On Lethargy. By Prof. K. AGADJANIANTZ (Warsaw).

LETHARGIC phenomena have often been studied and described in connection with hysteria and hypnotism. It has been pointed out by hypnotists that a state of lethargy occurs at times which takes its own time to disappear. Lethargic states in hysterical persons, whether under the influence of hypnotism or not, are not rare. According to Charcot's classical descriptions the lethargic state can be produced artificially during hypnosis by closing the eyes in the cataleptic phase, but it can likewise be primarily caused by fixation. During the lethargic state consciousness is obscured, sensitivity to pain is diminished, and the tendon reflexes are increased.

The following analysis concerns two patients who had never before showed any symptoms of neurosis, or any tendency to lethargy. Previous physical health had been good, and in neither case had they been subjected to any form of hypnotism or suggestion. In one case the attack of lethargy was the first symptom of an acute infectious illness. A close observation of some of the disputed symptoms of lethargy, and their analysis, will be found interesting when taken in connection with the origin of the whole phenomenon.

Observation I.

The patient, N—, is a girl, æt. 18, who is completing her studies at a high-school in St. Petersburg. He father died æt. 72, and her mother æt. 59. There are no indications in the family history of mental or nervous disease, of alcoholism, or of tuberculosis. The patient has three brothers and six sisters, all her seniors; nursed by her mother, she passed through a perfectly normal childhood—no convulsions, or other nervous symptoms. The menses commenced at the age of 15, and are regular; her sleep and appetite until the attack were normal.

She was seized with a headache on the morning of January 22nd, 1913, and later in the day, when called to the blackboard during a lesson, fell asleep. Dr. Ordoukhanoff and I saw the patient at about 4 p.m. (four or five hours after the onset of the attack), and after a thorough examination and consideration of the symptoms diagnosed a state of lethargy. The patient remained so for a period of sixteen hours, at the expiration of which she awoke with headache and photophobia, although the latter might be attributed to the inflammation of the conjunctivæ in connection with the onset of measles. In my presence the temperature was 99·2° F.

The patient is of medium height, normally, though somewhat underdeveloped for her age, and is moderately nourished, and of healthy appearance; the lymphatic glands and thyroid show nothing abnormal;

bones and joints are normal; no physical stigmata of degeneracy are present; there are no signs of paresis or paralysis of the face, tongue, or extremities; the pulse 80 to 90, and full; respirations 10–12; skin sensibility is abolished; motor system and stereognostic sense cannot be examined because of the unconscious state of the patient; pressure on the nerve-trunks gives no reaction; there are no cramps or contractions; passive movements of the muscles show slight resistance; the muscular irritability is much increased, also the vasomotor reaction of the skin; there is no catalepsy; the pupils of the eyes are equal and somewhat dilated, and react slightly to light; the eyes do not fix on any object when the closed eyelids are raised; examination with ophthalmoscope shows a slight contraction of the vessels of the *fundus oculi*; the conjunctival and the pharyngeal reflexes are absent; plantar and abdominal reflexes are increased; jaw reflex is normal; scapular reflex and knee-jerks are much increased; a slight false ankle clonus is observed; the tapping along the radial and ulnar nerves excites marked contraction of the corresponding muscular groups; tactile stimulation does not give any contraction of the underlying groups of muscles; the fauces and the conjunctivæ are red; the organs of the chest and the abdomen are normal; the patient is sleeping; her breath is even and deep; she takes no notice of orders; any mention of facts that should interest her does not produce any facial reaction; now and then she turns over in her sleep.

About an hour after my visit, the patient began to react to mechanical irritation of the mucous membrane of the nose; with difficulty she was half aroused to micturate, and drink some liquid. In spite of the slight stimulation of the skin she soon fell asleep again, which lasted on the whole sixteen hours from the beginning of the attack.

January 23rd.—The temp. at 4 p.m. was 100·5° F.; at 7 p.m., 98·6° F. There is photophobia, headache, drowsiness; bowels and urine are normal.

January 24th.—At 11 a.m., temp. 100·7° F.; at 2 p.m., 99·6° F.; at 4 p.m., 100° F.; at 7 p.m., 101·3° F. Headache; the consciousness is becoming clearer; no drowsiness; the bowels and urine normal; the mucous membrane of the fauces is reddened.

January 25th.—At 10 a.m., temp. 101·3° F.; at noon, 100° F.; at 4 p.m., 102° F.; at 7 p.m., 103·2° F.; on that day appears a rash characteristic of measles.

January 26th.—At 11 a.m., temp. 103° F.; at 4 p.m., 99·4° F.; at 7 p.m., 98·2° F. The conjunctivitis is less; the general condition is satisfactory; the bowels and the bladder act regularly.

January 27th.—At 11 a.m., temp. 97·6° F.; at 4 p.m., 98·6° F.; at 7 p.m., 97·8° F.

After January 28th, temp. did not reach 98·6° F. The patient feels well; she is regaining strength. She ultimately recovers completely.

Observation II.

I observed the patient X—, together with Dr. Bitchounsky; she was also examined by Profs. Sirotinine, Oppenheim, and others.

The patient, a native of the Caucasus, usually lives in St. Petersburg and London; æt. about 40; married; one daughter; father died at 55, mother at 50; her father was 45 and her mother 38 when patient was born; no consanguinity, she had four brothers and three sisters, five of them older; no hereditary diseases, general, mental, or nervous, were observed in the family; the patient's brother committed suicide, but it was on account of blindness caused by lues, which drove him to despair. The patient had a regular childhood; teethed at the proper time; her mental and physical development were parallel; had pneumonia years before; she was subject to atony of the intestines, and at times to headache; her vasomotor system was excitable; the menstruation was regular as a rule, only at the beginning of the attacks to be described were there some deviations from normal; her one pregnancy and the delivery passed normally; during the last few years she had passed through periods of emotion and sleepless nights.

The present illness began at the end of the summer, 1912, with headaches and nervous instability; at the same time she suffered from menorrhagia, with subsequent anæmia. The treatment was not successful, and at the end of October a general weakness and dilatation of the right heart, with a systolic murmur at the apex, was noticed, but at the subsequent examination of the patient by Prof. Sirotinine this murmur could not be heard. At times a slight œdema of the legs was observed; hæmoglobin, 62 per cent.; urine normal. She suffered from persistent headache, mental oppression, and sleeplessness until treated by iron, arsenic, bromide, and valerian, and dieting and rest did not improve her general health; menorrhagia began to disappear.

The first attack came on in December, after a passing emotion; she got a severe headache, a pain in the region of the heart, followed by sleep. The pulse was at 68–70, regular, and fairly full; in the beginning the patient answered when spoken to, but did not react to pin-pricks; the temperature of the body was 99·4°–99·6° F. Her first attack lasted several hours; it passed away gradually, while the respiration increased, and slowly became normal. After the attack she had sharp pains in the region of the heart, in the left side of the chest, and in the left arm; morphia did not give her relief. The attack returned in about a fortnight, and was repeated at various intervals, at first in connection with any kind of emotion, and later without any obvious reason, but the character of the attack was always the same; it was preceded by headache, heart-palpitations, and retardation of the respirations.

In the autumn, 1912, I examined the patient for the first time, not during an attack, and found the following:

The patient is of a medium stature, normal development, and satisfactorily nourished; temperature normal; skin somewhat pale; there is slight œdema of the legs; the lymphatic glands cannot be felt, the thyroid gland does not appear to be altered; no physical stigma of degeneracy present; the innervation of the face-muscles and expression are normal; the pupils of the eyes are equal, and react to light and to accommodation; there is no nystagmus, strabismus, or ptosis; sight, taste, hearing, and smell are normal; the tongue when protruded shows slight tremors, and no deviation; voluntary speech is free, no

stammering nor scannings; there is some paræsthesia, especially on the left side of the body; the cutaneous sensibility to pain is increased on the same side; temperature and tactile sensibility, and the sense of pressure are normal; muscle and stereognostic sensibility are preserved; the nerve-trunks are not painful to pressure; there is a slight increase of the mechanical excitability of the nerves and the muscles; the left arm and leg show slight motor weakness in comparison with the right side, on both active and passive movements (the patient is right-handed); there are no contractions, no tremors or cramps; co-ordination is normal; the gait is uncertain only when the eyes are closed; there is a slight Rombergism; the hand-writing is not altered, nor the comprehension of spoken words; the field of vision is not restricted; catalepsy absent, even during the attacks; the conjunctival and the pharyngeal reflexes are much impaired; those between the scapulæ, the abdominal, and the plantar reflexes are uniformly dull; the jaw-reflex is not pronounced; elbow-jerk is normal; the knee, and the Achilles reflexes are more pronounced than those of the other tendons, but equal; the ankle- and knee-clonus are absent; there is an insignificant enlargement of the heart dulness to the right; the pulse is from 70–80 a minute, of a medium fulness, without any arrhythmia; the pelvic organs are normal; the sleep, the appetite, and the temper are normal.

The fits began by a slight quickening of the respiration, and a slow pulse; afterwards the pulse accelerated gradually to 70, and the breathing slowed down to 2–3 per minute. As said before, the attack was preceded by headache, chiefly occipital. After the attack severe pains began in the left side of the chest.

A description of the attack is as follows:

The face retains its normal colour, as well as the skin generally, and the mucous membranes. The patient is mostly lying on her back, and at regular intervals (20–30 seconds) gives a deep sigh. The pulse is 70 per minute, of a good fulness without any arrhythmia; the sounds of the heart are clear; the cardiac dulness in a lying posture is normal. The skin is analgesic; ammonia applied to the nose causes a grimace of disgust; at times there are slight tonic contractions of the muscles of the face and extremities, mostly on the left side; now and then she opens her eyes, looks around her, and smiles at those she sees, then falls asleep again, but when thus "awake" she does not answer any questions; when properly awake she does not remember who was near her. I related during the attack something that at any other time would have attracted the patient's attention, but when she awoke and I repeated the same, she listened as if she had heard it for the first time, and had no recollection of hearing it before. The pupils of the eyes during the attack are equal, somewhat dilated; if one opens her eyelids she does not fix any moving objects, therefore it is impossible to examine the accommodation; an ophthalmoscopic examination during the attack showed quite a normal disc. Dermographism is clear and definite on the left side of the chest; the abdominal and the plantar reflexes are brisk during the attack; the knee-reflexes, as well as those of the scapulæ and elbow, are equal, and much increased; there is a pseudo ankle-clonus on both sides. Once during the attack there was

a trace of Babinsky's symptom on the left side; the mechanical stimulation of the facial, radial, and popliteal nerves causes marked contraction of the muscles they innervate, especially on the left side. Tactile stimulation does not cause any contraction of the underlying muscles; muscular resistance is reduced on passive movements. As regards differential diagnosis: Stokes-Adam's disease was excluded, in default of any disorder of the heart. As for narcolepsy, it is usually sudden in onset and of short duration, and the patient can be roused easily by means of mechanical stimulation. As to the present case, attacks occur at irregular intervals, they last for hours, and are followed by peculiar modifications of consciousness, of the cardiac and respiratory functions, and by attacks of pain.

The patient was treated by simple heart-tonics (convallaria majal, and others), general tonics, and psychotherapy. About the middle of April the patient went abroad improved. In Berlin she had an attack, which was witnessed by Professor Oppenheim, who diagnosed lethargy. In Paris the patient was treated in a sanatorium under the supervision of Professor Babinsky.

We can draw the following conclusions from these two cases:

Both patients had not previously shown any symptoms of neurosis. In the second case during the illness there was an inequality of pain sensibility, and impaired pharyngeal reflexes. Neither had been hypnotised prior to the illness. The thyroid gland in both patients was normal. In one case the lethargy coincided with the onset of measles. In both cases headache preceded the attacks, although in the first case this could be attributed to infectious disease. In both cases amnesia was complete. Towards the end of the attack the first patient showed clearly a reaction to the mechanical stimulation of the mucous membrane of the nose, and the second patient was roused by inhaling ammonia. In neither case were cataleptic symptoms or rigidity noticed. Muscular resistance on passive movements was reduced in both cases. The attacks in the second case recurred periodically, without definite intervals. The attacks in both cases were of rather sudden onset, but the symptoms disappeared gradually. After the attack the second patient had prolonged, sharp pains in the left side of the chest, which could not be relieved by morphia. The blood-vessels of the *fundus oculi* in the first case were contracted during the attack, but in the second case they showed no alteration. The respirations were diminished in both cases, especially in the second. In the second case the pulse slowed down before the attack, but during it there were no marked deviations from normal. During the attack in both patients the tendon-reflexes and the

knee-reflexes especially were increased, and cutaneous reflexes remained normal. During one attack in the second case a Babinsky was observed on the left side. The mechanical excitability of the nerve-trunks during the attacks in both patients was usually increased, but not as regards all the nerve-trunks, nor was it always the same on both sides. Judging by the way in which the patients moved their extremities, turned over or showed resistance, however slight, on passive movement, there was no paralysis during or after the attacks. The pupils in both patients were equally dilated during the attacks, and they reacted poorly to light, the eyelids were closed, and the eye did not fix any object. The steady rising of the temperature in the first case was most likely due to the onset of measles, and the temperature of the second patient reached usually 99·5° F. at the beginning of the attack. In the first case, the urine had not been analysed before the attack, and in the second the urine was normal.

I will now analyse some of these facts. The pupils during the attack were equal and somewhat dilated, but reacted to light but slightly. Braid and Heidenhain have already noted the dilatation of the pupils in hypnotism, and the absence of reaction to light has been pointed out by Luys and Bacchi.

The increase of the tendon reflexes evidently depends upon the reduction of muscular tone during lethargy; Moll points out that, in paralysis the outcome of suggestion, the reflexes are increased, and Vogt considers that, besides the state of the muscles, the tension of the opposing group is of great importance as regards the extent of the reflexes. The following question is undoubtedly of great interest: can a reflex absent in the normal state be present in lethargy? Heidenhain caused contraction of the muscles under hypnosis by stroking the overlying skin, thus confirming Charcot's and Richer's view of the somatic reflexes. Obersteiner, Schaffer, and others have come to the same conclusion. But to this it has been objected that the increased excitability of the reflexes may not be connected with the physiological alterations of conductibility, but may be a mere matter of habit.

There have been a great many discussions over the state of consciousness during lethargy. Our observations show that in the state of profound lethargy the consciousness is greatly obscured. Our patients did not remember anything that had

been said to them during the lethargy, neither did they respond.

We observed in our second case a retardation of the pulse, and an acceleration of breathing at the beginning of the attack, and *vice versâ* during its disappearance. I did not see the beginning of the attack in the first case, and its further course was analogous to the second case in respect of pulse-rate and respirations; the size of the pulse was medium in both cases. Tamburini and Sepilli observed that at the moment of entry into the hypnotic state there was accelerated and irregular breathing, as well as an accelerated pulse-rate with a larger pulse-wave, which was greatly altered by the respiration. Horsley did not find alterations in the curve of the pulse, although the method of fixation frequently used in hypnotism can of itself produce a fluctuation of pulse and respirations, in this sense Bernheim and Preyer do not consider it possible to attribute the change in the pulse and respirations to hypnotism *per se*. Putting aside the question of hypnotism, and of the lethargic stage of hysterical attacks, we may suppose that lethargy by itself in a clearly defined form probably gives fluctuations of pulse and respiration analogous to those during a normal sleep, with which our observations agree, as well as those of Oppenheim and others. The tendency of the pulse and respirations to weaken and to be slowed in lethargy, as distinct from the normal sleep, can reach pathological proportions, *e.g.*, in Pfendler's case, when during forty-eight hours there were no signs of life. But, putting aside extreme cases, we can but acknowledge the similarity of the alteration in cardiac and respiratory activity in lethargy to the alteration of these functions in normal sleep.

The study of the tone of the muscles during suggested or hysterical lethargy gives hardly definite results; in the former, suggestion plays a great part, and in the latter, besides self-suggestion there is a blending of hysterical cramps and contractures with the symptoms of a lethargical period, which may make their differentiation difficult. A certain lessening of the tonus, observed by us in our lethargical cases, where hysteria as well as suggestion could be with great probability excluded, represents most likely the true state of the muscles during lethargy.

There is likewise a difference of opinion on the subject of

the state of the vessels of the *fundus oculi* during lethargy. In our first observation there was a slight contraction of the retinal vessels, in the second the vessels were normal. We may suppose that the vessels of the fundus oculi scarcely present any alterations characteristic of lethargy, and therefore the state of the blood circulation of the brain during lethargy remains an open question. The literature on the subject is full of contradictions. Carpenter explains the phenomena of hypnotism by anæmia of the brain; others, *e.g.*, Moll, do not find any characteristic alterations in the fundus oculi. Heidenhain gave the patient amyl nitrite to inhale, and in spite of the dilatation of the vessels obtained phenomena of hypnotism; Bacchi noticed hyperæmia, which is in direct contradiction to Carpenter's observations. The vessels of the brain can be altered by so many accidental circumstances that we cannot see in their condition the physiological cause of the lethargical syndrome; on this point it is extremely easy to confound accompanying circumstances with the essential basis of the condition.

Finally, let us touch upon the ætiological side of the disease. The main point lies in the question whether lethargical attacks in any way depend upon hysteria, whether lethargical sleep appears as a separate link in the chain of hysterical or hypnotic symptoms, or whether the lethargical syndrome can take place episodically in connection with other causes. We know that hypnotism may call forth symptoms which have nothing in common with hypnotism itself. Evidently a lethargical fit can be provoked by suggestion or auto-suggestion, but observation shows us that it can be called forth even by an acute infectious disease, or any other somatic disorder, as well as by emotion. It is possible that the subsequent attacks in our second case were partly provoked by auto-suggestion, but her first attack, as well as the lethargical attack in our first patient, can hardly be attributed to this cause. Charcot's division of grand hypnotism into cataleptic, lethargic, and somnambulistic stages was controverted by the school of Nancy, which considered these stages to be the effect of unconscious habit. Wetterstrand and other observers could not as a rule distinguish Charcot's periods. It seems that if the lethargical syndrome enters in any form whatever into the phenomena of hypnotism or

hysteria, it does not exclude the possibility of a wider view as to the ætiology of lethargy. We rather have to admit that lethargy is not an independent disease, nor even a symptom of any definite disease ; but it may be considered as a complex of symptoms connected with suggestion or auto-suggestion, which may enter episodically into the course of hysteria and of hypnotism, or it may arise as the outcome of emotion, of infectious disease, of intoxication, of auto-intoxication, or may be produced reflexly, or by somatic disease, etc. Its ætiology is evidently complex, although it depends on a single condition. The cause must ultimately act homogeneously, and on defined elements. Thus it brings on a lethargical attack, with its heterogeneous symptoms, as a unity. But wherein lies the effect of the various causes, what does the final evoking factor consist of, and on what elements does it act? Unfortunately it is impossible to give any definite answer to these questions. The vaso-motor theory that might explain the whole matter breaks down just as it does with regard to the pathogenesis of mental diseases. Most likely the vascular phenomena are not of primary but of secondary importance. The anatomico-physiological basis of even normal sleep, in spite of the investigations of Duval and others, has not yet been explained ; the difficulty of explaining it by alterations in the morphology of the cellular processes has increased since the discovery of neurofibrils. L. Loewenfeld perceives the basis of normal sleep in fatigue, in the probable cortical anæmia, and in psychical representation of sleep. Many hypotheses have been advanced in order to explain the nature of hypnotism, and the essence of lethargy. Preyer considered hypnotism as developed from the one-sided effort of attention from fixation, due to the accumulation of the products of fatigue in the corresponding sections of the brain, and to the extinction of their functions. However, to this the objection was made that the ease with which hypnotism can be produced cannot be explained by this theory, nor the prompt awakening by order of the hypnotiser, for it remains completely unknown as to where the products of fatigue disappear to so rapidly. Among other theories the physiological theory of Heidenhain, and Wundt's theory of nerve dynamical phenomena, are noteworthy. We shall not enter into the details of these theories, against which there exist likewise

serious objections. It must be admitted that the lethargical complex, brought about by various causes, must have, from the point of view of psychico-physiological parallelism, a substratum, in which dynamical or chemical molecular perturbations take place, but the true nature of these phenomena can be solved only when we are able to understand nerve conductibility, and the law of activity and rest of the nerve-elements. The anatomico-physiological correlation of the phenomena of consciousness, of sleep, of a series of neuroses, and of lethargy can be treated only when the more elementary processes have been mastered, the further study of which will throw more light on the complicated phenomena than the most promising theory.

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