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Psychic Paralysis of Gaze, Optic Ataxia, and Spatial Disorder of Attention

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This paper was first published in *Monatsschrift für Psychiatrie und Neurologie, 25*, 51–81, 1909, under the title "Seelenlähmung des 'Schauens', optische Ataxie, räumliche Störung der Aufmerksamkeit."

This complex of three symptoms which I have tried to summarise in the above title was observed by me in one patient over a prolonged period of time. The three components of the syndrome had to be given new names since no detailed description of a similar syndrome could be found in the literature, although similar descriptions of a single symptom have occasionally been reported. What made it relatively easy for me to disentangle and interpret the extremely complicated phenomena observed in this patient was the fact that I was dealing with an intelligent person. This naturally also helps to strengthen the reliability of the symptoms described.

Apart from one brief interruption, we observed the illness until death. After the autopsy, the central nervous system was subjected to a detailed histological examination. The following is a brief description of the case.

The patient had always been perfectly healthy, with no evidence for syphilitic infection, and he also denied alcohol abuse. In November 1894, he suddenly suffered a dizzy spell which lasted about 15 minutes. Nevertheless he did not lose consciousness. Following the advice of his doctor he went to bed, where in the next four days he had a further attack, again without losing consciousness. When he got up again after a few days to go back to work, he realised that he had lost the ability to use his hands. The strength of his hands remained, but there had been a functional change which he could not define.

As a result of this he became unable to draw and write, which created problems in his job. At the same time he realised that his vision had

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changed. Again he was not able to characterise the change more precisely, although in addition to difficulties in drawing and writing it also affected his reading, even making it impossible in some circumstances. He was treated for four years without the slightest change or improvement of his condition. After those four years, in December 1903, he came to our clinic and had medical treatment until his death in 1906. His medical history is as follows.

GENERAL CONDITION

The patient was[2] strongly built, a little obese. There was no evidence of syphilitic infection. In relation to the facial portion of his skull he had a small, somewhat flat cranium which showed no particular asymmetry. The vegetative organs showed no major abnormalities. The arteries were a little rigid, the second aortic sound tinkling, cardiac action normal. He did not feel ill, had a good appetite with good intestinal function, and the urine contained neither albumen nor sugar.

NERVOUS SYSTEM

The muscles served by the facial, hypoglossal, and trigeminal nerves functioned normally and eye movements were accurate. Swallowing was intact. The musculature of the neck, torso, and lower extremities was in good condition and showed normal movement. The musculature of the upper extremities showed appropriate power, elementary movements were normal, and only under certain circumstances, which will be described in detail later, could functional disorders be shown. No sensory disturbance could be found anywhere on the surface of the body. Tactile sense as well as sensitivity to pain and heat remained intact over the whole skin surface. As will be discussed later in the text, the muscular sense of the extremities remained intact, as did the stereognostic sense of the hands.

The tendon reflexes of the upper and lower limbs were the same on each side, and cremasteric and plantar reflexes could be elicited. Babinski's reflex was not present. There were substantial changes regarding vision, which will be discussed later in detail. As for the other sensory organs, hearing, taste, and smell were all normal. The patient was educated in music and used to play the piano. He could not read music at the time of the examination but could apparently still play little pieces from memory after the onset of his illness. His reading will be reported later. His speech hardly differed from normal. During a conversation he sometimes used the words "my dear" and "my father" when addressing others. He noticed

[2]From this point onwards the tense has been changed from the present to the past tense.

this only when it was pointed out to him, and he apparently never used the words in this way before his illness.

His intellectual abilities seemed a little blunted, perhaps because he had not been engaged intellectually in any way since his illness. Nevertheless there were no signs of a marked dementia, and indeed he was able to solve simple mental tasks perfectly. For the convenience of the reader I shall describe the pathological symptoms in a logical sequence, and not in the order in which the medical condition emerged under observation.

VISUAL DISORDERS

The patient had a slight presbyopia, acuity with corrections right 5/5, left 5/10. The fundus of both eyes was normal.

Disorders of Visual Attention[3]

A strange phenomenon became apparent while testing visual acuity. When the patient was asked to read from the chart, he read the top letter, then the last letter of the second line, then the last of the third line and so on: always the last of each line. It was very obvious that to read any further he had to be prompted for each line separately. When asked why he always read just the last letter of each line, he expressed surprise, and asked whether there was something else on the chart. When we pointed out to him that apart from the letter he had read there were also letters to the left of it, he read those as well, after searching around for a while. This kind of reading was reminiscent of hemianopia, so we tested his visual fields using a perimeter; it was found that the fields were normal both for objects and for colours. A hemianopia was therefore out of the question.

But in any case, a closer consideration of the visual impairment described above will reveal that it was not characteristic of hemianopia; he did not read all the letters in the right hemifield, but only the last letter on the right. When questioned about this he answered that at first he only saw the top letter; when requested to continue reading, he proceeded to the line below but again saw only one letter: the one on the far right. After additional requests he went to the other lines. As noted above, when we then pointed out to him that there were other letters to the left of the ones he had read, he saw and read those as well. Once I had noticed the symptom, I began to enquire whether it was present in other circumstances. He told me that he always saw on the right only; if something happened in right space he would notice it. If it happened to his left, he would not be able to form an image of it. Nevertheless if it was pointed out to him

[3]This heading has been added for clarity of presentation.

that something had happened on the left or that there was an object there, he would notice it immediately.

We actually observed this phenomenon directly. On one occasion he was sitting in the garden of the clinic gazing ahead, seemingly unpreoccupied. Since there were carriages making noises on all sides I managed to approach his left side silently from behind and sit next to him on the bench; he did not notice anything. A few times I extended my hand into his left visual field; he noticed nothing. When I performed the same movement from the right, he noticed immediately. However, whenever I told him in advance to pay attention since an object would appear in front of his left eye, it would always be perceived immediately. I tested this phenomenon in different circumstances and always with similar success. One thing became obvious, however: Even when I tested him repeatedly on the same reading chart or with two or more objects over a very short period of time, he always reported the right letter or right object only, although, as he said himself, he knew that there were more letters (on the chart) than he could see. He always needed additional encouragement to attend to the other letters. I shall come back to this later.

From these examinations it follows that the patient's attention was constantly drawn to the right side of space, so that he saw the far right object only. The main question was now whether the object which captured his attention could be anywhere in right space or if the rightward orienting of attention was apparent only within certain boundaries. To answer this question I wrote a long row of letters horizontally on the blackboard. When asked, the patient read a letter on the right side, but not the furthest rightwardly placed letter. After several comparable experiments it transpired that the patient's attention was biased about 35–40 degrees to the right, an object located there being noticed first. During the experiment with the long row of letters I noticed that the patient would read a letter at about 35 degrees from his midline and say that he could not see any other letters. When I asked him to inspect the board more closely because there were other letters, he read all the letters to the right of the one he had read out first; then, after being prompted repeatedly, the ones placed to the left of it. From this, it became obvious that when he had to reallocate his attention after fixation, he preferred to do so to the right rather than the left.

I observed the same phenomenon on other occasions in different circumstances. For example, I would show him an object and ask him to fixate it; then I would have another object placed to the right and left of the fixated object, unknown to him. At first he would see neither of these objects, and after he was prompted he always noticed the one on the right first and only later the one on the left. Another important finding emerged from these experiments: In the patient's visual field there was space for

one single object only. As soon as this object occupied his central vision he took no notice of things lying to either side of it, and only when he was pressed, by being told to look, was he more attentive and able to see other things. The patient's visual field seemed therefore—if I may say so—concentrically narrowed. I have repeatedly convinced myself of the stability of this phenomenon in various experiments. For example I would put a text in front of him and lay another one adjacent to it on the right while he was reading; he would not notice it until I prompted him.

It is clear from these descriptions that the patient's attention or rather his central vision deviated to the right side of space. Moreover, the visual field was so narrowed that once a stimulus was in it, those to the right or left were not perceived at all. As mentioned before, the patient seemed to have a severe concentric narrowing of his visual field. Nevertheless the phenomenon cannot be interpreted in terms of a *visual field defect* since there were circumstances in which the field was of normal extent. This was shown by the perimetric testing. It seems that stimuli reached the cortex from all objects in the visual field, but that the patient did not perceive them consciously while his attention was concentrated on a single one. This means that it was not the real visual field which was constricted but rather the attentional field or psychic visual field—whichever we may call it. The nature of this narrowing will be discussed in detail later.

The psychic nature of the phenomenon is also apparent from the accompanying symptoms. Visual field defects can be so extensive that only one very small image can be seen, and when larger objects are presented the patient sees only parts. This has been observed by Förster after bilateral damage to the occipital lobes. This was not so in my patient—his visual field was not of a fixed size but rather had space for one image only. This image could be of any size and still he saw it completely; yet he did not notice other stimuli even when the fixated image was extremely small. For example, he could see a person easily, and could describe the person's size, colour of clothing, etc.; yet fixating a needle made it impossible for him to perceive a candle light placed at a 5cm distance from him.

I did the following experiment: I placed him in front of a blackboard and wrote a letter on it; after he had read the letter I drew, without his knowledge, a geometric form—a triangle—next to the letter in such a way that the letter overlapped the right side of the triangle. The patient then faced the board and after being asked what he saw, named the letter again.—*You do not see anything else?*—Oh yes, he saw it now, and named the geometric form to the left of the letter.—When now I drew the geometric form to the right of an already perceived letter, and in such a way that the letter overlapped the left side of the triangle, the patient said he saw a triangle.—*Have a closer look; do you not see anything else?*—Yes, now I see the letter as well. When I drew the letter at the top or bottom

of the triangle, he saw either the letter or the triangle, but never both at the same time. Finally I wrote a letter on the board; after he had read it I asked the patient to maintain his gaze at the place he had just seen the letter. While he did this I drew something next to it which he did not see. Even now the patient just read the letter although I had interfered with the actual letter itself; he only noticed the figure or drawing adjacent to it when I prompted him. These experiments demonstrate what we have noticed before. Firstly it can be seen that of two pictures he perceived only the rightwardly located one; secondly that while fixating an image he took no notice of objects lying on either side of it, until prompted. Furthermore independent of how small the object was, his visual field seemed to take in a single object only; and even when the object placed in his visual field was large, he perceived it adequately.

I should mention two strange phenomena apparent in these experiments. When the letter overlapped the left side of the triangle, he saw the triangle and reported the letter only when prompted. Thus while perceiving the triangle he did not notice that one side of it was intersected by the letter. This phenomenon has to be seen in the context of the patient's deficient attention. When looking at something, his attention seemed to be very superficial; even though he gained a general impression of what he saw and usually recognised it correctly, he did not want to look at it in detail. This was sometimes the main cause of gross errors. For example with the triangle he noticed at once that it was a triangle; having thus satisfied his curiosity he paid no attention to the letter embedded in the side of the triangle. When I pointed this out to him, he smiled and said: "Oh, I did not look at that." I was repeatedly able to observe this kind of superficiality or tiring of attention.

Something else which I have already mentioned but wish to emphasise again also comes out of these experiments. I repeated the experiment a few times, and in later sessions he would already know, and did know, that while he was not looking at the board, I drew something next to the letter; it is possible he even heard the sound of the chalk while I was drawing. Yet when he looked at the board he only saw one image, either the letter or the triangle; I had to tell him every single time to search more thoroughly in case there was something else on the board. Consequently he lacked spontaneity of attention; only after being prompted, i.e. with a stronger impetus, did he take notice and see.

The abnormal visual phenomena observed in the patient can be summarised as follows: The patient saw only a single object at a time; however, while perceiving that object his attention was very superficial, resulting in only a sketchy perception of the object; he only took notice of the details when asked to do so directly. He did not notice things placed near the object and did not pick anything up spontaneously; however when he was

pressed verbally, he became more attentive and noticed the other objects. The patient's attention was always biased towards right space and when, after fixating an object, he was asked to reallocate his attention to another object, this attentional switch happened more easily towards the right than the left.

Other Visual Testing

I must emphasise that all symptoms of agnosia (*Seelenblindheit*) were absent in this patient. Orienting in space was not impaired. He named objects correctly and used them correctly; he could describe the form, colour, and purpose of every object from memory; his visual memory was intact; and his colour vision was good.

Stereoscopic vision was tested in the usual way by asking the patient to say which of two objects was closer to him, which one was higher, etc.; he made hardly any errors. It was obvious, however, that solving these tasks took him a long time, and was only achieved after repeated prompting. He gave the following reason for this: "When I see one object I do not see the other one, and it takes time until I—after being told to do so—find the other one."

The patient had an interesting disorder in making distance estimates by eye. His efforts to mark the centre of a circle, rectangle, or other geometric form showed large errors. As we will see, a motor disturbance of the hand contributed substantially to this incorrect drawing: Yet he did not always notice the incorrect position of a centre he had already marked. Similarly he showed errors in the bisection of lines. He would fixate the line for a long time and then bisect it incorrectly. When he was asked to check whether the bisection was correct he fixated again for a long time and sometimes noticed the mistake. Simpler tasks such as estimating the length of a pencil or stick were generally executed correctly. Again the nature of these disturbances can only be the superficiality and extreme fatigue of his attention. He said himself that he was not capable of attending to several parts of a scene at once. For example, he could see the rectangle and the correctly placed centre but he was not capable of judging the interrelationship between the two; when he looked for the centre and fixated it, he no longer saw the rectangle. The following experiment shows how extreme this phenomenon was: I drew a cross and asked him to show me the intersection of the two straight lines. He was incapable of this, which again was probably due to the movement disorder of his hand. Therefore I modified the experiment by taking the chalk in my hand and placing its tip at several points on the board, asking him to tell me when I had hit the intersection of the two straight lines. He regularly indicated this incorrectly. I then modified the experiment further in that instead of the

chalk I used a piece of red cardboard paper attached to a rod. He was asked to say when I had covered the intersection with it. The task was now easier because of the difference in colour, but he could still solve it only rarely. "When I see the intersection," he said, "I don't see your hand, and when I see your hand I don't see the intersection. And even if I see it for a certain length of time I cannot judge the relationship of the two very accurately."

This disturbance of attention could also be demonstrated in the identification of forms. He recognised objects or images immediately. He also recognised and named simple geometric forms of an instantly obvious nature, such as a triangle, a rectangle, or sometimes even a pentagon. However, when he had to name forms which even a healthy person could only identify by counting the sides, he had to concentrate hard to do this and soon became confused.

Reading

He always read a single letter perfectly. But when a word was written on the board with its letters slightly spaced out, he saw the letter at the right end only, and only after being prompted again did he read the whole word. When I instructed him at the outset that he had to read the whole word, he started with the first letter of the word, but obviously had to search for it. As he said himself, his attention was directed from right to left until he found the right end of the word; he then looked for the beginning by tracing it back. This operation was rather time-consuming. Once having found them on the board, he could read short words perfectly. With longer words he often missed a letter or syllable, especially when he was a little tired. As he said, he recognised the individual letters but had to search for each one. In the meantime his attention fatigued and thus it happened that he would sometimes miss or interchange a letter. Evidence for this is that when the word was written in front of him and he could read each letter immediately, he never made an error, as if vision of the chalk helped him direct his attention. Similar difficulties were seen where he read from a book. When there were short simple words, he could sometimes read, especially when not tired, two lines perfectly. If there were longer words he would miss letters or interchange them: and especially when tiring, he would rather frequently jump from one line to a word in the third or fourth line. Again he said it was fatigue; to search for the next word caused difficulty and therefore he would sometimes not read that word but another one.

Something else became apparent during the tests of reading. I have pointed out that he always needed direct prompting for the recognition of letters or objects. However, when he read from a book this was not the case. On the contrary: When he was given a book and asked to read from

it, he continued reading after the first word without further prompting until he felt tired, i.e. he read and perceived more or less spontaneously. It therefore seems that the often-practised and habitual mechanism of reading was possible without external prompting. Unlike other visual acts, it was not necesary to direct his attention artificially; nevertheless his spontaneous attention fatigued rapidly. Similar phenomena could be observed for writing, as I shall indicate shortly.

While discussing the patient's visual disturbances I have pointed out that a motor disorder of the hand contributed to the symptoms. Let us now look at these motor disorders.

MOTOR DISORDERS

When describing the patient's general condition I mentioned that the muscular power of the upper and lower extremities was fully retained and that, for the most part, the patient executed elementary movements correctly. He was a little cautious and slow when walking, being anxious because of his visual disturbances. As he said, he could hear the sounds of trams and of their electric bells, but he found it hard to judge their distance and was consequently afraid that an accident might befall him. Consequently, for a long time prior to these investigations, he had hardly been out on the street. While staying in the clinic he also rarely walked around, as he often knocked against things. His usual walk took him into the garden, where he sat quietly on a bench until he had to go back to the ward. Apart from this cautious behaviour no abnormality could be observed in his gait. He had no ataxia; he did not sway when he closed his eyes; and the passive posture of one foot could be imitated by the other one perfectly.

A substantial abnormality became visible, however, in the movements of his right hand. He himself reported that while lighting a cigarette he often lit the middle and not the end. As another example, it sometimes happened that while cutting a slice of meat on his plate which he held with a fork in his left hand, he would search for it outside the plate with the knife in his right hand. He said the reason for this was that he could not see the object very well. He actually showed large errors when searching in space. Thus when asked to grasp a presented object with his right hand, he would miss it regularly and would find it only when his hand knocked against it.

When the patient had to search for the intersection of two lines with his index finger he never found it. Of course, as well as being due to a motor disorder, this error could also be partly due to his visual difficulties, since as we saw earlier, he was also unable to locate an intersection even when I indicated it to him. One fact, however, proved beyond doubt that

for this action, as well as others to be discussed below, the motor rather than the visual disorder was the dominant one; this is that all the movements performed deficiently with the right hand were executed perfectly or with very little error with the left hand.

Let us now consider this motor disorder of the right hand. All movements which Liepmann defined as reflexive movements and which involved touching certain parts of the body were performed flawlessly. When asked to do so he touched his ear, nose, and other parts of the body very quickly and without the slightest lack of co-ordination. With closed eyes, the passive posture of his left hand could be imitated perfectly with his right hand. But when I asked him to imitate the posture of *my* hand he always produced a different one. *From these experiments it is clear that only movements which required visual control were faulty*. Naturally this disorder of movement was especially obvious during drawing and writing.

Drawing

The severe motor disorder of the hand became apparent during the simplest tasks, for example while bisecting an already drawn line with another one. He always placed his intended bisection line either above or below the presented line. When asked to join two points with a line he never managed to do so. He never located the first point with the chalk. When we placed his hand and the chalk onto one point he looked for the other one with his eyes and tried to join them; the direction of the line, however, always showed a gross deviation. With the left hand he managed decidedly better, in fact he was often perfect. During this simple drawing test, as well as in the aforementioned tasks, the inappropriate movements of his right hand can best be explained as due to poor visual guidance of the movements of that hand. It was easy to become convinced of this explanation. When I took the patient's right hand and placed it on one point and the index finger of the left hand on the other point and asked him to join the two, he could always do so perfectly. This can only be due to the fact that the sense of touch, or rather proprioception, has now taken over the guidance which formerly had to be executed by the visual system.

This motor disorder also affected more complicated drawing tasks. As already mentioned, his visual memory was completely intact. He could describe the appearance of objects in great detail; yet he could not draw them. With more complicated drawing tasks he acted as follows: To draw a triangle, he drew one side of the object and then, beginning from the endpoint of that line, drew another side without lifting the chalk from the board and without fully concentrating. He now had to join the two lines with a third; this was unsuccessful, like the joining of two points. In a similar way he drew rectangles, pentagons, etc., correctly as long as he did not lift the chalk from the board; once he had taken it off he was no longer able to find his place, and he also always failed when drawing the last line.

To perform even more complicated drawing tasks was impossible for him. When asked to draw a fork, for example, the drawing consisted only of entangled lines. When trying to draw a house, his beginning seemed quite correct: he drew the roof in one movement. But while drawing the walls he lost touch with the roof, and also drew them incorrectly, even placing the windows outside the house. The patient's visual or rather attentional disturbance contributed partly to these errors; but the motor disorder dominated, as is evident from the fact that all of these tasks were executed much better with the left hand, despite its lack of practice.

Thus we can see that his mistakes during drawing were largely based on the fact that he was unable to direct the movements of his right hand using vision.

Writing

When copying as well as when writing from dictation, single letters and short one-syllable words were written correctly. No disorder was present while writing these letters and words. Nevertheless we observed the same phenomenon as seen in drawing: As soon as the patient lifted the pen or chalk from the paper or board he could no longer find the right place to continue the words. Because of this the next letter would be placed either above or below the previous one, and similarly with the next word. When asked to write above a line he was not capable of keeping at the same height. The word on the paper was always sloping. Although he wrote very slowly and clumsily with the left hand there were none of the aforementioned mistakes.

When writing longer words it sometimes happened that he interchanged two letters or perhaps omitted one. When it was pointed out to him that he had made an error, he noticed the fact. He said that the reason for these mistakes was that while writing each letter he had to concentrate so hard that he sometimes did not see the previously copied letter and therefore missed the place where he had to continue. We are dealing here with a memory error which occurred due to the concentration of attention in another direction. This in turn was caused by the lack of visuomotor control.

Thus we have now seen that the motor disorder of the patient while drawing and writing was caused by the fact that he was not able to guide the movements of his right hand through vision. It seems that the writing and drawing difficulties were attributable to these more primitive disorders of the hand.

It has to be asked now if the disturbance was of a higher, associative character or simply a disorder of co-ordination. There are two higher

associative disorders which come to mind; one is optic agraphia, the other is apraxia. Agraphia can be ruled out for several reasons. First of all, the disorder could be demonstrated not only for writing and drawing but for all movements; secondly the patient's visual memory was intact, which is not the case with optic agraphia; and what finally excludes the possibility of agraphia is the fact that all visually controlled movements were performed rather accurately with the left hand.

Concerning apraxia, it could be argued that the connection between visual memory and kinaesthesis might be broken; yet the patient's movements were not apraxic, only unco-ordinated. He did not execute another purposeful and co-ordinated task instead of the required task, as an apraxic would do. His movements were purposeful; he made the movement he wanted to make, but in an unco-ordinated way. That this was the case was also shown by the fact that he could draw the shape of a fork with his finger in the air; in this case the connections between the individual lines were not so crucial. The motor disorder of the right hand was therefore an elementary inco-ordination, a disturbance caused by the disruption of a sensory component; and this component, which plays a crucial part in the co-ordination of movement, was visual.

The lack of visual control in this motor disorder is comparable to the disturbance of proprioception in the case of tabetic ataxia. In a tabetic patient, movements controlled by the muscular sense are affected; in our patient, however, it was those controlled by the eye. The tabetic patient replaces the lack of muscular sense with vision and can thus correct his movements; our patient, however, replaces the lack of visual control with his tactile and muscular sense. Consequently our patient is lacking one component of motor co-ordination: the visual component. The motor disorder is a disturbance of co-ordination: An ataxia which, in order to distinguish it from other ataxias, I would like to call *optic ataxia*.

DISCUSSION[4]

I have not found a description of these impairments in the literature, either as a group or as separate symptoms, although traces of each symptom could be found. Therefore I would like to discuss each symptom in detail.

All of this patient's symptoms were related to the act of vision and an explanation should therefore be found in an impairment of the visual sphere. However, in this case, the visual sphere not only includes the periphery, the optic nerves, the primary centres, the optic radiations and those cortical areas which J. Müller encapsulated as the visual sensory system (*Sinnsehsubstanz*), but the whole complex of cortical areas which

[4]This heading has been added for clarity of presentation.

serve the visual system, from light entering the eye right up to the conscious visual image.

We have to assume that the so-called visual sensory system was intact in our patient. The pathological changes therefore have to be located in that part of the visual system which enables stimuli to pass from visual cortex to consciousness, that is in the psychic part of vision. The disorder in this psychic part is, as we have stated, an attentional impairment. Calling this a disturbance of the visual sphere could be questioned, given that it is an attentional impairment. The patient's attention, however, was not impaired per se; a stimulus directed at the other sensory organs, even a very subtle one, would be noticed immediately. He paid perfect attention to everything that was not related to vision.

Only with regard to visual stimuli had his attention been altered, and only these failed to enter consciousness; this is the reason I have called the attentional disorder a disturbance of the visual sphere.

These considerations make it clear that we have reached the border between rather firmly-founded physiology and the rather shaky grounds of psychology and speculative philosophy (or a combination of those two); it is therefore tempting to enter the psychological domain while explaining this case. However, for the time being, I intend to discuss the clinical and pathological relationships only, and shall therefore avoid any psychological interpretation, saving it for another occasion.

The patient's attentional disorder manifested itself in the fact that he noticed only one object at a time and failed to attend to others. However, even a single object was examined only superficially, its details as well as things surrounding it being noticed only after prompting. This phenomenon testifies to a crucial weakness of attention. Attention is a tonic cortical function; it is sometimes stronger and sometimes weaker, but always present to a certain degree, allowing us constantly to notice things surrounding us. If our interest is aroused by a conscious image—I am talking about vision now—we increase our attention. This tonic attention was severely weakened in our patient, with the consequences that only the strongest stimuli, that is, macular images, entered his consciousness; and even with those images his attention seemed superficial. Objects lying at the periphery were ignored; that is, although the inputs reached the centre of vision correctly, he was unaware of them. The patient could have seen, but he did not "look"; that is, he was lacking in the voluntary element of perception. However, it was not lacking completely. When asked to do so he looked and could see, noticing the details of an object and the objects surrounding it; but he never did so spontaneously. It can thus be said that *he was lacking the spontaneity of vision, or that this was weakened.*

A similar disturbance of spontaneity of perception has not been described previously. However we do find a similar phenomenon in the

motor sphere. Bruns was the first person to observe and describe this phenomenon in a patient. His patient did not have a paralysis as such, but used his right arm so infrequently that it conveyed the impression of a completely paralysed arm. On request, however, any movement could be performed accurately with the right hand. There was therefore no paralysis: only a lack of spontaneous action. Bruns called this condition "psychic paralysis (*Seelenlähmung*)." Similar phenomena have been observed by Anton, Oppenheim and others in their patients.

From clinical observation and autopsy, Bruns explained his case in the following way. Voluntary movements are just higher reflexes achieved by cortical associations; these associations are in turn influenced by varying sensory factors taking messages from the relevant extremity. When—as with his patients—these sensory factors break down and the major part of the pathways leading to the motor centre have atrophied, a state can develop which to superficial observation gives an impression of paralysis although it is in fact a lack of voluntary movement of the extremity. When the remaining pathways are somehow aroused, for example through acoustic associations (verbal prompting), the patient can perform the movements.

Is the state of my patient comparable to this? One disturbance presents itself in the motor sphere, the other in the sensory sphere. But I do not think that this difference is crucial. Closer observation shows that neither of the two is really rooted in a disturbance of the motor or sensory sphere.

In one case movement, in the other case perception, can proceed flawlessly under certain conditions. The disturbance is therefore located among higher cortical functions and an impairment of these higher functions results in one case in a disturbance of movement and in the other in a sensory disorder. In psychological terms, this higher cortical function is called will. Attention is a function depending on will: It forms the voluntary component of sensation, carrying all the signs of voluntary function (Wundt); and when one talks about spontaneity of attention (or vision) one is also talking about a disturbance of will. This is similarly true for a disturbance in the spontaneity of movement.

Consequently there is no major difference between the two. Of course, the physiological explanation of the two phenomena cannot be identical as in our case the disorder cannot relate to a lack of associations with respect to a particular sense organ. It is nevertheless very likely that the disorder is based on a breakdown of associations in this case as well, especially if the following consideration is kept in mind.

It is a well-known phenomenon that we do not notice anything happening in our surroundings while being absorbed in the close inspection of something; focusing our attention on a certain object may occur to such an extent that we cannot perceive other objects placed in the peripheral

parts of our visual field, although the light rays they emit arrive in full at the visual areas of the cerebral cortex. Consequently, in order to notice something it is not sufficient that the light rays trigger activity at the cerebral cortex; this would only require the integrity of the so-called *sehsubstanz* (visual sensory system). On top of that there is a psychological component, and the physiological equivalent of this component, namely association, is also necessary. Mental activity is based on associations. The aforementioned example demonstrates that associations can be concentrated in a certain direction only at the expense of other associations; when, as in this case, other associations cannot be constructed, perception of peripheral stimuli fails to occur.

Considering this, the state of our patient could be explained as a breakdown of large parts of the associative pathways connected with the visual act. The remaining associative pathways were excited by the strongest stimuli, which are those that enter the cortex through the macula. The patient perceived the images coming from these stimuli, and did not notice the weaker stimuli from the periphery. Nevertheless when, through prompting, the remaining associative pathways were channelled with the help of other (acoustic) associations, then—as in psychic motor paralysis—weaker peripheral stimuli could enter the patient's consciousness as well.

To render this explanation acceptable it has to be shown that the patient's visual cortex was intact and only the associative pathways disrupted; the lack of field defects is in favour of an intact visual cortex, but only the autopsy which I will mention later proved that the visual cortex was anatomically intact and that the associative pathways were damaged.

I would like to point out one last thing which is similar in both motor and sensory psychic paralysis. Brun's patient could not use his right hand voluntarily, yet he performed movements requiring less conscious volition without prompting; for example he used the hand while getting out of bed. My patient, who had to be prompted in order to notice single letters or objects, could read from a book without prompting as long as he did not tire of the attentional demands. He did not need new associations to exercise the well-practised mechanism of reading.

In the light of these discussions we can call the phenomenon whereby the patient did not look or gaze spontaneously a *psychic paralysis of gaze*.

Some previous authors have mentioned a disorder of spontaneous attention, but only in relation to the patient's whole attentional system. Thus Liepmann writes of his famous apraxic patient that his spontaneous attention for everything around him was weakened, but when he was spoken to, one got the impression of a perfectly healthy person. In that instance, however, the patient's attention was weakened for all kinds of stimuli.

I found a phenomenon similar to the one in my patient, i.e. only for vision, in a case described by Hartmann. Hartmann writes as follows:

"Even after repeated testing of the patient's visual field there was a peculiar visual impairment in that the patient sees the objects only when they are pointed out to him although he is not really hemianopic. There is no interest or attention concerning objects in the impaired visual field; it has lost its content value." The patient thus behaved similarly to ours, though only on one side. Hartmann merely mentions this symptom, he does not discuss it in any detail.

The visual disturbance in my patient also included the phenomenon whereby the patient only noticed the object on the far right in a group of several objects: his attention was always directed to the right side of space. We also noticed that this tendency to direct attention to the right was present within certain boundaries only and did not exceed 40 degrees.

[Two pages omitted here in which Bálint draws a parallel between his patient and Loeb's experiments with dogs.]

There is little to say about the third factor of the clinical picture. As we mentioned, it manifested itself as a lack of co-ordination of the right hand during visually controlled movements. We therefore have to assume that the phenomenon was produced by a disruption of those pathways that connect the cortical centre at which the visual stimuli arrive with the motor centres of the hand.

[One paragraph is omitted summarising the further history of the patient.]

AUTOPSY

[Four pages are omitted which give a detailed description of the lesions in the brain.]

To summarise the changes seen in the brain at post-mortem: Both hemispheres were characterised by extremely softened tissue and part of this softening was almost exactly symmetrical. The softening involved parts of the cortex and underlying white matter including the centrum semiovale. The localised symmetrical softening was mainly in the posterior parts of the parietal lobes. On the left the posterior part of the inferior parietal lobe was destroyed; the angular gyrus and the posterior parietal gyrus completely, and to a lesser degree the superior parietal lobule and the first occipital and second temporal gyri. There was also damage in the post-central gyrus, and to a lesser extent in the upper part of the pre-central gyrus (Fig. 1). On the right also, the inferior parietal lobe suffered the greatest destruction; the occipital portion of the superior parietal lobe was atrophied as well as the upper part of the 1st occipital gyrus and the dorsal part of the 2nd temporal gyrus, the softening also extending to the supramarginal gyrus (Fig. 1). The white matter showed large defects in the

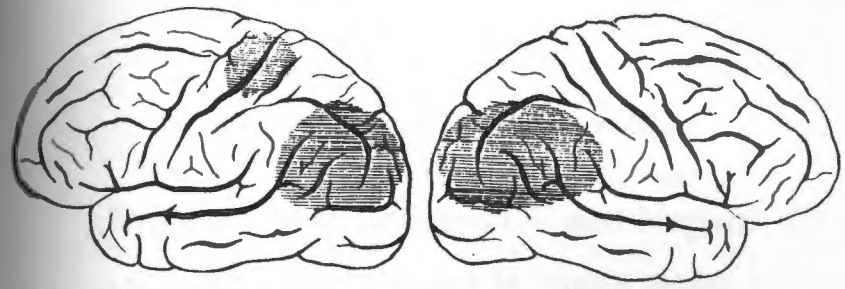

FIG. 1. The left and right hemispheres of Bálint's patient. The shaded areas indicate the locations of the principal lesions as determined at autopsy.

centrum semiovale on both sides. In dorsal sections this softening extended anteriorly up to the frontal lobe on both sides, but in sections cut below the parietal sulcus it involved the parietal and occipital lobes only to the extent of partially damaging the sagittally running fibres in the white matter. The softening was deeper on the left, more superficial on the right; on the left minor defects could be found even at the level of the upper parts of the thalamus, whereas the corresponding sections on the right showed no such softening. As far as the sagittally running fibres are concerned, only the dorsal layer was damaged, the ventral and possibly also the middle layers remaining completely intact.

Apart from these primary softenings we have found other lesions that I would like to mention briefly. One of these was degeneration of the left internal capsule, which was visible in the retrolenticular part of the capsule. On the right, damage in this retrolenticular part of the internal capsule was only slight. We also found defects in the dorsal part of the corpus callosum on both sides, and in the pulvinar and the region of the ventral nucleus of the thalamus, these being more pronounced on the left than the right. Finally we would like to point out a band of degeneration extending between the temporal and occipital lobes on the left.

[Last five pages omitted in which an attempt is made to link the findings with those of others and relate each one of the symptoms with the lesions found.]

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[5]The author has been unable to trace the Müller, Bruns, and Hartmann references cited in the original.