PROGRESS IN BRAIN RESEARCH

VOLUME 20

PHARMACOLOGY AND PHYSIOLOGY OF THE RETICULAR FORMATION

EDITED BY

A. V. VALDMAN

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PREFACE

During the last decade, interest in the study of the mechanism of the action of neurotrophic substances has increased considerably. In several laboratories various new methods have been developed to elucidate the site of action in the central nervous system. In the department of Pharmacology of the 1st Medical University at Leningrad (USSR) a large group of investigators have concentrated their efforts on the analysis of neuropharmacological problems. These investigations were started about 20 years ago by professor V. V. Zakusov, an active member of the Academy of Medical Sciences of the USSR.

The following lines of research have been developed:

(a) The nature of the morphological substrate in subcortical brain structures (reticular formation of the brain stem) as far as the site of action of drugs is concerned.

(b) The degree to which the effect of neurotrophic substances is connected with the mechanism of inhibitory processes in the central nervous system.

(c) The kind of changes which will be evoked in nerve cells upon the action of neurotrophic substances with regard to the various components of synaptic transmission.

It is evident that, for the synthesis and preparation of new psychoactive drugs, a thorough knowledge of known active neurotrophic substances is necessary. Further clarification of the localization and action of neurotrophic substances is therefore a major task of pharmacologists of the nervous system. The results of our laboratory have been summarized in two books: '*New Facts on the Pharmacology of the Reticular Formation and Synaptic Transmission*', Leningrad, 1958, and '*Investigation on the Pharmacology of the Reticular Formation and Synaptic Transmission*', Leningrad, 1961. The present volume is the third book in this series. It deals with the action of neurotrophic substances on visceral functions of the reticular formation *i.e.* the regulation of the cardiovascular system and respiratory mechanisms.

M. G. Bondaryóv and G. V. Kovalyóv have used the method of local stimulation of different structures in reticular formation and have investigated the changes in arterial pressure. In this way, they have studied the influence of various excitatory and inhibitory substances on the morphological structures in the medulla oblongata and the pons, which take part in the regulation of arterial tone. It has been shown that pharmacological substances have different actions on the structures of medial and lateral reticular formation and also on the vestibular nuclei and on the nuclei of the 10th nerve.

The action of a number of neurotrophic substances on the expiratory and inspiratory area of the bulbar respiratory center has been studied by Ma Chuang Gen. His data clarify the inhibitory and excitatory nature of many drugs. In the paper by M. A. Buryak, it is shown that different types of experimental arrhythmia can be evoked by stimulation of the reticular formation in the medulla oblongata.

The regulation of vascular tone, heart activity and respiration is effected by many neuronal structures. However, only a few brain stem structures can be regarded as specific 'centres' for the regulation of these functions. Certain groups of neurons in the reticular formation, which are part of the respiratory centres, have very complicated connections both of facilitatory and inhibitory nature.

A. I. Shapovalov has used the method of intracellular recording of potentials and has shown that different neurotrophic substances are active at the synaptic level. A. I. Shevchenko has also employed the method of intracellular recording and has analysed the functional properties of smooth muscle fibres in order to elucidate the action of pharmacological substances. More detailed schemes of topography of nuclei in reticular formation in the medulla oblongata and the pons of the cat are presented in the paper by A. A. Grantyne. Topographic schemes of nuclei in the hypothalamus and an atlas of microphotographs of serial sections of the hypothalamus are presented in the paper by M. M. Kozlowskaya.

It is hoped that this volume will give sufficient insight into the research programme of our pharmacological laboratory and that the papers will broaden our understanding of the function of the reticular formation.

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Pharmacology of the Brain

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The enormous progress made in the development of neurology, especially in the field of morphology and physiology of the reticular formation and diencephalic systems, created the possibility of a profound investigation of neurotropic drugs acting upon the higher regions of the central nervous system. It became possible to deal in an entirely new fashion with the mechanisms of the action of a series of pharmacological drugs on the function of the brain. A new chapter of pharmacology emerged — psychopharmacology. A detailed study of the influence of neurotropic drugs (newly developed synthetic, as well as already known ones) on the different structures and functional systems of the brain seems to be one of the most urgent tasks of modern pharmacology of the nervous system.

It is a pity that until now such research in the field of pharmacology has not been done in sufficient amount. There are only scattered facts about the action of neurotropic drugs, obtained in a fragmentary manner in physiological experiments, and the drugs considered comprise only a comparatively small number of the pharmacological compounds; moreover many of these were often determined under inadequate experimental conditions (*e.g.* on anesthetized animals). However, the author considers it nevertheless useful to mention essential data from the literature, mainly of the electrophysiological action of neurotropic drugs, and on the action of a series of suppressing and stimulating drugs on specific and diffuse afferent systems, that is the reticular formation and the medial thalamic system, as well as parts of many intracortical processes. Where it became inevitable we have also touched upon some questions from the field of neurochemistry.

NARCOTIC DRUGS

While studying the central action of narcotics we paid special attention to the explanation of their influence on the propagation of excitation along the afferent pathways. The solution of this problem is directly related to the deciphering of the nature of the narcotic effect. The afferent pathways ascending to the brain can be divided morphologically and functionally, and can be separated into two principally different categories: the so-called specific (lateral, 'classical'), and the non-specific (extralemniscal, medial, diffuse) afferent pathways*.

* A circumstantial dissertation of the morphology and physiology of these afferent systems was submitted to us in a well-arranged work entitled '*Structure and Function of the Reticular Formation and Connections with this System*', published in the collective work '*Pharmacological Analysis of the Reticular Formation and the Synaptic Transmission*', Leningrad, 1961, pp. 11–73.

The classical ascending pathways serve various forms of sensibility, being only interrupted by specific nuclei of the optic thalamus. Every point on the surface of the body has its own spatial representation in the specific nuclei and of course, in the somatosensory field of the cortex. The functional significance of this system lies in the conduction of discrete impulses of a particular qualitative characteristic and localization.

All ascending afferent pathways start at the level of the medulla oblongata, and send collaterals (or separate direct fibers) to the reticular formation of the brain stem. This structure, together with the so-called non-specific thalamic complex, is the morphological substrate of the diffuse afferent system. The distribution of the stimulation from the reticular formation to the cortex of the brain is carried out either through extrathalamic connections, especially the hypothalamic field, or through a system of non-specific nuclei of the optic thalamus.

The most essential thing for characterizing the non-specific conducting system is the loss of the qualitative characteristic of the afferent signal. Whereas with a specific conducting system the stimulation is always led from the given receptor itself to the cortical projection area along rigidly defined pathways, with the system involved with the reticular formation numerous interactions of the afferent input take place nullifying specificity. The collaterals of the different afferent systems all converge on one neuron of the reticular formation. As a result, the ascending rostral conduction is no longer specific for *e.g.* tactile, acoustic or visceral impulses, but rather a summated excitation of a series of neurons, brought into an active state by integration of the local potentials of the collaterals of the different afferent pathways.

As an electrographic equivalent, reflecting the incoming of the afferent stimulation along the specific afferent pathways, there is the so-called primary response, which is led off from a sharply delimited projection area of the cortex, *i.e.* the field which corresponds to the recording of the afferent signal. Fig. 1 gives a scheme, made by Roitbak (1956a,b), representing the relation between some neurons of the projection area of the cortex and the ascending afferent systems. At the bottom of the scheme the mechanism of the origin of the different components of the primary response is easily recognized. The afferent impulses go along the specific pathways to the neurons of the 4th, and partly also to those of the 3rd, layer of the cortex, where they form manifold synaptic endings. In these neurons (1, 2, 3, 4, 5) a local stimulation starts, which with a lead-off from the surface of the cortex can be seen as the initial electropositive wave of the primary response. However, with a surface lead-off we can detect the local excitation of only those neurons whose dendrites ascend to the 1st layer, *i.e.* principally of the pyramidal neurons of the 4th layer (1), but the local stimulation of the neurons with short axons (2, 3, 4, 5) is not detected.

When the local stimulation reaches a critical level, the cells of the 4th and 3rd layers give a discharge that, depending on the origin of a local stimulation in the apical dendrites, which can also be seen as the second electronegative component of the primary response, spreads over the ascending axons (neurons 3 and 4) and the returning collaterals (neuron 1) upwards to the surface of the cortex.

As a result of the discharges of the short-axoned neurons, supplementary bio-

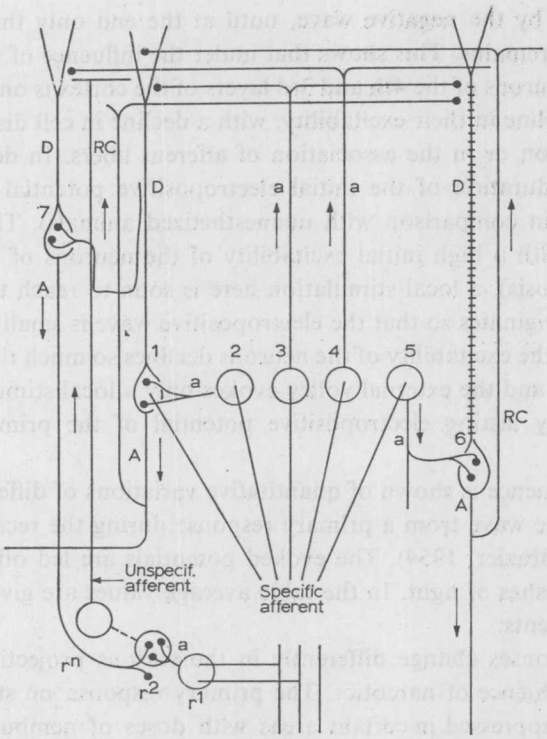

Fig. 1. Scheme of some neurons of the projection area of the cortex and its connections (Roitbak, 1956a,b). 1, pyramidal neuron of the fourth layer. 2–5, cells with short axons of the fourth layer. 6, pyramidal neuron (projectional) of the fifth layer. 7, pyramidal neuron of third layer, participating in the cortical circuit: reticular formation of the thalamus. r^1, r^2 . . . r^n, chain of neurons of the reticular formation. A, axons of pyramidal neurons. RC, reverberating collaterals. D, apical dendrites. a, neuron with short axon.

electric reactions may be seen; these may considerably interfere with the recording of the primary response recorded from the surface of the brain in unanesthetized animals. During the recording of the bioelectric reactions of the cortex in its response to the peripheral stimulations of different quality (namely optic, acoustic, tactile, pain, interoceptive) it was found that a narcosis does not hamper the possibility of the appearance of a primary response. With the depth of the narcosis the recording of the primary response becomes even easier, because owing to this the background noise level of the brain is subdued. The depth of the narcosis, however, is substantially reflected in the appearance of the primary response. Under the influence of narcotic drugs (and more so, with deeper degrees of anesthesia) the latent period of the electropositive component of the primary response increases. This points to an alteration of the functional condition not of the cortical, but of the subcortical neurons of the corresponding specific nuclei of the thalamus (Roitbak, 1955).

The primary responses in unanesthetized animals are complicated and, except for the first two waves (positive and negative), consist of a series of consecutive waves. The response is simplified as the narcosis becomes deeper, the supplementary waves

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disappear, followed by the negative wave, until at the end only the initial electropositive component remains. This shows that under the influence of narcotics a local stimulation in the neurons of the 4th and 3rd layers of the cortex is only accomplished as a result of the decline in their excitability, with a decline in cell discharge in either an ascending direction or in the association of afferent fibers. In deep narcosis the amplitude and the duration of the initial electropositive potential of the primary response increases (in comparison with unanesthetized animals). This is connected with the fact that with a high initial excitability of the neurons of the 4th and 3rd layers (without narcosis), a local stimulation here is soon to reach the critical level, where a discharge originates so that the electropositive wave is small and not lasting. During the narcosis the excitability of the neurons declines so much that the discharge does not take place, and the external volley evokes only a local stimulation. Hence a clear and sufficiently lasting electropositive potential of the primary response is obtained.

In Table I the influence is shown of quantitative variations of different parameters on the electropositive wave from a primary response during the recess of a narcosis (data according to Brazier, 1954). The evoked potentials are led off from the optic area during short flashes of light. In the table average values are given from a series of repeated experiments.

The primary responses change differently in the various projection areas of the cortex under the influence of narcotics. The primary response on stimulation of the vestibular nerve is suppressed in certain areas with doses of nembutal that produce no suppression in other projection areas (Mickle and Ades, 1952).

Optic primary responses are easier to suppress than acoustic ones (Narikashvili, 1954). According to Roitbak (1955), the most stable primary responses are to be found in the projection areas of the sensibility of the skin. This suppression technique makes it possible to list the projection areas of primary responses in the following order of declining sensibility to narcotics: the vestibular area, the optic area, the acoustic area and the area of the sensibility of the skin. The question on which special cortical projection area the different sensibility to narcotics depends — that is, the specific thalamic nuclei of the character of the afferent input — requires special experimental research. The different changes in the primary responses also depend on the kind of narcotic used. Barbiturates create the best conditions for the recording of primary responses (Bremer, 1937; Forbes and Morison, 1939; Arduini and Arduini, 1954; Brazier, 1955). During a sufficiently deep narcosis the essential components of the EEG are suppressed, and the primary response is well-developed, only the latent period being increased. Chloralose in average doses (15–35 mg/kg) even enlarges the response a little in the acoustic projection area (Arduini and Arduini, 1954), but in larger quantities (50–60 mg/kg) decreases the cortical evoked responses. A light ether anesthesia hardly changes the primary responses and alters the latent period less than barbiturates (Forbes and Morison, 1939; Marshall *et al.*, 1941; Arduini and Arduini, 1954); in a sufficiently deep ether and chloroform narcosis the primary responses may be suppressed (Table II). It was shown in a series of tests (Marshall, 1938, 1941; Marshall *et al.*, 1941) that the primary response, arising even from a very

TABLE I

CHANGES OF PHYSIOLOGICAL PARAMETERS CONCERNING PRIMARY AND SECONDARY RESPONSES ELICITED DURING PROGRESSIVE NARCOSIS (NEMBUTAL)

	Primary response					*Secondary response*		
	Latency period of secondary positive deviation (msec)	*Increase (%)*	*Latency period of maximal spike of positive wave (msec)*	*Amplitude of positive wave (mV)*	*Latency period of secondary positive deviation (msec)*	*Increase (%)*	*Latency period of maximal spike of positive wave (msec)*	*Amplitude of positive wave (mV)*
Very slight narcosis	12		22	368	74		100	353
After additional administration of nembutal (30 mg/kg body weight, intraperitoneally)								
First 10 measurements	17	42	27.5	312	110	49	132	710
Second series of 10 measurements	19	58	27.5	256	117	58	136.5	699

TABLE II

RELATIVE INFLUENCE OF DIFFERENT NARCOTICS ON PRIMARY AND SECONDARY RESPONSES

	Primary response	*Secondary response*
	Single afferent pathways	*Complex polyneuronal pathways*
Ether		
Slight narcosis	Facilitation	Deepening
Deep narcosis	Maintained	Reinforced
Barbiturates	Maintained	Reinforced
Chloralose		
Moderate doses	Facilitation	Reinforced
Large doses	Diminished	Diminished

light tactile stimulation (the stir of a hair on a cat's paw) is not changed during a narcosis with ether or barbiturates. There is only a prolongation of the refractory period of the responses, which are led off cranially from the ventrolateral nuclei of the thalamus (the internal capsule and the sensory cortex). With the use of nembutal the absolute refractory period of the responses of the thalamic radiation was increased to 30 msec, and the relative period to 500–700 msec. During an ether narcosis the values were 16 and 72 msec respectively. More caudally from the thalamus (the medial lemniscus, the tegmentum) the narcotics did not arouse a perceptible increase in refractoriness. Consequently, all changes are localized directly in the ventrolateral nuclei of the optic colliculi (*i.e.* in the nuclei of transmission of the specific afferent pathways).

The changes of conduction in the thalamic synapses are more distinct during rhythmic excitation. At the same time the elevation, which is caused by the preceding impulses, is also suppressed. As a result of the increase in the recovery period of the thalamic neurons a transformation of the afferent input takes place, entering along the fibers of the lemniscus. A continuous stream of synchronous volleys going through the thalamic neurons is grouped into short (6–8 msec) volleys of high frequency spikes separated from each other by an interval of refractoriness. A detailed study of the influence of barbiturates on the conduction of an excitation along the afferent pathway was made by King (King, 1956; King *et al.*, 1955; King and Killam, 1957). Fig. 2 gives a scheme explaining the methodology of the tests. Either the peripheral nerves, or the nuclei of the posterior columns, or the medial lemniscus are subject to an excitation. Double stimuli were used at an interval of 10–400 msec, and an alteration in amplitude of the second potential (in % of the amplitude of the first) was established after the introduction of a barbiturate. In various tests a recording was made from the lemniscus, the capsule, the sensory cortex and the reticular formation. Small doses of nembutal (5 mg/kg) did not alter the relative refractory period in the capsule and medial lemniscus, but increased it considerably (two-fold) in the neurons of the reticular formation. The authors associate the small fluctuations, of

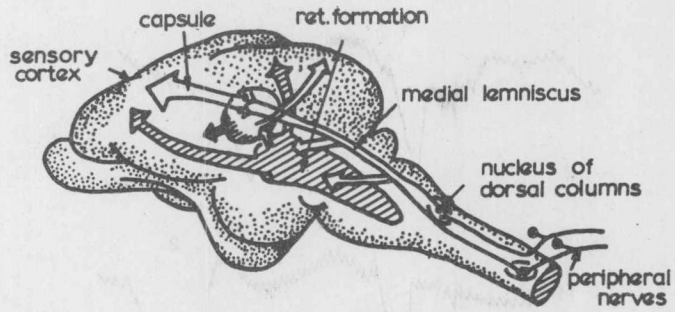

Fig. 2. Schematic plan of specific and sensory pathways not belonging to medial lemniscus.

the conducting period in the specific nuclei of the optic colliculi, with changes in tonus of the reticular formation which influences the conduction in the specific afferent pathways. In larger doses (10 mg/kg) nembutal raises the absolute and negative refractory period of the thalamic neurons about twice (lead-off from the capsule), without a noticeable change in the conduction in the medial lemniscus. Analogous data on the prolongation of the recovery period of the neurons of the specific afferent pathways under the influence of narcotics are given for the acoustic (Tunturi, 1946), optic (Morin *et al.*, 1951) and somatosensory pathways (Jarcho, 1949).

During the stimulation of the different afferent systems in the cortex of the brain the so-called secondary response can also be detected, following the primary response. This secondary response has a rather large latent period (30–80 msec) and shows an electropositive deviation (Forbes and Morison, 1939; Dempsey and Morison, 1943; Forbes *et al.*, 1949). The secondary response has no association with the input, and enters along the classical sensory pathways through the specific nuclei of the optic colliculi, as it does not disappear with the destruction of those structures (Dempsey *et al.*, 1941). This response spreads diffusely over the ipsi- and contralateral hemisphere and disappears with the destruction of the medial regions of the brain (mid-line, subthalamic field). Without going into a detailed review of the physiological mechanisms of the origin of the secondary response, we must emphasize that the origin of this response is through complicated polysynaptic pathways.

The secondary response can also be detected in unanesthetized animals (Brazier, 1955), but is better shown during the recess of a barbitural narcosis (Fig. 3). Here the amplitude of the response increases, but the latent period is also increased (Table I). Ether suppresses the secondary response even during a light narcosis (Bremer, 1937; Forbes and Morison, 1939). Thus, there are important differences between the influences of ether and barbiturates on the generated potentials of the cortex. To a less extent ether alters the conduction in the specific thalamic nuclei, but suppresses the secondary response. Barbiturates intensify the secondary response, but, more important, increase the refractory period of the thalamic neurons. Chloralose in average doses somewhat increases the primary response, but even in a deep narcosis does not suppress the generalized responses that are generated through the associated thalamocortical system (Buser *et al.*, 1949). A light chloralose narcosis shortens the latent period and

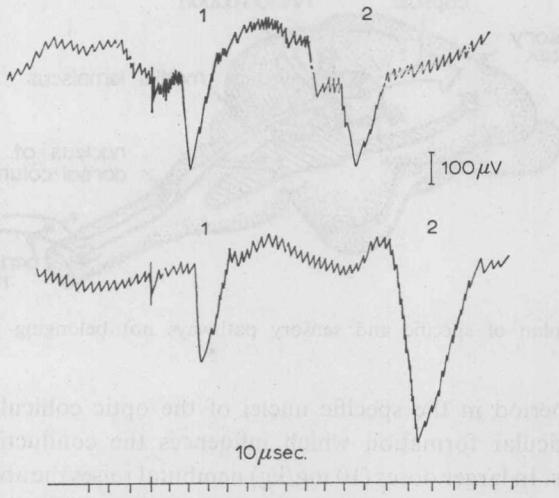

Fig. 3. Changes of primary (1) and secondary (2) cortical responses to single optic stimulation during deepening of narcosis (Brazier, 1955). Upper beam, slight narcosis. Lower beam, deep narcosis with nembutal. The vertical line indicates stimulation artifacts.

lengthens the amplitude of the secondary response of the cortex on stimulation of the visceral nerves. In deep narcosis the latent period increases (Kullanda, 1960).

Consequently during the narcosis there still remains the capacity to handle bioelectrical processes related to the propagation of a stimulation not only along relatively easy pathways (the primary response), but also through complicated multisynaptic systems. In response to an afferent stimulus in the sensory cortex after the primary response, consecutive waves in the modality of 'sensory after-effects' can be recorded. These bioelectrical waves arise as a result of a circular stimulation along the thalamocortical (and evidently intracortical) recurrent cycle. According to the observations of Chang (1958) the repeated waves are more easily demonstrated during a narcosis with barbiturates, though here the number of repeated fluctuations is reduced. According to Bremer (1937), on the other hand, barbiturates very strongly suppress the after-effect, whereas ether has little influence on it. A substantial suppression of the cortical after-effect by barbiturates is also noted by Swank and Watson (1949).

According to observations by Gangloff and Monnier (1957a–c) the threshold of excitability of the cortical after-effect undergoes two-phase changes depending on the dose of the barbiturate. Using phenobarbital for their tests they discovered that in small doses (20–25 mg/kg) this lowered the threshold of the after-effect in the cortex (with a substantial rise in threshold of the thalamic nuclei) and only in large doses (50–100 mg/kg) was there a substantial rise in the threshold and a decline in the duration of the after-effect (Fig. 4).

In this way the bioelectric reactions generated in the borders of the specific afferent system undergo relatively little change due to the action of the narcotics. Therefore it is not possible to understand the nature of the narcotic effect on the basis of the influence of these substances on the input of the cortex from the specific afferent

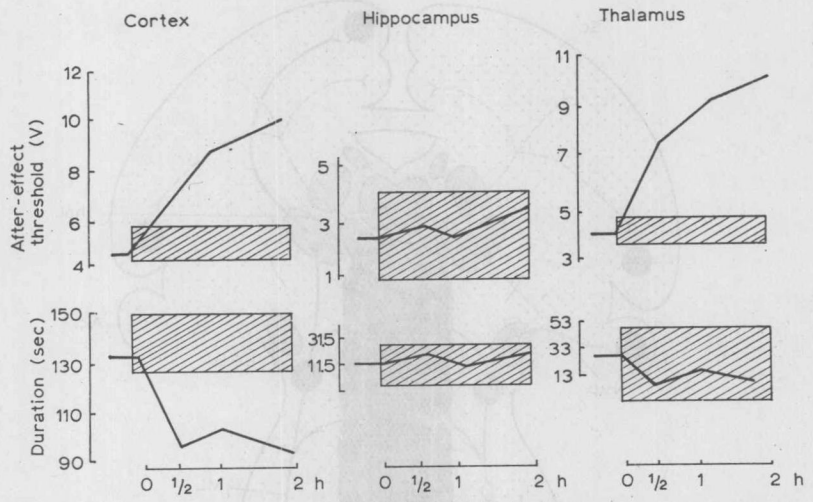

Fig. 4. Influence of phenobarbital (100 mg/kg) on threshold and duration of after-effects in different sections of the cortex (Gangloff and Monnier, 1957a–c).

pathways. The narcotics suppress the non-specific afferent system in a substantially stronger manner. Schematically this system is shown in Fig. 5. The stimulation enters into the reticular formation of the brain stem along the collaterals of the primary afferent pathways. Inside this system the speed of the spread of the stimulation is low, evidently as a result of the existence of many deviations along the conducting pathways.

One of the functions of the reticular formation seems to be the diffuse activation of the cortical neurons. Hence the term 'ascending activating system' has been adopted. A stimulation of the reticular formation manifests itself as a generalized synchronization of the background noise level of the cortex of the brain (a reaction of activation of the EEG, a reaction of awakening), as well as a complex of motor and vegetative reactions. The suppressing influence of narcotics on the reticular system was observed in a direct experiment by French and co-authors (1953). During an ether or nembutal narcosis the response potentials, arising in the lateral afferent pathways, were not changed, but they were strongly influenced in the field of the pons, the mesencephalon and the medial areas of the optic colliculi. There were also changes in the conduction pattern of the intrareticular pathways. However, the same authors remarked that the latent period was not altered under the influence of narcotics as much as the form of the response potentials. This is due to potentials of some (but not all) reticular elements dropping out of the complex bioelectric response.

The degree of suppression of the activating system of the reticular formation is proportional to the dosage of narcotics. Thus, according to data given by Bradley and Key (1958), nembutal in a dose of up to 1.5 mg/kg did not alter the threshold of the reaction of activation of the EEG, arising either through afferent stimuli or an electric stimulation of the reticular formation. In a dose of 3 mg/kg nembutal suppressed the response reaction on an afferent stimulation, but (only insignificantly) raised the threshold through direct stimulation of the reticular formation. In doses of 8–10

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