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RUSSIAN THEORY AND RESEARCH ON SCHIZOPHRENIA

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Most Russian work follows Pavlov's theory that schizophrenia is due to excessive protective inhibition in the cerebral cortex and is devoted to demonstrating in detail the truth of this hypothesis. According to a large number of reports, schizophrenics differ from normals in orientation reactions, sympathetic reactivity, EEG, conditionability, and word association tests. There appear to be 2 groups of schizophrenics: a majority group in whom sympathetic tone and reactivity are low, and a minority group in whom these are high.

In the past few years a number of investigators in the West have advanced theories of schizophrenia in terms of disorders of arousal (e.g., Lynn, 1962; Venables, 1960) or some related concept such as anxiety (Mednick, 1958), sympathetic tone and reactivity (Gellhorn, 1958), or some biochemical disturbance affecting these (e.g., Hoffer & Osmond, 1960). Russian investigators have made a considerable number of studies along somewhat similar lines, although presented in Pavlovian terminology, and the object of the present paper is to review the work of the last decade for Western readers.

Pavlov's Theory of Schizophrenia

Russian research on schizophrenia is still almost entirely dominated by Pavlovian theory and a brief account of this is necessary for an understanding of the rationale of the researches to be described. Pavlov (1941) held that intense or prolonged stimulation of the nerve cells induces a state of protective or transmarginal inhibition, the purpose of which is to protect the cells from further stimulation which would be harmful to them. When nerve cells are in a state of protective inhibition, they do not conduct excitation. It is important in following Pavlovian theory to distinguish between protective inhibition and internal (active) inhibition, which is responsible for the extinction of conditioned reactions with absence of reinforcement and for discrimination ("differentiation"). Protective inhibition has the effect of weakening both the excitatory and (internal) inhibitory processes. Hence, there are certain conditions in which internal inhibition cannot be generated readily because the process has been weakened by protective inhibition.

Pavlov put forward the theory that schizophrenia results from the generation of protective inhibitions in the cerebral cortex. The protective inhibition can be generated as a result of various kinds of shock, drugs, or physical illnesses. There is also an important constitutional factor in liability to schizophrenia, namely, the strength of the nervous system. This is the extent to which the nervous system is sensitive to stimulation, weak nervous systems being those that are most sensitive. A weak nervous system that is sensitive to stimulation is more likely to become overstimulated, generate protective inhibition, and hence, succumb to schizophrenia. The presence of protective inhibition in the cortex accounts for the slowness and poor conditionability of schizophrenics. The other variegated symptoms of schizophrenia are determined by the effect of protective inhibition in the cortex or the other parts of the brain. In cases of catatonic stupor

the protective inhibition spreads down to the subcortex and affects also the sympathetic nervous system. But in those schizophrenic states in which violent outbursts occur, Pavlov suggested that the subcortex is overexcited, due to the removal of cortical control and the operation of the law of positive induction, according to which inhibition in one region of the brain induces excitation in other areas. Pavlov compared this process with the excited outbursts of tired children and intoxicated adults, arguing that in both cases the cerebral cortex is weakened, its control on the subcortex diminished, and hence, the subcortex is freed and activates the emotional outbursts.

Orientation Reactions

A considerable volume of work has been done recently in Russia on the so-called orientation reaction. The orientation reaction covers what is sometimes called in the West the arousal reaction; i.e., the reaction by which the organism pays attention to new stimuli and is mobilized to deal with them. It has three chief components. First, there are changes in skeletal muscles, the animal pricks up its ears, turns its body or head towards the new stimulus, muscle tonus rises, and there is an increase in muscular electrical activity. Secondly, the sympathetic division of the autonomic nervous system is activated: there is an increase in palmar skin conductance and pupil dilation, vasoconstriction in the limbs and vasodilation in the head, and variable changes in heart and respiration rates. Thirdly, the electroencephalogram (EEG) shows an increase in frequency and the alpha rhythm is blocked.

The orientation reaction is distinguished from the defensive reaction which occurs if the stimuli are intense, moderately intense and prolonged, or if the subject is in a state of tension. In the defensive reaction the subject shows signs of being frightened by the stimulus rather than interested in it, and there is vasoconstriction in the head as well as in the limbs.

A number of the components of the orientation reaction in schizophrenics were recorded by Traugott, Balonov, Kauffman, and Luchko (1958), namely, movements of eyes and head, galvanic skin response (GSR), respiration rates, heart rates, and vascular reactions from the shoulder. The stimuli used were auditory (a tone, bell, and whistling sound), visual (lights), and tactile. In chronic deteriorated schizophrenics there were often no orientation reactions of any kind; where reactions were present, however, the autonomic reactions were much weaker than the motor components. In hallucinated-paranoid patients the size of the orientation reaction and its extinction with repeated presentation of the stimulus were very variable, sometimes being stronger and sometimes weaker than in normal subjects. Some patients showed a defensive reaction, while others showed a poor orientation reaction. Reactions to stimulations were also observed during the course of insulin treatment. It was found that in the initial stages of treatment the patients became overactive and gave defensive reactions to the stimuli. Later, with recovery, the stimuli elicited normal orientation reactions.

A similar experiment is reported by Gamburg (1958) on 69 schizophrenics, mainly simple and paranoid. Motor and autonomic components of the orientation reaction were recorded to auditory stimuli and electric shock to the fingers. It was found that very few of the schizophrenics gave normal orientation reactions. Patients diagnosed as simple schizophrenics tended to give no reaction at all, while paranoiacs tended to give defensive reactions. In four out of

five catatonic patients the initial stimulus elicited a defensive reaction, but subsequent stimuli elicited no reaction at all. It was also found that when the schizophrenics did give an autonomic reaction, the autonomic disturbance continued much longer than is usual in normal subjects. In those patients who had not given any orientation reactions to stimulation, caffeine restored the reactivity, but bromine and luminal had no effect. The author interprets this finding as supporting Pavlov's theory that the lack of reactivity is a result of excess inhibition, since it is assumed that caffeine dissipates the inhibition but bromine and luminal further increase it.

Sympathetic Nervous System

A number of Russian investigations indicate that there is a depression of the sympathetic nervous system in schizophrenia, both in its level and its reactivity to stimulation (Ekolova-Bagalei, 1955; Stanishevskaya, 1955; Streltsova, 1955; Vertogradova, 1955). Ekolova-Bagalei (1955) investigated 85 catatonic patients and reported low sympathetic tone as assessed by pulse rate, respiration rates, blood pressure, pupil diameter, sweating and vasometer tone; there was also little reactivity to stimulation. Similar results using the plethysmograph to measure vascular reflexes to hot and cold stimuli were reported by Vertogradova (1955) on 30 early cases of paranoid and simple schizophrenia.

In an investigation by Streltsova (1955) four studies were made of effects of stimulation on the pupil reaction in schizophrenic patients, the first being concerned with determining how far stimulation produces the normal reaction of pupil dilation in schizophrenics. The patients studied were 136 schizophrenics of different kinds (85 men, 50 women, aged 14–55 years, length of illness 2 months to 25 years). The stimuli used to elicit pupil reactions were hot and cold pricks (at 45° and 15°C.), a bell, and an olfactory stimulus. In normal subjects it was found that these stimuli elicit pupil dilation of the order of an increase of one eighth over the initial pupil diameter. Of the schizophrenics, 27.4% reacted normally. The majority of the patients, 65%, showed strikingly subnormal reactions to stimulation; 40% showed no reactions at all and in 25% it was greatly reduced. These patients were long standing hebephrenics and hallucinated paranoiacs, and simple schizophrenias independent of the duration of the illness. The remaining 7.6% of patients showed other abnormal pupil reactions. An excessively large pupil dilation was shown by 3.9% to the extent of an increase of 40%–50% over the original pupil diameter. Streltsova states that increases of this size never occur in normal subjects. These patients were tense and anxious and had confused thought processes. The final group of 3.7% of the patients showed pupil constriction. This reaction is said never to occur in normal subjects except when they are in pain or ill. Streltsova argues that the low reactivity of the majority of her schizophrenics is a result of the high level of inhibition (she does not explain the high reactivity of the small minority of schizophrenics).

In a second experiment, Streltsova (1955) goes on to investigate the extinction of the pupil reaction in schizophrenics using as subjects 50 patients in whom it had been possible to elicit a reaction. In this experiment a bell was used as a stimulus and was presented in two conditions: continuously, and a number of times in short bursts. The results showed that the schizophrenics fell into two groups. Thirty-four out of the 50 patients failed to extinguish the pupil reaction. In normal subjects the

pupil reaction is extinguished after an average of 15 seconds when the stimulus is presented continuously, and after 4–25 presentations when it is presented successively for short intervals, but in the schizophrenic patients the reaction had not extinguished after 3 minutes in the continuous condition or after 50 presentations in the successive condition. A paradoxical result is now reported. After this failure of extinction the intensity of the stimulus was raised considerably. In normal subjects this procedure increases the size of the orientation reaction, but in the schizophrenics the reaction promptly extinguished and remained extinguished for 40 or more minutes.

The second group of 16 patients extinguished the orientation reaction as quickly or nearly as quickly as normal subjects, but these subjects took much longer than normal to recover from the extinction procedure and it was not possible to restore the reaction through the presentation of an intense (disinhibiting) stimulus, as it is in normals. In considering her results Streltsova favors an explanation in terms of the tendency of schizophrenics to generate "protective inhibition." She argues that this accounts for (*a*) the sudden appearance of inhibition, previously absent, when an intense stimulus is presented; and (*b*) the length of time schizophrenics take to recover from the effects of extinction once their orientation reaction has been extinguished.

In a third experiment Streltsova (1955) investigated the effects of caffeine on the orientation reaction in schizophrenics. The rationale of this investigation springs from the hypothesis that the absence of reaction characteristic of most schizophrenics is a result of the strong inhibitory state of the nervous system. It is assumed that caffeine dissipates inhibition and hence, the hypothesis is advanced that if schizophrenics are given caffeine their orientation reactions should be restored. Fifteen patients who had shown the most persistent failure to give pupil reactions were taken as subjects. They were given doses of .1, .3, .8, and 1.3 milliliters of caffeine and were tested before and at intervals of 15, 30, 45, and 60 minutes after injection.

Thirteen of the subjects showed a normal orientation reaction or pupil contraction after .1 and .3 milliliter of caffeine. Streltsova argues that this supports her hypothesis that the schizophrenics were previously in an inhibited state. However, only 3 patients gave orientation reactions after doses of .8 and 1.3 milliliter of caffeine, the other 12 failing to respond. This failure to respond with the higher doses is attributed to protective inhibition.

In her fourth study Streltsova (1955) compared the reactivity of patients early in the morning with that obtained later in the day. She based this investigation on the Pavlovian view that protective inhibition accumulates during the day, as a result of the stimulation received, and is dissipated during sleep. If this is so, an implication of the theory that schizophrenics are characterized by high levels of protective inhibition would seem to be that, if patients were tested immediately on waking in the morning, they would not have time to generate protective inhibition to any appreciable extent and should react more like normals. In this connection Streltsova cites an observation by Naumova to the effect that catatonic symptoms are less marked early in the morning and increase in the course of the day. Streltsova tested this theory using the pupil dilation measure of reactivity. Twenty-two schizophrenics were tested immediately on waking in the morning and again 2–5 hours later. Twenty-one of

the patients responded normally immediately on waking, or occasionally with overreactivity, but later in the day they failed to respond.

An investigation of the characteristics of the vascular system in schizophrenics has been reported by Stanishevskaya (1961). Young simple schizophrenics and anxious hallucinated paranoiacs gave an unusually great number of spontaneous reactions, which is interpreted as indicating a high level of excitation. This in turn is due to the weakening of the cortical inhibitory control over the subcortical areas. In catatonic schizophrenics and in hallucinated paranoiacs who were not anxious, there was very little or no spontaneous activity. When the patients were stimulated, catatonics gave no reactions, hallucinated paranoiacs gave normal reactions, and simple schizophrenics gave generalized vascular reactions but not the local vascular pressor reaction which in normal subjects succeeds the generalized reaction when stimuli are presented a number of times.

Several investigators have reported that sympathetic tone and reactivity are improved in schizophrenics by stimulants including caffeine (Gamburg, 1958; Trekina, 1955), cocaine (Ekolova-Bagalei, 1955), and atropin (Taranskaya, 1955).

EEG

The EEG activity characteristic of schizophrenia reported by a number of Russian workers includes low frequency or absent alpha rhythm, a reduction or absence of blocking of the alpha rhythm to light and other stimuli, large latencies in alpha blocking when it can be obtained, and the presence of "constellations" and "overflows" (Belenkaya, 1960, 1961; Frenkel, 1958; Gavrilova, 1960; Segal, 1955; Trekina, 1955). In Gavrilova's experiment 10 normal subjects were compared with 14 schizophrenics (5 cases of simple schizophrenia, 2 catatonic, and 7 paranoids; duration of the schizophrenia was 8–12 years). The EEGs were recorded when the patients were relaxed and after the presentation of visual and auditory stimuli. When the patients were relaxed, a low frequency alpha rhythm was present in the paranoiacs; but in the simple and catatonics the frequency was below that of the alpha rhythm. No reaction to auditory and visual stimuli was obtained in the simple and catatonic patients, but some reaction occurred in the paranoiacs. With auditory stimuli, a low intensity stimulus produced increased EEG frequency; but when the intensity of the stimulus was increased the reaction disappeared. The results are interpreted as indicating the inhibited state of the cortex in schizophrenics, especially in the simple and catatonic forms and to a lesser extent in paranoia. In paranoid patients weak stimuli evoke some EEG reaction, but strong stimuli increase the inhibition and hence no reaction is obtained.

Gavrilova (1960) also notes the presence in her paranoiacs (but not in the simple or catatonic patients) of frequent constellations, i.e., apparently causeless bursts of high amplitude potentials lasting .5–2 seconds from one cortical area accompanied by low amplitude potentials from another.

These constellations have also been reported in acute, tense, and delirious schizophrenics by Belenkaya (1960). The suggested explanation of these constellations is as follows. The development of protective inhibition in schizophrenia attacks both excitatory and inhibiting processes. It first weakens the process of internal inhibition in the cortex, thereby upsetting the balance between excitatory and inhibitory processes and increasing the strength of

excitation. Strong excitatory stimuli increase the protective inhibition and prevent any reaction, but weak stimuli can still "get through" in the less advanced (paranoid) forms of schizophrenia. When these weak stimuli do get through they produce a violent effect. The reason for this is that the cortical process of internal inhibition is normally concerned with damping down incoming stimuli, and since this process has been weakened, the incoming stimuli can no longer be contained.

In paranoid forms of schizophrenia the presence of overflows has been reported; i.e., a burst of activity in one cortical area appears to spread and is followed by bursts of activity in other cortical areas. These overflows rarely occur in normal subjects when they are awake; but are present in falling asleep and during sleep, and also occur in epileptic cases and in patients with subcortical tumors. Gavrilova observed that external stimuli can induce overflows in paranoiacs, and argues that overflows are caused by subcortical stimuli acting on the cortex and inducing excitation which spreads to other areas. She argues from the absence of overflows in her groups of schizophrenics that subcortical-cortical relations are impaired. But Belenkaya (1960) reported overflows in all stages of paranoia, from the initial acute delerium to the final "secondary catatonic" stage.

A number of Russian investigators have followed the course of EEG changes during the administration of drugs to schizophrenics. Trekina (1955), working with 35 chronic deteriorated schizophrenics, reports absence of alpha rhythm, lack of any reaction to light stimuli, and unsynchronized random oscillations which are interpreted as indicating excitation in the reticular formation. Moderate doses of caffeine brought general improvement and restored the alpha rhythm and the reaction to light stimuli, and abolished or decreased the pathological activity from the subcortex.

A similar experiment by Ekolova-Bagalei (1955) reports the EEG activity of 85 catatonic patients (aged 17–45, duration of illness from a few days to several years) before treatment and after administration of cocaine. The cocaine improved the patients' behavior, so that in the majority of cases they began to move to instructions, speak, and negativism and waxy flexibility disappeared. (In eight cases of very long standing schizophrenia, no improvement was obtained even with increased and extended dosage.) At the same time the cocaine had the effect of increasing the alpha frequency. In a small number of cases, however, large doses of cocaine made the patients worse than before. The explanation of the findings is that small doses of cocaine reduce the amount of cortical inhibition, but larger doses induce protective inhibition, which further intensifies the inhibited state of the cortex. It was noted that cocaine acted first by increasing alpha frequency, then by increasing sympathetic tone, and finally by bettering the patient's voluntary behavior. It is argued that this indicates that cocaine acts first on the cortex and that an effective attack on schizophrenia can be made by restoring the cortical excitatory processes.

Two papers of Belenkaya (1960, 1961) report the effects of chlorpromazine and the stimulant meratran on the EEG activity of paranoid schizophrenics. It is argued that there are typically four successive stages in the evolution of paranoid schizophrenia: first, paranoid delerium without hallucinations; secondly, with hallucinations; thirdly, paraphrenic delerium; and fourthly, a state of secondary catatonia with hallucinations. Forty patients were

divided into the four groups, and without drugs the increasingly advanced stages showed decreasing EEG activity and lesser reactivity to light (in these experimental conditions normal subjects gave a reaction to light on all testings compared with 24% of the first group of patients and only 5% of the last group). All the groups showed overflows. Both meratran and chlorpromazine had a beneficial effect on the first group and to some extent on the second, improving their general condition and making their EEG records more normal. With chlorpromazine, however, there was a delayed effect. For 2 or 3 weeks the pathological features of the EEG increased, especially the number of overflows. After this time the EEG became normal. Belenkaya explains these results in the following way. Chlorpromazine depresses the excitation in the reticular formation, and during the 2- or 3-week period this depression of reticular excitation allows the internal inhibitory processes to be restored. The increased presence of overflows is a sign of the increasing restoration of the inhibitory processes (assuming the equivalence of inhibition, sleep, and the occurrence of overflows). In the next stage, the reticular formation recovers from the effects of chlorpromazine and exerts a normal excitatory effect on the cortex, controlled by the restored inhibitory processes. Patients with the severer forms of paranoia did not benefit from either drug and showed paradoxical reactions to them, viz., after meratran EEG activity decreased and after chlorpromazine it increased.

Fedorovsky (1955) compared the EEG activity of normal subjects and 36 schizophrenics (hallucinated paranoiacs and simple) during sleep therapy. He found that the schizophrenic records tended to show slow alpha rhythms when they were awake, but that during sleep they showed lower amplitude slow waves than normals. It is suggested that this indicates that schizophrenics sleep less deeply than normal people. A similar result is reported with catatonic patients by Popov (1955).

Conditioning

Both autonomic and motor conditioning techniques have been used on schizophrenics by Russian investigators (Dobrzhanskaya, 1955; Kostandov, 1955; Saarma, 1955; Sinkevitch, 1955; Vertogradova, 1955).

All investigators have found that conditioning is poor or cannot be obtained in schizophrenics. An attempt to condition vascular reactions has been reported by Vertogradova (1955), working with 30 schizophrenics (mainly paranoid and simple, duration of illness 1 month to 3 years). Unconditioned vascular reactions to heat and cold were less than normal. A light was used as a conditioned stimulus and conditioned vascular reactions were typically acquired after 2–18 pairings of the light with the unconditioned stimuli. However, the conditioning was not stable; i.e., frequently the presentation of the conditioned stimulus did not elicit a response, and generally the conditioned response had disappeared on subsequent days. Firm conditioned responses could not be acquired with up to 100 pairings. It was also observed that in a number of cases the presentation of the light during the conditioning procedure inhibited the vascular reaction, so that it was either reduced or disappeared entirely. Thirteen patients were retested after a course of insulin, and in the 10 of these who improved, the vascular reactions became stronger and the inhibiting effect of the light during conditioning disappeared. In the other three patients the lack of behavioral improvement was accompanied by a correspond-

ing absence of increase in the vascular reactions.

A somewhat similar experiment has been reported by Trekina (1955) on 35 chronic deteriorated schizophrenics subject to excited outbursts. Plethysmograph recordings were made of the reactions to the unconditioned stimuli of cold, heat, pain, light, and touch. Some vascular reactivity was present, and it is argued that this indicates that excitatory processes are present in the subcortex. It was found that on the second or third day of the investigation the unconditioned vascular reactions extinguished much more quickly than is normal, i.e., after 4–6 presentations. This is taken as evidence for the strong cortical inhibitory processes in schizophrenics. Attempts were then made to condition the vascular reactions to light and to verbal stimuli, but the conditioning was very slow and the conditioned reactions, once established, were very unsteady and kept disappearing and reappearing. This again is taken as evidence for the strong inhibitory processes in the cortex.

A number of experiments use some variety of motor conditioning in which the subject is instructed to give a response to a certain stimulus (e.g., pressing a buzzer to a light) and is given verbal reinforcements when the response is made correctly. This conditioning procedure is then made more elaborate by investigating the discrimination of the stimulus from similar stimuli, extinction of the response through nonreinforcement, effects of extraneous stimulation, and developments of conditioned disinhibition. The findings most commonly reported using these techniques on schizophrenics are as follows:

1. The speed of conditioning is impaired in all schizophrenics, but more in catatonic patients than in paranoiacs. Several investigators have found it impossible to condition catatonics whereas the conditioning of paranoiacs is possible, but slow. Typically 3–5 trials are required to condition normal subjects and 15–20 trials in schizophrenics.

2. The associations are very unstable, but can be stabilized with a very large number of reinforcements (100 or more).

3. The conditioned reaction is very easily inhibited by extraneous stimuli i.e., changes in laboratory conditions, etc., even though these are quite slight.

4. There is a great variability in response latency.

5. Discriminations are very difficult for schizophrenics to make. Many investigators found only a minority of patients would make correct discriminations.

6. Improvements in behavior following treatment are paralleled by improvements in conditionability.

The impairment of schizophrenics in this type of conditioning is generally interpreted as reflecting the inhibited state of the cerebral cortex. The instability of the conditioned reactions, inhibition by extraneous stimuli and variability in response latencies are regarded as due to the strengthening of cortical inhibition through negative induction from the subcortex. Saarma (1955) reports two further findings consistent with this explanation. When discrimination is attempted the schizophrenic frequently ceases to respond at all. It is inferred from this that inhibition has readily become attached to the stimulus to be discriminated and has spread to the original stimulus. Secondly, when reversal shifts (i.e., the positive stimulus is changed to a negative and the negative stimulus to positive) are attempted on schizophrenics, the positive stimulus can easily be changed to negative, but in many cases it was impossible to change the negative

stimulus to positive. A similar finding is reported by Sinkevich (1955).

A possibly unexpected finding from this point of view is that two groups of paranoid schizophrenics, while showing the typical features of poor conditioning listed above, take a large number of non-reinforced trials before the conditioned response is extinguished. This finding has been reported by Dobrzhanskaya (1955) and Kostandov (1955). The explanation advanced by both authors is that the slow conditioning of schizophrenics is due to the protective inhibition. Extinction and discrimination are brought about by the accumulation of internal inhibition and the process of generating internal inhibition has itself been weakened by the protective inhibition. Hence, the slow extinction and discrimination characteristic of schizophrenia.

The effect of drugs on motor conditioning has been reported by Taranskaya (1955). Moderate doses of the stimulant atropin improved schizophrenics' performance on a motor conditioning task, increasing speed of conditioning, the stability of the response, and reducing the latency. At the same time hallucinations disappeared. Larger doses produced less beneficial effect on conditioning and increased hallucinations. The inhibitory drug phenamin further impaired conditionability and increased hallucinations.

Word Association Tests

Apart from conditioning, word association tests are sometimes used in Russian research on schizophrenia. A typical experiment is that of Dokuchaeva (1955). The method consists in presenting a stimulus word, to which the patient has to give a response. The response is scored both for the time taken to give it and for its adequacy; e.g., repetitions of the word, etc., are scored as inadequate. The general findings are that schizophrenics are slow and give inadequate responses. In Dokuchaeva's experiment 60 schizophrenics were given the word association test before and after varying doses of caffeine. It was found that moderate doses of caffeine increased the speed of the response and improved the quality of the associations. This improvement appeared to depend on the reactivation of the sympathetic system, since in cases where caffeine had no sympathetic effect the associative reactions remained unchanged. With large doses of caffeine the speed of reaction became slower and the adequacy of the responses deteriorated.

Treatment

As a logical implication of his theory of schizophrenia, Pavlov recommended and experimented with prolonged sleep as a therapeutic measure. The rationale of this procedure was that it strengthened the protective inhibition and allowed the cortical cells to recover from their exhausted state. The most common drugs used to induce prolonged sleep in the '30s were Cloetta's mixture and sodium amytal, and many Russian psychiatrists have been impressed with the success of these treatments. On the other hand, stimulants have also been used for therapeutic purposes, some of the most common being insulin, convulsive therapy, and caffeine. Ivanov-Smolenskii (1954) considers that the best results are given by combined sleep and stimulant treatment and gives the explanation that sleep allows the cortex to recover, while stimulants reactivate the sympathetic system. Combined administration of bromide with caffeine has also been used successfully in the treatment of dogs with experimental neurosis (Ivanov-Smolenskii, 1954).

In the past few years chlorpromazine

has been widely used in the treatment of schizophrenia and a large number of experiments have been published on its site and mode of action. It is generally agreed that chlorpromazine has an inhibiting effect on both conditioned and unconditioned reflexes; according to Savchuk (1960) dogs and rabbits given 1 milligram per kilogram chlorpromazine give weaker salivatory reactions, but 4 to 5 days after administration these increase. There is no agreement among Russian investigators about whether chlorpromazine acts first on the cerebral cortex or on the reticular formation, but it is generally held that both are depressed by large doses.

A theory to account for the therapeutic effects of chlorpromazine on catatonic schizophrenia has been put forward by Zurabashvili (1960). He assumes that this variety of schizophrenia is initially induced by some toxic substance in the blood, citing as evidence in favor of this view experiments showing that the injection of catatonics' blood into dogs has an impairing effect. In these experiments, dogs were trained on a discrimination task; after injection with catatonics' blood, the learned discrimination broke down and the dogs reacted to the negative as well as to the positive stimulus (this breakdown was not obtained after injection with the blood of normal adults). However, if before injection with catatonics' blood the dogs were injected with chlorpromazine, the impairing effect of the catatonics' blood was counteracted. Since it is known that chlorpromazine has a depressant effect on the reticular formation, he infers from this that the toxic substance in schizophrenia has an excitatory effect on the reticular formation. During the development of schizophrenia the toxic agent raises the level of reticular excitation, which in turn stimulates the thalamic areas and the cerebral cortex. This stimulation becomes "above strength" and induces protective inhibition in both the cortex and the thalamic areas, and hence, the signs of inhibition in the cortex (low frequency EEG activity and slow conditioning) and the low sympathetic tone and reactivity. The effect of chlorpromazine is to depress the reticular excitation; reticular stimulation of the thalamic region and the cortex is thereby reduced and the protective inhibition dissipates. As a result of this, there is clinical improvement and the sympathetic system becomes more active. In support of the last point, Zurabashvili cites evidence that in catatonics receiving chlorpromazine there is a fall in the number of erythrocytes and increases in hemoglobins, sweating, and pulse rate. This theory is a departure from the commonly held Pavlovian view that protective inhibition in the cortex is the primary cause of breakdown in schizophrenia, and is an indication that Russian workers in this field are not necessarily fettered by strict observance of Pavlov's original theory.

Conclusion

At an empirical level, Russian work on schizophrenia at some points coincides with that in the West; and at others, breaks entirely new ground. From this point of view the principal findings can be summarized as follows:

1. The Russian evidence as a whole indicates that there are two types of schizophrenics: a majority group characterized by low sympathetic tone and reactivity; and a minority group, in whom sympathetic tone and reactivity are unusually high. The majority group would appear to consist mainly of cases of catatonic and simple schizophrenia and the minority group mainly of acute and agitated patients, especially paranoiacs. In this respect, Russian investigators seem to have arrived in-

dependently at similar conclusions to those of several workers in the West who have reported two groups of schizophrenics characterized by high and low sympathetic reactivity (Gellhorn, 1958), high and low arousal (Venables, 1960), and high and low anxiety (Mednick, 1958).

2. The Russian findings on the EEG of schizophrenics indicate patterns characteristic of low arousal and drowsy states. There are a small number of similar Western findings although many investigators have failed to find EEG differences between schizophrenics and normals (Brackbill, 1956). This is possibly because Western investigators have not looked for the overflows and constellations described in the Russian literature.

3. Behaviorally, the Russian work showing slow reactions in conditioning experiments is to some extent paralleled by the findings of Eysenck and his associates (Eysenck, 1952; Payne & Hewlett, 1960) of slowness in schizophrenics over a variety of tasks. In general, however, Russian conditioning techniques have been very little used in Western research on schizophrenia.

4. A large number of Russian experiments show that schizophrenics are unusually sensitive to stimulants, being improved by small quantities but impaired by large. If these results are interpreted as being due to the effects of stimulants on arousal, they accord quite well with the somewhat similar results of Venables and Tizard (1956) and Venables (1960), which showed that schizophrenics are unusually affected by intense stimuli and by the level of background stimulation. In this respect, the Russian findings support the hypothesis advanced by Venables (1960) that schizophrenics can only operate efficiently within a narrower range of arousal than normals.

At a theoretical level, it will be evident that Russian work on schizophrenia is strongly tied to Pavlov's theory and is largely concerned with demonstrating in detail the truth of Pavlov's hypotheses or of making modifications to explain discrepant findings within the Pavlovian framework. It would appear that the recent experimental results can be made to fit reasonably well with Pavlovian theory and the theoretical significance of the Russian work on schizophrenia depends on the acceptability of general Pavlovian theory, especially on the concepts of protective inhibition and induction. A critical appraisal of Pavlovian theory as a whole would demand a long essay and would be out of place in this review. But it is apparent that, at any rate in the eyes of Russian investigators, Pavlovian theory has withstood the attacks of its critics.

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