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Massive Injury of the Frontal Lobes**

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TWO KINDS OF MOTOR PERSEVERATION IN MASSIVE INJURY OF THE FRONTAL LOBES

BY

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PROBLEM

It is known that focal cerebral injuries can seriously impair nervous motility and lead to pathological inertia. It is also known that this pathological inertia can show itself in different spheres: in cases of frontal lobe injury it occurs particularly in the sphere of motor functioning (Luria, 1948, 1962, 1963*a*; Filippycheva, 1952), increasing sharply in severity in proportion to the patient's degree of exhaustion, and appearing in particularly pronounced forms in periods of exacerbation, as when removal of a cerebral tumour is followed by œdema (Spirin, 1951).

This pathological inertia of motor processes may be of different kinds. In some cases it appears as motor perseveration or compulsive repetition of a movement that has been initiated. In such cases the intention governing further action remains unaffected; switching from one action to another presents no difficulty; and the programme of action is preserved. But it becomes impossible to execute the movement required, because of pathological inertia of the stimulus previously initiated. This type of disturbance may be called "efferent" perseveration (perseveration at the motor periphery).

The second type of perseveration is radically different. Pathological inertia extends here to the *programme of action*. In such cases the programme of action, once initiated, becomes inert and the patient, having once performed the required task, is incapable of switching to the fulfilment of any other task but continues, even when instructed otherwise, to perform the first task on which he has "stuck." This symptom occurs characteristically against a background of general asthenia and

as spontaneity, and may or may not be accompanied by motor excitation or by the kind of motor perseveration just described. This second type of disturbance is called inertia of a previously recorded programme of action.

There is reason to suppose that these two types of perseveration are connected with pathological inertia of different cerebral systems.

The first type may be observed in cases of massive injury to the frontal lobes, extending to the subcortical motor ganglia. Sometimes it occurs in deep-seated injuries to the posterior parts of frontal lobes in which case the symptom described appears against a background of extrapyramidal disturbance of motility (Luria, 1963*a*). Sometimes it is seen in cases of injury to the mediobasal parts of frontal lobes, again extending to the subcortical ganglia, in which case it presents as part of a much more general and massive impairment of psychological processes.

The second type of disturbance—pathological inertia of a previously recorded programme of action—usually occurs in massive bilateral injuries to the prefrontal cerebral regions, not extending to the subcortical motor ganglia: it points to a general impairment of the higher forms of regulation and of the mechanism which enables the outcome of action to be compared with the initial intention: such impairment constitutes one of the essential characteristics of pathological frontal conditions, as has been confirmed both in animals (Anokhin, 1955; Shumilina, 1949; Shustin, 1958; Pribram, 1959, 1961) and in man (Luria, 1962; Luria and Homskaya, 1963, etc.).

In this paper we will describe two cases of massive injury to the frontal lobes. The first is a particularly clear example of “efferent” perseveration, in which the programme of action is relatively well preserved. The second describes gross inertia of the programme of action, with almost complete absence of motor perseveration of the kind seen in the first case. The different localization of the injuries in question permits useful conclusions to be drawn.

Case 1.—V. V. (Massive olfactory meningioma).

V. V., aged 42, female factory worker, admitted to the Neurosurgical Institute complaining of headaches, impaired vision and general weakness.

The headaches had begun about ten years previously. In 1962 her eyesight began to fail, she became careless, irritable, inactive, greedy in eating. Sleep was disturbed and memory poor for recent events.

Clinical examination.—Bilateral anosmia, bilateral papilloedema, with secondary atrophy; vision limited in both eyes to 0·09. No sensory disturbance. Tremor of hands. Tendon reflexes on left side lively, no pathological reflexes. C.S.F. 1·65%₀₀ albumin.

Psychological examination (N. A. Filippycheva).—Orientated, aware of individual defects, though this awareness is not always complete; most marked symptom is disturbed affective attitude to illness; euphoric, coarse, behaves familiarly with doctor.

Objective tests.—Gross disturbance of concentration and attention. Carries out very simple intellectual tasks but only with constant encouragement. Easily strays from essential train of thought. Gross disturbance of voluntary and involuntary retention.

Movements slow, with marked perseveration and tendency to frequent repetition of movements that have been initiated.

EEG: Pathological focus. Slow waves in frontal regions.

X-ray: Gross deformation of the sella turcica. Angiogram: Inverse, arched bend in both frontal cerebral arteries, indicating large olfactory meningioma situated largely to the right.

First operation (S. N. Fedorov) 10.9.63.—Frontal incision revealed a large falciform tumour involving almost the entire anterior fossa.

Second operation, 4.10.63.—Resection of the pole of the right frontal lobe, after which access was obtained to the tumour, which was firmly located in the olfactory fossa. Partial removal of tumour showed that it extended across the destroyed lamina cribrosa into the nasal cavity and that at the back it reached the middle of the skull, spreading upwards to the hypothalamic region and to the region of the third ventricle. The last part of the tumour was not removed.

Neuropsychological analysis of the patient six weeks after operation showed the following picture. She was not anxious, but much exhausted. She readily set about performing the tasks set her, but after a few trials she stopped answering questions. Her attention was very unstable and she reacted to every side stimulus, turning her head towards it and interrupting whatever task she has been set. In bed she was slovenly but did not mind this. She was disorientated in place, thinking she was in the town of C., not far from her home. Knew she had had an operation but not sure where or when. Did not quite recognize the doctor when he approached her, thought she has met him in the town of C., but when he came a second time two days later she knew him and asked him "Are you going to give me some figures to draw again today? I like that." Behaved familiarly towards the doctor, joked in the course of conversation, brought proverbs into her talk, was easily diverted. Once when there was a sudden noise in the water pipes in the ward, and at that very moment the doctor took a step towards the patient's bed, she said to him "What are you afraid of, what's drowning you?" When questioned she explained that he was presumably afraid of being drowned in the water.

Her speech was not impaired; she found it easy to repeat words, or series of words and phrases, to name objects. No real defect in her understanding of words. Able to do simple arithmetical sums, but is apt to stop suddenly and refuse to answer questions.

Results of motor examination were particularly interesting. While she generally lacked spontaneity, it was noticeable that she *preserved the ability to perform motor tasks involving fairly complicated programmes of action: The defects* which appeared in known conditions, arose only *at the periphery*, taking the form of *pathological inertia of individual links in the chain of movements to be performed.*

The following examples illustrate the position:

(1) *Simple movements (either copying a movement or carrying out a movement in verbal command).* Examples: Raise your hand; form a ring with your fingers; point the second and fifth fingers, etc. The patient could do this without noticeable difficulty. There was no difficulty either in switching from one movement to another, and even more so, no "sticking" at the previous movement and thus preventing a switch to the next.

(2) The patient found it easy to copy a *series of two alternating movements* (e.g. point the second and third fingers, and then clench the fist). Difficulty in switching from one chain of movement to another appeared only after several successive attempts made at short intervals, when there was a tendency to stick at the preceding link in the chain. This difficulty disappeared after a short rest.

(3) She was also able to carry out conditioned responses to verbal instructions (e.g. in response to one tap, raise the right hand, to two taps, the left hand) although after a series of 5 or 6 tests she often raised the wrong hand: this happened particularly when the patient had developed a stereotyped response to two alternating signals and the sequence was suddenly broken (1 2, 1 2, 1 2, 2).

(4) It was easy to evoke in the patient, in response to verbal instructions, *motor reactions which did not correspond to the image before her* (allomorphic reactions). Thus, when suitably instructed, she would clench her fist when the experimenter pointed a finger, and would point a finger when the experimenter clenched her fist, never making a mistake.

(5) Difficulties arose only when the task set her was a *complicated serial one*, especially when it had to be completed without visual aids, in response to verbal instruction only.

Thus the patient could relatively easily tap out a rhythm of two or three beats in response to a visual/auditory image, switching without any noticeable perseveration, from one to the other. But if the visual image was removed and she was asked to do the same task in response to verbal instructions only (tap two beats), the required rhythm was lost; the patient would remember the verbal instruction and repeat it correctly, but would begin to tap a random number of beats, not noticing that she was inserting extra beats and not maintaining the rhythm she had been set. She would make the following kind of mistake: "Tap two beats": " " " " " ". "Is that correct?" "Yes." "Tap two beats" " " " " " " " ". "What rhythm are you tapping?" "Two beats." "And did you do it correctly?" "I don't know . . . I do everything correctly."

Attempts to surmount these defects by utilizing the patient's own speech did not meet with much success.

Analogous findings were obtained in an experiment in which the patient was asked to reproduce a known sequence of black and white dots (e.g. ●○○●○○) either by copying them or in response to verbal instruction. While she mastered the former task relatively easily, she was unable to master the latter, replacing the required programme by stereotyped alternation of a few groups or perseverating with the repetition of dots of one colour.¹

This relatively well preserved ability to carry out complicated visual programmes of action is in sharp contrast to the *gross inertia of initiated motor processes* which occurred each time the patient's movement assumed a circular character and which prevented her from fulfilling the motor programmes which she nevertheless retained. This was best seen in her drawings, and showed up clearly even when she was not particularly exhausted.

The relevant data are given in fig. 1, (Plate I).

The patient is asked to draw a small circle. She does this correctly several times (1). She is then asked to draw only *one* circle; she begins to make a large number of circular movements and is unable to stop (2). She is then asked to draw a cross and a circle. She does this, but draws each figure with a lot of perseverating movements (3, 4). She is asked to copy a circle or a square; she does this, with motor perseverations which she cannot overcome (5, 6). She is then asked to write her name (Njuza) and this she does with very gross motor perseveration (7). She is again given a complicated series of instructions—draw two circles, a cross and a window (8), or two crosses and a circle (9); she does not experience any difficulty in changing from writing to drawing, but carries

¹For a description of the changes produced in these cases in patients with massive frontal injuries, see Luria, K. Pribram and E. D. Homskaya—1964.

out both programmes with gross motor perseveration. This motor perseveration disappears to some extent if the patient is given a task that is structurally more complicated, as, for example, when she is asked to draw a house (10): she is now well able to draw successive parts of the whole structure and the motor perseveration is hardly apparent.

In the light of these facts the patient's basic defects may be summed up as follows:

A massive basal tumour involving both frontal lobes and the subcortical motor ganglia, led to a characteristic syndrome in which crude affective changes in personality and marked lack of spontaneity were accompanied by impaired selectivity of mental processes. A further characteristic is that the *programming of fairly complicated actions (especially in the visual field) was here relatively well preserved* and performance was *impaired only by pathological inertia of already initiated motor movements: this took the form of "efferent" perseveration and cannot be controlled.*

In contrast to the above we present a second case, in which massive injury on the convexity of the frontal lobe region produces a different type of disturbance of action.

Case 2.—V. Fed. (Tumour of the posterior parts of the frontal lobes at the mid-line: gross frontal syndrome).

V. Fed., aged 56, working woman. Admitted to the Neurosurgical Institute in a state of general asthenia and aspontaneity. In the course of the last five years, constant headaches, occasional attacks in which she suddenly lost consciousness, but no convulsions. For a month before admission the headaches grew much worse; usually active and stable, she became dull, lacking in initiative; often sat silent, talked to no one. When the headaches were at their worst she vomited. Brought to the Institute by ambulance.

The patient was disorientated, untidy, feels pain when she shakes her head, especially to the left. Bilateral papilloedema. Vision —0.3. No disturbance of sensation. Bilateral anosmia. No paresis of extremities, but tonus greater in the right hand. Loses balance when she stands, cannot walk alone. Right corner of mouth droops. Spontaneous bilateral Babinski reflex, grasp reflex and palm to chin reflex less marked on right.

X-ray examination.—Disparity of the walls of sella turcica. EEG: Significant overall changes: fixed pathological focus in anterior regions of the brain, both right and left. Arteriogram: arc-like bend to the right, in both frontal arteries, indicating a massive tumour in the posterior left frontal region, near the mid-line.

Psychopathology.—Patient dull, lacking in initiative, asthenic. Answers questions monosyllabically, sometimes after a long pause, often falls silent and looks at the doctor with fixed gaze. Indifferent with no clearly expressed emotional reactions, but some critical insight into her condition; sometimes corrects wrong answers of her own accord. Speech: no primary defects but occasional signs of inertia. Thus, when asked how old her sons are, she said "Twenty-eight." "Both of them?" "Both." "That means you have twins?" "Yes, twins." (One son is in fact 28, the other 26, and her answer can be taken as a symptom of pathological inertia.)

The patient can without difficulty repeat isolated words, and understands what is said to her, pointing to objects that are named (though gradually becoming exhausted). She finds it easy to name pictures presented to her, though as she becomes exhausted she takes longer to answer and sometimes perseverates instead. She can repeat easily a series of two to three words but very quickly the repetition is marred by perseveration of part of the preceding series (house, night (correct); window, cat: "window . . . and night." Snow, table: "window . . . and snow," and so on).

She can repeat automatic series of words, but when asked to say the days of the week or the months backwards, she continues to give the series automatically in its normal order. She has difficulty in naming a series of objects presented to her, e.g. five objects, all red. She cannot reproduce unaided a story that is read to her, but when questioned can reproduce its content. Intellectual capacity grossly impaired, because of her lack of spontaneity, but she can still do simple sums as long as she does not refuse to co-operate.

Motility.—She is very dull and slow in carrying out simple movements, both when asked to copy them and in response to verbal instructions; she becomes slower and slower and soon all movement ceases. She finds it difficult to execute either a series of consecutive movements or a somewhat complicated conditioned movement, particularly if these do not coincide with the image before her: the correct response was easily replaced by echopraxic repetition of the model or by perseverating repetition of the preceding movement. *Performance of a somewhat complicated motor programme is thus severely disturbed and is easily replaced by a more simple echopraxic or perseverating motor reaction which the patient does not correct.*

The following examples will serve to illustrate the position:

(1) *The patient is asked to reproduce a movement made by the investigator (clench her fist, point a finger, etc.). She does this a few times without difficulty but soon becomes exhausted and stops making the required movement. If, however, she is asked to carry out a series of two successive movements (e.g. clench her fist and point a finger), she can do this once at the most: thereafter the first movement is perseverated and she is unable to switch to the second.*

(2) *The patient is asked verbally to execute a movement (raise her hand, clench her fist, etc.). If her hand is lying outside the bedclothes she does this easily, carrying out the instruction two or three times: then the latency period increases, and after a few trials she stops making the movement, shaking her head. If she has first of all to take her hand out from under the bedclothes, in order to make the movement, she is unable to do so: her hand stays passively under the bedclothes or begins to ruffle the bedclothes. She is unable to carry out a series of two successive movements in response to verbal instructions.*

(3) *The patient is instructed to make certain movements according to a verbal instruction.*—If the movement required is neutral in relation to the signal (as when she is told "When I knock, raise your hand") she can do this two or three times, but thereafter she becomes exhausted and stops reacting in any way at all. If the movement conflicts with the signal (e.g. "When I raise my fist, you raise your finger") she does it once, but then substitutes her echopraxic reaction, raising her fist in response to a raised fist. After a few times even this reaction is extinguished.

(4) *The patient is asked to tap out a rhythm (two or three beats) and is given a model to follow which is presented in both visual and auditory modalities. She does the two-beat rhythm without difficulty, making no superfluous uninhibited movements, but cannot switch to the second rhythm: the stereotyped rhythm of two beats is so inert that she continues to repeat this even when the image is changed and is unable to switch to the rhythm of three beats.*

The motor disturbances observed in this patient are particularly pronounced when she is asked to draw any kind of figure. In contrast to the previous case, *we find in her drawings no trace of compulsive movements* or "efferent" motor perseveration. But (again unlike the first patient) she seems *unable to switch from one task to another; once stereotypy has set in, the inertia is such that the patient is unable to switch to the performance of any other action*. She continues to repeat inertly the preceding action, although such repetition serves no adequate purpose; she does not even try to correct her mistake.

Fig. 2 illustrates this.

The patient is asked to draw a square. She does this. Asked then to draw a triangle, she draws part of a square, but when she is asked to draw a circle or a cross, she continues to draw a square (1). When asked to write the letter "B," she writes "D," and after that she writes the word "Baba" (woman) as D D G E D V V D E E (apparently giving fragments of the alphabet). She is then asked to draw "two circles and a cross," in response to which she first writes "D + 2," then "20 + 20," and then "2 2 2 2." Similarly when asked to draw a cross she writes "2 2" and even when asked to *copy* a circle she writes "2" (2).

A visual example does not help her to break from the stereotyped movements once they are established, and when she is asked to copy a cross she continues to reproduce inertly a circle (3). Even when she is given a cross to trace, inert repetition of the "stuck" figure of the circle still intrudes and in consequence the patient first produces something like a flower and then a bulging cross (4). When she is asked to write her name she again writes "g g g g," repeating this again when she is asked to copy a triangle. She feels that she has not done this correctly and says "Not that," but on trying again, instead of copying the triangle she writes a row of letters underneath (5). She is then given the outline of a triangle and asked to trace it, her hand being guided over the first line: she continues by writing inertly in the framework of the triangle the "stuck" series of letter. This she does several times in succession, in spite of the fact that the investigator guides her hand in tracing the whole outline of the triangle (6). Even after a pause of thirty minutes, during which other observations are made, she seems unable to overcome this inertia but begins to inscribe whorls, resembling letters, round the outline of the triangle, or to write letters. Asking her to draw a house, or a human figure is also, of course, of no avail and she continues instead to repeat inertly the stereotype at which she has "stuck."

In this case, therefore, pathological inertia does not involve a particular link in the chain of motor activity, but *substitutes, for the correct response to a verbal instruction, the repetition of an inert stereotyped action*.

COMPARATIVE ANALYSIS: TWO FORMS OF MOTOR PERSEVERATION

Comparative analysis of the data permits us to distinguish clearly between the two forms of motor perseveration found in the two cases described.

In each case, a massive tumour of the frontal lobes has impaired normal functioning of both lobes. In each case the disturbance described takes place against a background of marked lack of spontaneity, with disorientation in place and time and disturbances of behaviour. But against this common background we find two sharply distinguished syndromes of impaired psychological functioning.

In the first case described, a large bilateral olfactory arachnoid-endothelioma was accompanied by profound personality changes which showed already in the initial stages of the illness. At the height of the illness there were characteristic behavioural changes. The patient became asthenic and lacking in spontaneity, *though she was still able to carry out relatively complicated programmes of action*. She could switch easily from one task to another, performing a series of successive motor actions. It was relatively easy to evoke conditioned actions in which the motor response was not the same as the signal. Switching from one programme to another (as in drawing a series of figures) presented no marked difficulty. Her defect became apparent only when she had to finish a movement already begun: it took the form of reinforced repetition of the action that had been begun, i.e. efferent perseveration. This phenomenon was particularly pronounced when the patient was performing a "contained" movement (e.g. drawing a circle) and disappeared almost completely when she changed to a more complicated task (e.g. when she was asked to draw a house).

The motor disturbance found in the second patient was completely different. The patient, who had a massive tumour spreading into the posterior parts of the frontal lobes, which also destroyed normal functioning of both lobes, did not initially show the affective changes, with uninhibited behaviour and disturbed personality, which we found in the first patient. At its height the illness was characterized by asthenia and lack of spontaneity, and by a different type of motor impairment. In contrast to the first patient, she was unable to carry out any complicated programme of action. She showed no compulsive movements or inability to stop movements once they had been begun (efferent perseveration). But she had difficulty in carrying out even simple movements in response to visual models or verbal instructions. She could not easily switch from one action to another and was not able to perform a series of two linked motor actions, and even less able to carry out a series of three. Once an action had been performed, it became so inert that she could not switch to the second link of the series. It was equally difficult for her to carry out any kind of conditioned response, particularly if the response required was different from the signal. This defect, caused by pathological inertia of established stereotypes, showed particularly clearly in the patient's drawings. The perseveration (repetition of the type of response evoked) was so pronounced that the patient could not carry out a motor task involving a switch from one stereotype to another. Visual examples were no help, nor was switching to a higher level of intellectual action.

Comparative analysis thus shows that though both patients had massive tumours of the frontal lobes, and showed marked lack of spontaneity, *the motor disturbances observed in the two cases were of different kinds*.

In the first case a complicated motor programme was preserved, but its performance was blocked by motor perseveration. This kind of defect is found in its most characteristic form in cases of injury to the basal parts of the frontal lobes, affecting also the subcortical motor ganglia. If the disease develops further, and elements of increased intracranial pressure are removed, it may spill over into a general syndrome of uninhibited impulsiveness, such as is often found in cases of basal lobe injuries: it does not, however, cause gross impairment of motor synthesis or of intellectual functioning.

The second case showed a gross primary disturbance in the performance of motor programmes, but no inhibition of efferent motor activity. In its most characteristic form this syndrome is seen in cases of injury to the convex areas of the frontal lobes, also affecting the posterior parts of the frontal lobes. While not necessarily accompanied by gross disturbances in the affective sphere, this syndrome always includes disturbance of complicated forms of regulation; the source of this disturbance is pathological inertia of programmes of action and inability to compare effects of action with the initial intention, which is a basic sign of injury to the convex areas of the frontal lobes.

SUMMARY

This communication examines two cases of patients with massive tumours of the cerebral lobes—a meningioma situated in the basal regions of both lobes, and the other a tumour spreading into the posterior lobes, at the mid-line.

Against a common background of asthenia and lack of spontaneity, two different kinds of motor impairment were found: the first patient had preserved relatively well the ability to perform complicated programmes of action, but these were blocked by compulsive repetition of already initiated motor movements (efferent motor perseveration): the second patient showed gross impairment in switching from one programme of action to another and inertia of motor stereotypes once they were established, though there were no signs of compulsive movements or efferent motor perseveration.

These two syndromes represent two different kinds of motor perseveration that accompany injury to the frontal lobes. They differ in their psychological character and are related to different cerebral systems.

REFERENCES

- ANOKHIN, P. K. (1955) New data on the characteristics of the efferent apparatus in conditioned reflexes. *Vop. psikholog.*, No. 6.
- FILIPPICHEVA, N. A. (1952) Inertia of higher cortical processes in cases of local injury to the cerebral hemispheres. Dissertation. Moscow: AMN SSSR.
- LURIA, A. R. (1948) Restoration of brain functions after trauma. Moscow: AMN SSR.
- (1962) Higher cortical functions in man. Moscow: Moscow University.
- (1963a) The human brain and psychological processes. Moscow: APN RSFSR.
- (1963b) "Restoration of Function after Brain Injury." Oxford
- and HOMSKAYA, E. D. (1963) *Neuropsychologia*, 1, 9.
- , PRIBRAM, K. H., HOMSKAYA, E. D. (1954) *Neuropsychologia*, 2, 4.
- PRIBRAM, K. (1959) *Behav. Sci.*, 4, 265.
- (1961) *Exp. Neurol.*, 3, 432.
- SHUMILINA, A. E. (1949) Functional significance of the cerebral lobes in conditioned reflex activity in dogs. In "Problems of Higher Nervous Activity," Moscow: AMN SSSR.
- SHUSTIN, N. A. (1958) *Z. Vyssh. nervn. Deyatel.*, 8, 246.
- SPIRIN, B. G. (1951) Impairment of nervous plasticity after cerebral operations. Dissertation. Moscow: AMN SSSR.

PLATE I

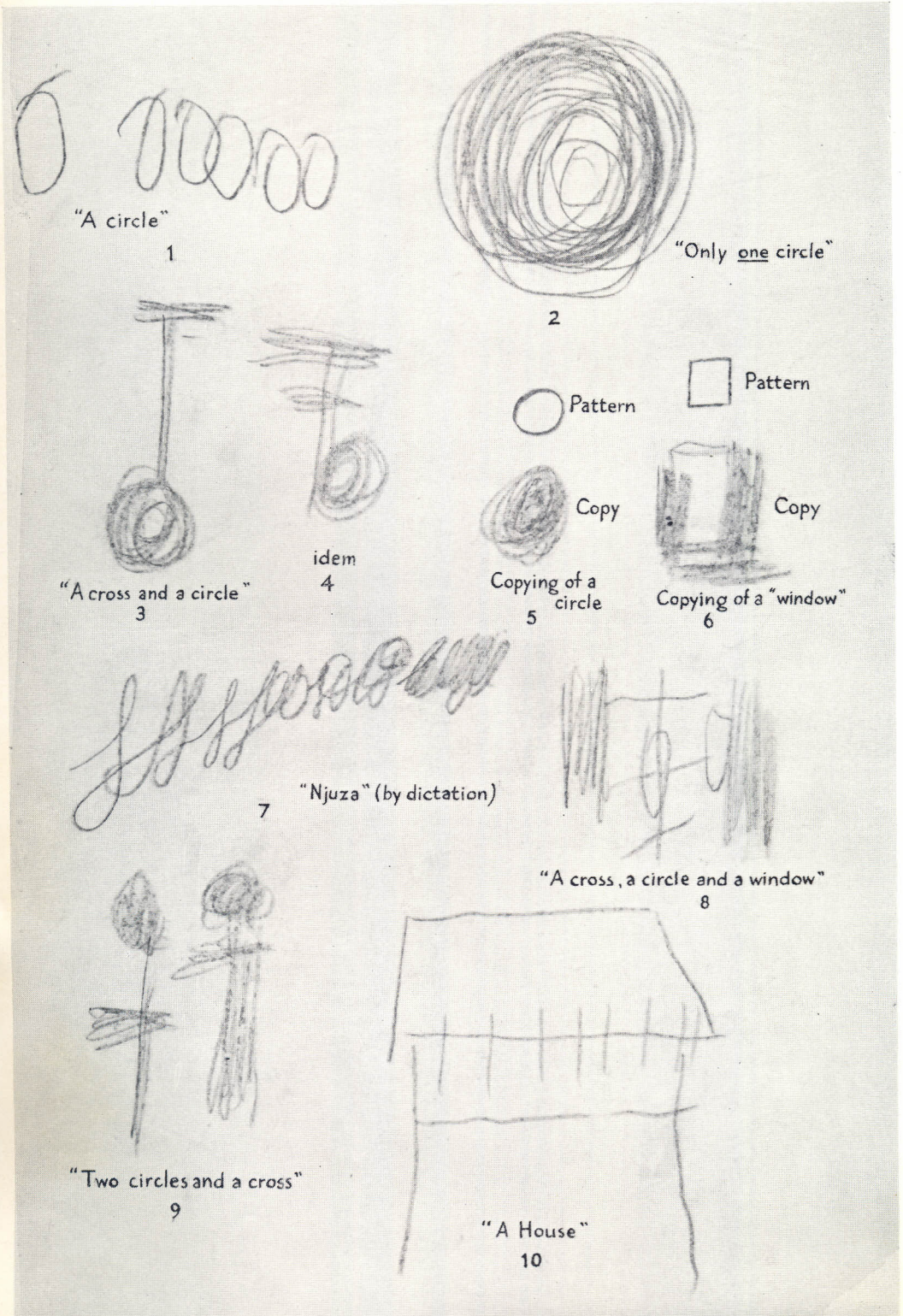


FIG. 1.

To illustrate article by A. R. Luria.

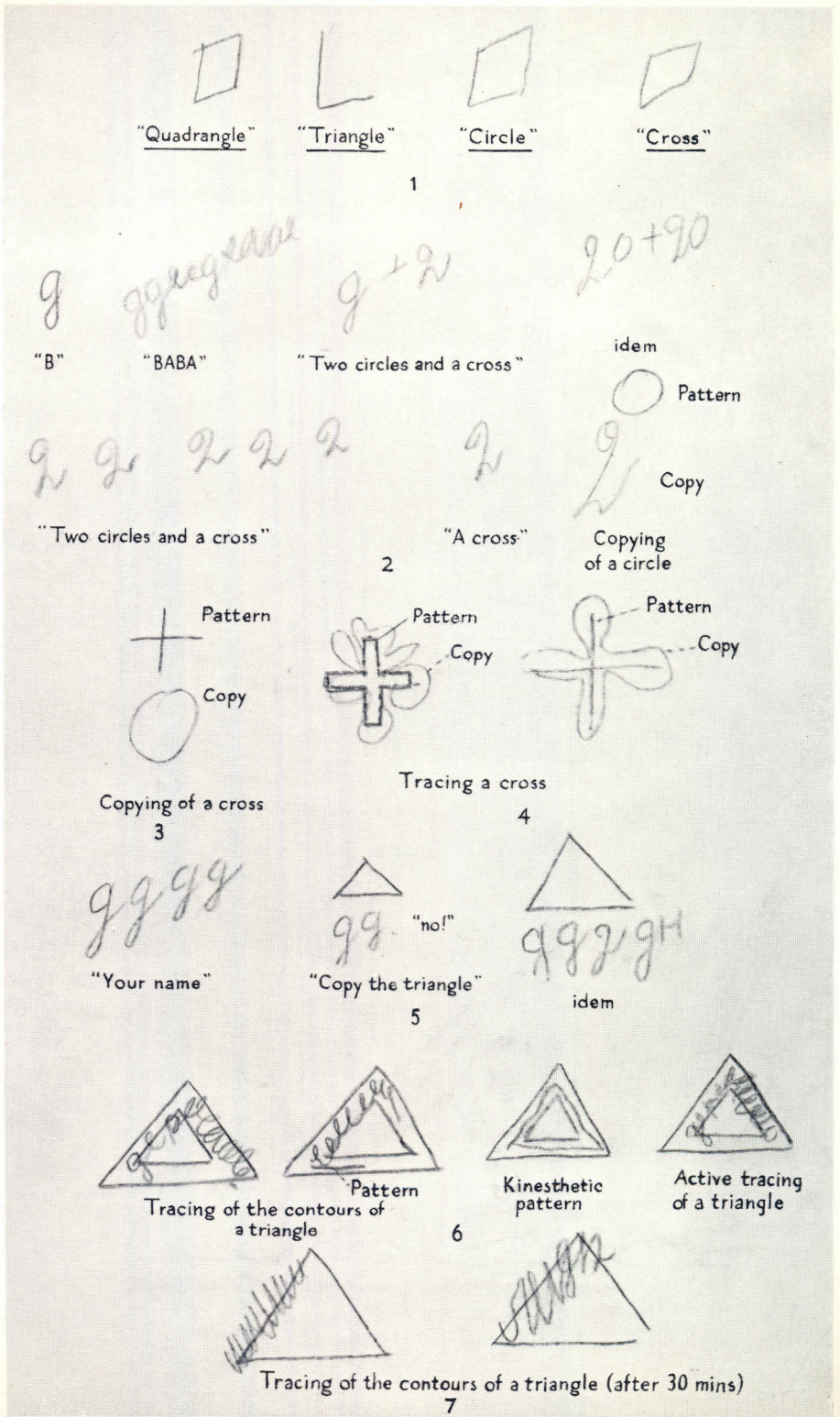


FIG. 2.

To illustrate article by A. R. Luria.

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