stimulation at an intensity below threshold for cortical desynchronization immediately induced synchronized activity without any need of previous repetitions of stimulation.

3. Stimuli which were followed by synchronization excited only the large (Aβ) splanchnic afferents.

The authors conclude that intestinal receptors may be one of the sources of synchronizing influence which can contribute to the regulation of the sleep—wakefulness cycle and that the large splanchnic afferents may play a role in the induction of synchronization.

Résumé

Synchronisation électroencéphalographique induite par la stimulation intestinale et splanchnique chez le chat

1. Chez le chat implanté libre, la désynchronisation corticale, provoquée par la distension rythmique non douloureuse ou la stimulation électrique à faible voltage de l'intestin grêle pendant l'assoupissement et le sommeil lent, disparaît après quelques répétitions de la stimulation. Des stimulations identiques, consécutives à l'extinction de la réaction d'éveil sont alors régulièrement suivies de l'apparition d'une activité synchronisée, ou bien d'une facilitation de la synchronisation spontanée sur le cortex exploré (pariéto-occipital).

2. La stimulation intestinale ou splanchnique à une intensité inférieure au seuil de la désynchronisation corticale induit directement la synchronisation, sans nécessité de répétitions préalables de la stimulation.

3. Les stimulus, qui induisent une activité synchronisée, n'excitent que les afférents splanchniques de large diamètre (Aβ).

On conclut que les récepteurs intestinaux peuvent être une des sources d'une influence synchronisatrice contribuant à la régulation du cycle de veille—sommeil et que les fibres splanchniques de large diamètre peuvent jouer un certain rôle dans l'induction de la synchronisation.

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