Finger Agnosia and Isolated Agraphia — A New Syndrome

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(From the Psychiatric-Neurological University Clinic in Vienna, received on January 25, 1927)

In 1924, in the special issue of the *Wiener Klinische Wochenschrift* published on the occasion of the Innsbruck meeting of the "Society of German Neurologists", I described under the term *finger agnosia* a peculiar phenomenon, which I regarded as a localized disorder of body orientation. It presented as follows:

In a 52-year-old female patient, objective examination revealed, as a prominent symptom in the clinical picture, an isolated loss of the ability to orient herself with that quasi-automatic precision regarding the individual fingers of both hands, a skill she demonstrably possessed prior to the onset of her illness, in a manner consistent with the norm. With eyes open, she was unable—or unable with accuracy—to differentiate the individual fingers from each other. She repeatedly made errors and mistakes in recognizing, naming, pointing to, and selecting the fingers in question, without being aware of her incorrect responses.

This disturbance was paralleled by an analogous impairment in the recognition, naming, and orienting differentiation of the fingers on the hands of other persons. She also showed, as a consequence, a certain lack of spontaneity in individual finger movements—although the fingers were otherwise completely intact in terms of motor and sensory function—and some difficulty in their separate use during actions.

With respect to other body parts and limbs, recognition and orientation abilities were essentially unchanged. There was only—albeit to a lesser degree—a disturbance in the recognition and orientation regarding left and right on paired parts of her own and others' bodies.

1. Based on a lecture given at the Association for Psychiatry and Neurology in Vienna on December 14, 1926.
2. *Wiener Klinische Wochenschrift* 1924, No. 40.

With regard to other body parts and limbs, her ability to recognize and orient remained essentially unchanged. Only one further disturbance was present—albeit to a lesser extent—namely, an impairment in recognizing and orienting between left and right on the paired parts of both her own and others' bodies. This corresponded to a marked uncertainty in choosing sides, particularly when performing crossed reaching or pointing movements.

In addition, the following symptoms were observed:

- A pure agraphia, presenting as a severe and general loss of writing ability, including difficulties with initiation, line guidance, letter formation (with confusions, compressions, and distortions of letter shapes), and with spontaneous and dictation-based writing being more affected than copying.
- A right-sided hemianopic restriction of the visual field, with varying severity.
- A severe loss of calculation ability, especially involving disturbance of place value concepts when writing or reading numbers.
- Finally, a mild impairment of memory, especially for digits and numerical relations.

Finger agnosia and isolated agraphia formed the cardinal features of the clinical picture. In my case, the patient was suffering from cerebral arteriosclerosis. She had already experienced a stroke one year prior to the observation, which had caused right-sided paralysis and loss of speech, but these disturbances had completely resolved within a few weeks.

However, the symptoms of finger agnosia and agraphia emerged much later, during the observation period. These were accompanied by complaints indicating the onset of a new area of cerebral softening. The hemianopic visual field deficit also appeared as a result of this presumed new softening.

Her reading ability was preserved, except for some slight difficulty in following long sentences or passages aloud. No aphasic, apraxic, or other agnostic symptoms were present during the observation period. This allowed for the clear manifestation of a syndrome composed primarily of the symptoms of finger agnosia and isolated agraphia, a combination previously unknown.

It also appeared that the severe disturbance of arithmetic ability was closely related to this syndrome.

Shortly after my original description of finger agnosia, Pötzl and Hermann were able to identify this symptom—also in a clearly marked form—in their well-known case of agraphia. They reported this in detail in their recent monograph.

In their case, a tumor in the parieto-occipital convexity was diagnosed, confirmed by surgery and later by autopsy. From the beginning, the main clinical symptom had been pure agraphia. Later (before surgery), as the tumor caused increasing localized damage, an elective disturbance in the recognition, pointing to, and naming of the individual fingers of both hands appeared, which—according to the authors—"at least very largely" corresponded to what I termed finger agnosia.

Other details of my observation matched well with the (preoperative) clinical picture of Pötzl and Hermann's case, in which, among other features, a left-sided hemianopia and

a severe disturbance of arithmetic ability were also present. This supported the assumption of a common localization in the brain for the underlying pathological changes responsible for these symptom complexes.

In my case, I considered it probable that a focal cerebral softening had occurred in the area of the left inferior parietal lobe, especially near the angular gyrus. However, due to the lack of an anatomical diagnosis, I was unable to determine a more precise localization.

Pötzl and Hermann, on the other hand, based on their autopsy-confirmed findings, associated the symptom complex of finger agnosia and isolated (or pure) agraphia with the lesion located in the transition area between the right angular gyrus and the second occipital convolution. They thus attributed the probable left-hemispheric softening in my case to the corresponding cortical region in the affected hemisphere.

In my previously cited publication, I had characterized this peculiar loss of orientation ability in the fingers of both hands as a purely agnostic phenomenon, arguing that the accompanying—though relatively minor—impairment in finger movement precision was a secondary consequence of the selective recognition disorder.

However, Pötzl and Hermann interpreted the phenomenon of finger agnosia—while retaining the same term—as an isolated innervatory-apraxic disorder, meaning a primary impairment of the freedom of individual finger movements, accompanied by a corresponding alteration in the ability to freely orient to them.

What led me to regard the symptom as purely agnostic were mainly the following considerations:

1. The impairment in recognizing and selecting fingers on others, which coincided with the disturbance in recognizing one's own fingers, and which could logically be seen as a natural consequence of the latter.
2. The circumscribed disturbance in naming the individual fingers of both hands, despite no signs of aphasia.

Of course, one must acknowledge that orientation on the body, as well as among its various parts, can also be impaired by apraxic disorders. In such rare cases, however, the impairment is usually limited to recognition of one's own body, while the recognition of others' bodies remains intact or is not similarly affected.

In my case, and as far as I can tell also in that of Pötzl and Hermann, there was a clear congruence between the disturbance in recognizing one's own fingers and that of others' fingers.

Moreover, the selective disturbance in naming fingers did not seem plausible as a purely apraxic phenomenon, while it naturally arose from a primary recognition disorder.

Pötzl and Hermann, in their above-cited monograph, pointed out that in parietal lesions, gradations of damage to the same brain region can produce variants ranging from agnostic to apraxic forms. They suggested that quantitative differences in a lesion within the transition zone between the angular gyrus and the second occipital convolution could give rise to a purely agnostic, an amnestic, or an innervatory-apraxic type of the same fundamental disorder, which I had termed finger agnosia based on the main symptom in my case.

It is difficult to reject the view of a researcher as experienced in neuropathological questions as Pötzl. One must then assume that there is no essential difference between interpreting finger agnosia as a primarily agnostic or as a primarily innervatory-apraxic disorder of an elective nature—that is, the two perspectives refer only to different appearances of the phenomenon, not to its core nature.

I am now in a position to report on two additional cases exhibiting the symptom of finger agnosia, which I have had the opportunity to observe recently. A closer examination of these cases shows that the co-occurrence of finger agnosia (along with left-right disorientation) and isolated agraphia represents a central syndrome within the clinical picture—analogous to what was already apparent in my initial report and in the case observed by Pötzl and Hermann.

To be sure, there are gradual differences in the severity of this symptom complex.

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The medical history of the first of the two cases is, in summary, as follows:

F.M., 56 years old, a seamstress, was referred to us by the First Medical Clinic on September 16, 1926, for investigation due to a writing disorder and remained under my observation for more than six months. The patient, who previously had flawless and very fluent handwriting, reports that approximately 4½ years ago she suddenly lost the ability to write—without any concurrent disturbance of consciousness. Her language production and comprehension remained completely intact. She did not perceive any disturbance in reading or in understanding what she read. No other symptoms of illness were present; she did not notice any motor or sensory impairments in her arms or legs and was able to continue her previous occupation without difficulty. She had not noticed any particular clumsiness in her fingers when sewing, threading needles, etc.

Her main complaint is the loss of writing ability, which has remained unchanged for

about 4½ years. The only words she has been able to write since then are her first name and occasionally her son's name; otherwise, she is unable to express any word in written form. Writing numbers is also difficult, and she reports severe problems with arithmetic. She has never experienced any visual disturbances. — No other relevant medical history. There is no indication of any renewed cerebral episode since the initial insult. No known history of syphilis.

As for the objective clinical findings, it is first noteworthy that, aside from a slight reduction in memory and recall, there were no general psychiatric abnormalities. The memory impairment mainly affects information involving numbers or numerical relationships. There are pronounced deficits in the ability to operate with numbers and to orient herself in numerical contexts. Her arithmetic ability is severely impaired—especially in multiplication and division, less so in subtraction, and least in addition. She also exhibits difficulties in reading numbers and even more pronounced issues in writing them. The former (reading) only becomes apparent with four-digit numbers or longer, while the latter (writing) appears even with two-digit numbers and almost consistently with three-digit numbers. Two- and three-digit numbers are still read relatively well.

A four-digit number like 7684 is read as "seventy sixty-four" or alternatively as "seven six hundred eighty-four."

For instance:

- The four-digit number 7684 was read as "seventy-six eighty-four," and then again as "seven six hundred eighty-four."
- A five-digit number like 95321 became "ninety-five thirty-two," and then "nine five three hundred twenty-one," and so on.

When writing three-digit numbers, although the individual digits are usually written correctly, the last two digits are often reversed in the order corresponding to the spoken form—for example, dictated numbers like 573, 629, and 493 are written as 537, 692, and 439, respectively. Even with two-digit numbers, it is not uncommon for the ones digit to be written before the tens digit. In four- and five-digit numbers, the first one or two digits are often omitted or swapped, or written as round hundreds or thousands, while the last two digits are written in reversed order (i.e., ones before tens). Copying numbers proceeds without difficulty. However, assembling number tiles into multi-digit numbers is impaired.

In the domain of cranial nerves as well as motor function and sensation (touch, pain, temperature, localization ability, discrimination, deep sensory modalities, stereognosis), no disturbances could be found in any other body regions.

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Spontaneous speech proceeds without the slightest disturbance. Serial repetition and repetition of spoken phrases are fully intact. Word and language comprehension, as observed in repeated examinations, is completely preserved in every respect. No paraphasias are present. There is no notable difficulty in word-finding.

Praxis (i.e., the ability to carry out purposeful motor acts) shows no abnormalities—neither in terms of sensorimotor abilities, reflexive actions, expressive movements, nor object-related actions, whether from memory or when using presented objects.

In color sense testing, variable errors appear in recognition and, accordingly, naming, particularly with the colors red, orange, and violet. These colors are most commonly confused when sorting wool samples. However, optical object recognition and naming of visually presented items are otherwise entirely unimpaired. Likewise, no gnostic disturbances can be demonstrated in the domains of other sensory modalities.

Reading of printed or written letters, words, sentences, or entire passages is fluent, without notable paralexic distortions. Reading impairment for words only appears when the patient is presented with nonsense syllables or complex/foreign words, and even then, inconsistently. While reading from a newspaper or book, she often omits or rearranges articles and prepositions, and occasionally misses the initial or final syllables of longer or less familiar expressions. However, common words and terms are always read correctly. Occasionally, there is difficulty in line progression and uncertainty in line continuity. Overall, comprehension and overview of the read material are consistently preserved, regardless of whether it is print or cursive.

The primary symptom is a profound agraphia affecting letters and words. In repeated examinations, spontaneous writing is found to be virtually abolished. Attempts to write "from within" (i.e., self-initiated) always fail, except for her own first name and that of her son, which she produces only in highly distorted forms—typically omitting, substituting, or conflating final letters; often she fails to go beyond the initial letters. Most of what she produces in spontaneous writing bears little resemblance to actual letters and usually consists of a series of short or long shadowy and stray lines, which she often discards, abandoning further writing attempts shortly thereafter.

Of all the letters, she can spontaneously write only a capital and lowercase "a", as well as an "f"; upon further prompting, she perseverates these same letters. Writing from dictation is similarly impaired. Only a few dictated letters—such as "a", "m", "f", "r", and occasionally "g", "v", "b"—are sometimes produced correctly; other letters are either severely distorted or cannot be written at all.

When tested on word dictation, she completely fails. Perseveration frequently sets in—usually on her signature or the initial letters—along with repetitive responses, so persistent that any further writing attempt is thwarted. Even during periods when perseverative responses are not particularly dominant, she remains unable to correctly write even very short and simple words from dictation, despite being able to recite and read them perfectly in all written forms.

At best, she manages a distorted reproduction of some initial letters; usually, however, she produces only letter fragments, curved or slanted strokes, and then gives up further attempts. In striking contrast to this severely impaired spontaneous and dictated writing is a relatively well-preserved ability to copy, both from cursive and printed text. Perseveration occurs only rarely or not at all in these cases. She copies words and short sentences (in both cursive and Latin script) by continuously referencing and imitating the model.

The ratio of correct to incorrect responses during copying is highly variable. She is often unable to assemble given letter tiles into words, even though she can accurately and meaningfully read both the presented letters and the intended target words beforehand.

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The writing disorder is not limited to the right hand. The left hand also exhibits complete failure in repeated writing attempts. No indication of mirror writing was ever observed. When asked to recognize words or letters traced on her hand with a finger by the examiner, she made gross errors, even though her sensitivity was intact. It was found that the patient was unable to "draw" letters in the air with her feet, and likewise unable to describe letter shapes through head movements, despite understanding the task.

She was unable to correctly write punctuation marks, such as a comma, question mark, exclamation mark, colon, or brackets—though she could read them well. She was also incapable of drawing basic geometric figures (like a triangle or rectangle), despite being able to accurately describe their appearance; a circle was the only shape she could reproduce correctly.

She was further incapable of drawing simple objects (e.g., a chair, table, key, fish, pin, fork, etc.) in a recognizable way, even though she could readily recognize and name these objects or their images when shown. Copying pre-drawn figures, however, was significantly more successful, and much like her writing, she relied heavily on direct imitation of the visual model.

This impairment in drawing ability, particularly of simple geometric or object figures, was considerably less pronounced than her writing impairment. Similarly, the

previously noted impairment in number writing was also of much lower intensity than the impairment in word and letter writing.

Repeated examinations revealed that both the impairments in drawing and in writing numbers were not quantitatively parallel to the loss of writing ability. There was also the impression that qualitatively, they were largely independent phenomena. A review of the examination protocols seems to justify the conclusion that this cluster of impairments—at least to a certain extent—can be attributed to a different factor, and may perhaps be related to the apparent primary disturbance of arithmetic ability.

Alongside the near-complete agraphia, another striking and consistently observed symptom dominates the clinical picture:

There is a severe impairment or complete loss of the ability to recognize, name, select, and indicate individual fingers on both hands—not only the patient's own fingers but also those of the examiner. This ability remained completely preserved for all other parts of the body.

The patient is unable to determine where the individual fingers are on her hands in the given moment—which is the index, middle, or ring finger, etc. She appears disoriented, constantly confuses finger names, and cannot distinguish the fingers as individual units. She makes repeated mistakes when asked to grasp, point to, extend, or separate specific fingers, either directly or by imitation—despite motor and sensory function being completely intact upon close examination.

She appears confused and unsure, often reaches incorrectly, misidentifies fingers, or searches without success, showing a clear loss of fine control and precision in isolated finger movements.

This limitation becomes evident in tasks that require independent finger use, and it affects the index, middle, and ring fingers more than the thumb and little finger.

When she focuses intently, she is sometimes able to correct errors involving the thumb and little finger, though only hesitantly and inconsistently; with the other fingers, even intense visual attention often fails to yield correct identification.

Typically, it takes multiple repetitions of a task before she can sometimes respond correctly—if at all. Even repeated verbal instructions often do not improve her performance.

Nonetheless, her comprehension of the task is consistently intact, as evidenced by her ability to carry out similar tasks involving other body parts without error.

When asked to point to or name other specific body parts, she does so correctly, promptly, and without mistakes, indicating that her recognition and orientation abilities for other body parts are unimpaired.

Learning attempts and exercises, designed to improve her finger agnosia, were unsuccessful and often resulted in a worsening of the disorder.
Closely associated with the described finger agnosia is another symptom—though less pronounced:
An impairment of the ability to recognize and orient left and right—both on her own body and on others.
This is most evident in tasks that require left-right discrimination, particularly with paired body parts such as the hands and fingers.
She shows notable difficulty and uncertainty when asked to distinguish sides during tasks involving left-right terminology, such as pointing to the opposite eye or ear with a specific hand or finger.
She often fails to complete the task correctly, despite clearly understanding and verbally repeating the instructions.
She becomes confused about which side is which, mislabels left and right, or reverses directions.
For instance, she might use the correct hand to point to the wrong (ipsilateral) side, or use the wrong hand to point to the correct contralateral part.
These same side-selection errors occur when the examiner demonstrates a movement and asks her to replicate it.
Even in tasks that mention only one side, such as "point to your left eye with your left hand," she may still make orientation and recognition errors, though these are less frequent and less intense than those seen in crossed pointing or reaching tasks.

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The clinical picture described remained essentially unchanged during a months-long observation period.

The case history of the second patient is summarized as follows:
K.F., a housewife, is a 50-year-old patient suffering from arteriosclerosis, who was under observation at our clinic from September 30 to December 24, 1926.
According to the medical history, the previously healthy patient suffered an apoplectic stroke about a year ago, which was followed by a transient (lasting several weeks) hemiparesis on the right side and a brief, mild speech disturbance.
Since then, no other stroke-like episodes appear to have occurred.
During her stay at the clinic, she exhibited the following clinical picture, which showed no significant change during the extended observation period:

In the objective clinical findings, in addition to a general intellectual weakness of moderate degree, there is a marked impairment of memory and recall, particularly concerning numbers and temporal relationships.
There is also a significant inability to perform operations with numbers.
Calculative ability shows major deficits; multiplication and division are significantly more affected than other arithmetic functions.
In addition, her reading and writing of numbers is clearly impaired.
She can usually read two-digit numbers correctly.
For example, she initially read the number 987 as eighty-seven, then as ninety-eight, and finally correctly.
On another occasion, she read 758 as "seventy-five eight", then as "seventy-five and eight hundred", without being able to correct the error.
In the same session, she read 1926 as "nineteen and twenty-six", and shortly afterward read 2467 first as "two hundred and forty-six", then again as "twenty-four and seventy-six".
When writing numbers, she occasionally reverses the order — for example, placing the units before the tens:
she once wrote 52 as 25.
She wrote the dictated number 164 as 146, 789 as 70098 ("seven hundred ninety-eight"), and 1345 as 10003054 ("one thousand thirty..." etc.).
However, this reversal of digits is not constant, but it appears in most multi-digit numbers when written from dictation.
Copying of three- and four-digit numbers is usually done correctly, especially when she can refer to a visual model.
The assembly of number tiles into multi-digit numbers is impaired.
Her spoken language abilities, as such, are entirely intact.
Serial and repeated speech remains unchanged.
There is only mild bulbar indistinctness and a slight palilalia (repetitive speech); speech is occasionally explosive in nature.
Word and language comprehension is unaffected, both in ordinary conversation and in specifically directed testing.
There are no signs of literal or verbal paraphasias in her spontaneous speech or during object naming, nor any other form of naming disturbance.
No significant word-finding difficulties were observed either.

There were no significant changes found in the area of the cranial nerves, the upper and lower extremities, or the trunk regarding motor function (gross strength, active mobility, muscle tone, reflexes, etc.) or sensation (superficial and deep sensory qualities, localization ability, discrimination, etc.).
Only the gait is somewhat disturbed, presenting in the form of a "marche à petits pas" (short-stepped gait).
There is, despite preserved sensation, an isolated difficulty and uncertainty in tactile recognition of objects with the left hand, while the right hand is unaffected in this regard. However, the disturbance is highly variable and mild in degree.
Additionally, there is an impairment in color perception for red and some related hues, with fluctuating errors in their recognition and confusion during sorting of colored wool samples.
Otherwise, as repeated testing of object recognition and similar tasks has shown, there are no agnosic symptoms — whether in the visual, tactile, or other sensory domains.
Praxic abilities show no disturbances.
More complex purposeful movements proceed just as smoothly as simpler ones.
There are no observable abnormalities in expressive gestures, object manipulations, or descriptive hand actions.
The most pronounced and dominant symptoms in the overall condition are:

1. An isolated severe impairment of writing ability.
2. A selective loss of the ability to recognize, differentiate, and name the individual fingers of both hands,
   along with a disturbance of left-right orientation in paired body parts — all qualitatively similar to the previous case, though less pronounced quantitatively.

As for the behavior of the agraphia, we again find (throughout the observation period) the striking contrast between relatively well-preserved or only slightly impaired reading ability and severely impaired writing, as was evident in the previous case history.
Reading shows only minor changes, of the same type seen in the first case:
During reading aloud of printed or handwritten sentences or passages, there are frequent omissions or confusions of articles and prepositions,
occasionally omission of a syllable at the beginning or end of longer or more complex expressions,
sometimes also difficulty with line progression or skipping lines.
However, reading as a whole remains fluent,
familiar words and terms of daily use are read correctly,
and comprehension of what is read is always preserved.

The writing disorder manifests primarily as an inability to correctly assemble letters into words during spontaneous writing and writing to dictation,
even though the patient can correctly pronounce, understand, and read the words.
Repeated testing reveals various paragraphic distortions, perseverative responses, omissions, transpositions, confusions, and conflations of letter forms and syllables, nonsensical letter combinations, etc.
These errors recur consistently, though with varying frequency and intensity, and are more pronounced in spontaneous writing than in writing to dictation.
Handling of the writing instrument is unproblematic.
Most letter forms can be written correctly both from dictation and spontaneously —
except for the uppercase and lowercase d, g, n, r, t, y, x,
which the patient typically cannot reproduce correctly from memory.
Copying from a visual model is, in proportion to the severity of the agraphia, relatively better than in the previous case.
With continuous reference to the model, the patient is able not only to copy, but to some extent also to convert printed letters into handwritten script.
The errors during copying vary from trial to trial, manifesting as occasional omissions or mergers of letters and syllables (particularly in longer or more complex words), and occasional repetitions.
However, these errors never approach the severity of those seen during spontaneous or dictated writing, where no visual reference is provided.
Assembling letter tiles into words is impaired, while reading and spelling them is generally error-free.
The patient is unable to produce punctuation marks correctly, either spontaneously or to dictation, although she can recognize them when reading.
For example:

- For an exclamation mark, she draws a vertical line with a dot beside it.
- For a colon, she makes two dots side by side instead of stacked.
- For a semicolon, she draws a horizontal line followed by a dot.
  Tracing these symbols from a model is carried out correctly, consistent with this pattern.

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The writing disorder affects both hands.
There are no signs of mirror writing.
The patient is unable to maintain a horizontal line direction when writing, deviating

irregularly either slanting downward or upward.
When asked to trace letters in the air with head movements or with her feet, she knows what is being asked, but does not know how to initiate the movements.
Drawing simple geometric or linear figures, and drawing simple everyday objects from memory, is generally impossible or inaccurate, although recognition and naming of these items and their images is always intact.
When asked to draw a triangle, she only produces two lines converging downward, forming an acute angle;
the same happens when asked to draw a square — despite having been shown the shape shortly beforehand and recognizing it promptly;
a circle is drawn approximately correctly.
When asked to draw objects such as a pin, knife, hammer, or scissors, the patient is completely at a loss.
Copying from a model is performed more successfully, as long as the patient keeps her eyes constantly on the model.
Another notable symptom is an isolated disorder of recognition, naming, pointing to, and selecting the individual fingers of both hands.
This symptom — like the agraphia, which throughout the patient's clinical stay dominates all other local symptoms — has the same character as in the previous case, but with somewhat less intensity.
In contrast to the writing disorder, for which the patient has full insight, she lacks awareness of her finger orientation disorder.
This only becomes fully apparent through objective examination.
The patient is unable to identify with certainty the thumb, index, middle finger, etc.,
is not properly oriented regarding location and sequence of the fingers she visually inspects,
cannot distinguish them on demand, constantly mixes them up,
regularly points to the wrong one,
frequently mixes up the names when asked to name each finger.
There is no sensory or motor impairment of the fingers in either hand,
nor is there any disturbance in language production, comprehension, or task understanding.
The phenomenon affects both the patient's own fingers and those of others in exactly the same way.
For example, although the patient can recognize that another person has fingers,
she fails completely or is entirely at a loss when required to select, point to, or name

individual ones like index or middle finger.
Left and right fingers are affected equally.
This disorder is remarkably consistent.
As in the previous case, the index, middle, and ring fingers are more affected than the thumb and little finger in the various tests.
Focused attention and repeated task instructions usually have no corrective effect.
Practice often only worsens the disorder.
In contrast, recognition and orientation for other body parts and limbs is completely intact.
The patient can identify, point to, and name each body part promptly and without error.
However, there is also an impairment in orientation and distinction between left and right,
especially concerning paired body parts, notably hands and fingers,
leading to marked uncertainty in recognizing, naming, and choosing the side — both on her own body and on others.
In addition to her inability to orient to individual fingers, the patient frequently confuses left and right on these parts.
However, these laterality errors are milder compared to the finger recognition deficits.
In tasks that require independent use of individual fingers, there is often some difficulty in utilizing the fingers separately, with a decrease in the precision of finger movements.
Finger coordination does not proceed freely.
In crossed pointing or grasping movements with hands and fingers towards various body parts or objects in space, there is — despite good understanding of the task — noticeable uncertainty and difficulty in localizing left and right, and frequent reversal of directions.

A critical review of the clinical pictures in the two cases just described, along with my first previously reported case and the analogous case published by Pötzl and Hermann, leads to the conclusion that the finger agnosia and isolated agraphia must be considered the central phenomena of interest.
While in the last-mentioned case, after surgical removal of the tumor damage, both symptoms regressed, the long observation period in my cases (in addition to the anamnestic data) confirms that, in the context of persistent cerebral lesions (e.g., softening), these symptoms can become long-lasting deficits.

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In the first of the two cases reported above, the agraphia was nearly absolute.

Not only was spontaneous writing and writing to dictation of words entirely impossible, but even individual letters could mostly not be written at all or were written incorrectly, despite intact pronunciation and comprehension, and the ability to read them remained unaffected.

In the second case, the combination of letters into words was severely impaired, while the individual letter shapes could still be reproduced from memory onto paper with varying accuracy.

Punctuation writing was also significantly impaired in both cases.

Copying was far less affected than spontaneous writing or dictation, and could only be carried out in close reference to the visual model.

In the first case, copying of both words and letters occurred only in the exact script form of the template.

In the second case, transferring printed letters into cursive handwriting was still somewhat possible.

Even drawing simple figures showed clear deficits — despite intact recognition of those figures — whereas tracing from a model was much better achieved.

There was, however, no clear correlation between the severity of this impairment and the degree of word or letter agraphia.

Similarly, impairment in writing numerals was relatively minor compared to letter agraphia, becoming apparent only with multi-digit numbers, mainly as errors in digit order and place value.

The finger agnosia in these two cases essentially had the same character as that in my first case:

a primary disorder of recognition, naming, pointing to, and selecting individual fingers of both hands — of one's own body as well as others' — leading consequently to a reduction in the precision of individual finger movements and in the freedom to use them independently in action.

In my first case and in the Pötzl-Hermann case, this recognition disorder affected all five fingers equally, while in the two present cases, a certain gradation could be observed — namely, the index, middle, and ring fingers appeared to be more affected than the thumb and little finger.

This pattern seems to align with the common observation that, even under normal physiological conditions, the identification, naming, and pointing out of the thumb and little finger tends to be more prompt than that of the other fingers.

In addition to finger agnosia and isolated or pure agraphia, another noteworthy symptom was an impairment of the ability to recognize and orient with respect to right and left on paired body parts, especially the hands and fingers.
This disturbance was significantly less pronounced than the aforementioned phenomena and was not as consistently present, but it appeared to be very closely linked to the finger agnosia.
Naturally, it led to varying degrees of difficulty and uncertainty in side discrimination when performing tasks involving left-right concepts, such as pointing and grasping movements with the hands and fingers toward contralateral parts of the body.
Isolated agraphia and finger agnosia appeared in the clinical presentation as cardinal symptoms, around which the other symptoms clustered irregularly and variably, more as adjacent or peripheral phenomena.
While the writing impairment and the other manifestations were clearly perceived as pathological by the patient, there was a lack of awareness of the deficits in finger agnosia and the accompanying right-left recognition disturbance.
The co-occurrence of finger agnosia with isolated or pure agraphia proved to be a consistent finding in the relevant cases.
Moreover, a notable parallelism between the two disturbances could be observed.
Even in my first observation, their association appeared likely, as periods of greater severity of agraphia coincided with more pronounced manifestations of finger agnosia.
This co-occurrence was particularly marked in the tumor case reported by Pötzl and Hermann.
Before surgery, pure agraphia was the core symptom.
During that period, finger agnosia was also observed.
With the general postoperative improvement of symptoms, the agraphia regressed almost completely, and at the same time, the finger agnosia disappeared.
Of the two cases reported in this paper, finger agnosia was more pronounced in the patient who exhibited a higher degree of agraphia.
In the second patient, where the agraphia was relatively milder, the finger agnosia—despite her more generally reduced intellectual capacity—was also less prominent than in the other cases.

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As for the remaining clinical symptoms—whose relatively variable occurrence I previously described as adjacent or marginal phenomena in comparison to agraphia and finger agnosia (along with the associated disturbance in right-left discrimination)—the

following pattern may be reported:

While in my first case as well as in the Pötzl-Hermann case, a hemianopsia developed during the further course of the illness, no visual field deficits could be detected in the two cases presented above.

In the first two cases, there were also disturbances of body balance, with a marked tendency to fall backwards or towards the side contralateral to the lesion, while no changes in body coordination were demonstrable in the latter two cases.

In the Pötzl-Hermann tumor case, motor and sensory hemiphenomena were present, whereas in my cases, no changes in basic motor or sensory function were observed during the observation period.

In one of my cases—the second case described today—a pure tactile recognition disorder limited to the left hand was observed, although only to a mild degree. In the other cases, tactile gnosis remained completely intact.

In the Pötzl and Hermann case, a color agnosia was documented; this was considered an indirect symptom of the tumor. A similar disturbance was also part of the clinical picture in the two present cases but was absent in my first case.

In none of the four cases was reading ability significantly impaired. Least of all in my first case, in which—despite the severe agraphia—the ability to read was well preserved, with only a difficulty in maintaining lines during prolonged reading.

In the Pötzl and Hermann case, the reading disturbance was more pronounced. However, it remained secondary to the agraphia, which represented the cardinal symptom of the clinical picture, and only emerged later, as tumor pressure and damage expanded.

The reading disturbance in their case consisted primarily of the patient frequently reading incorrect words or word parts, although overall comprehension of the printed text was not demonstrably disturbed.

In the two cases presented above, reading ability was somewhat more affected than in my first case, but still significantly less impaired than in the Pötzl-Hermann case. Comprehension and overview of the text were not disturbed, and it was uncommon for the patients to read correct and meaningful words (known to them from before the illness) incorrectly; only articles and prepositions were occasionally misread.

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In all four cases, there was a severe impairment of calculation ability, appearing as an apparently independent disturbance. This was combined with a disturbance—varying in degree—of the comprehension, writing, and reading of multi-digit numbers, particularly affecting the sequence and positional value of individual digits.

Aphasic, apraxic, and other agnostic disturbances were absent both in my first case and in the two cases presented here, in which softening lesions in the parieto-occipital brain region were likewise assumed, and they did not occur during the observation period. Only in the case reported by Pötzl and Hermann did apparent indirect tumor effects manifest as difficulty in word-finding in the form of amnestic aphasia, as well as episodic apraxic symptoms.

It can be said that in the four cases discussed here (leaving aside the co-occurring left-right disorientation, which will not be further addressed here), the phenomena of finger agnosia and isolated agraphia, in contrast to the variability and relative inconsistency of the other symptoms, appeared as shared phenomena.

They presented themselves in the clinical picture as the most consistent and prominent components, and they clearly stand in a close—though still unclear—relationship with each other.

It thus appears justified to consider the selective loss of the ability to recognize, name, point to, and select individual fingers on both hands, in combination with an isolated loss of writing ability in the sense of pure agraphia, as a distinct syndrome of cerebral disease.

In discussing the two newly presented cases of finger agnosia, I have described the associated writing disturbance—just as I did in my first case—in equivalent terms as isolated and also as pure agraphia.

The first expression may be regarded as unprejudiced, as it is supported by the separate presence of the writing disturbance, the lack of any identifiable causal relationship to other complicating accompanying symptoms, and the marked dissociation in my cases between the functioning of writing and that of speech, action, recognition, and even reading.

The second designation—"pure agraphia"—is, as is well known, still controversial, and is subject to certain limitations due to the long-standing and still unresolved disagreement over whether so-called pure agraphia should be regarded as an independent clinical phenomenon.

For those authors who deny the validity of distinguishing a true agraphia and instead attempt to subordinate all writing disturbances to other functional disorders, I would note that by using this term, I did not intend to express any classificatory tendency, but rather to highlight the combination of finger agnosia with agraphia, which manifests in a distinct and isolated form.

For the majority of authors, however, who acknowledge the existence of true agraphia in principle, the above description of the case histories and objective findings provides

both formally and substantively sufficient evidence that this is indeed a case of true agraphia.

The writing disturbance in these cases displays—clearly and distinctly—all the features that have been attributed to pure agraphia since Wernicke.

In one of the two cases, where the ability to reproduce letter shapes in writing was already lost, the writing disturbance essentially presented as the literal form of agraphia.

In the other case, where the ability to combine letters into words during writing was primarily impaired, it took the form of verbal agraphia in Wernicke's sense.

The two forms appear to be gradual variations of the same cerebral lesion.

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There appears to be little reason to reopen the problem of pure agraphia at this point—especially as this complex of questions has only recently been subjected to a thorough treatment by Pötzl and Hermann, who carefully drew upon the relevant literature and integrated their own observations.

The results of the writing assessments in my three patients agree so closely with both the previously published cases of isolated or pure agraphia (by Bastian, Wernicke, A. Pick, Erbslöh, Forster, Berger, Kramer, Böttiger, among others), as well as with the findings in the aforementioned case of Pötzl and Hermann, that the characterization of pure agraphia as a distinct and unified disorder—as offered by these authors (following earlier researchers)—and their differentiation of it from other types of writing disturbances seen in broader cerebral disorders can be fully applied to my cases as well.

Two features of the agraphic disorder in my cases deserve particular emphasis:

- First, it did not manifest as a loss of writing ability in one hand, but rather as a disturbance of the writing motor engram for all effectors, regardless of which limb was used.

  This generality of the writing disorder is, according to Wernicke, an essential characteristic of pure agraphia.

  Pötzl and Hermann also place primary emphasis on this and identify it as a key distinguishing feature compared to apraxic agraphia, which typically shows a limb-specific distribution and often accompanies other dyspractic disturbances.
- Secondly, in my cases, copying from visual models was noticeably more successful than spontaneous writing or writing to dictation.

  As Pötzl and Hermann, in agreement with Wernicke, point out—and as my own experience confirms—this pattern clearly distinguishes pure agraphia, even when reading ability is impaired, from the writing disturbances seen in

subcortical alexia, where the relationship between copying and spontaneous/dictated writing is typically reversed.

To these features should be added the marked isolation of the agraphia in my cases, which remained demonstrable throughout the observation period, as well as the absence of complicating aphasic, apraxic, or related symptoms, and the only slight impairment of reading ability—all of which align my cases, from a clinical standpoint, with the purest forms of agraphia reported in the literature.

The association of agraphia with lesions in a specific, bilaterally organized cortical apparatus has been further clarified by the work of Pötzl and Hermann.

The cortical regions previously implicated in agraphia—the Exner area at the foot of the second frontal gyrus, the Wernicke-Pick area at the junction of the first temporal and parietal gyri, and the Déjerine area at the junction of the angular gyrus and the second occipital gyrus—are now, based on the cited study, understood to be nodal points of a longitudinally arranged cortical writing network, analogous to what is seen in praxis.

Lesions to these areas are most likely to result in isolated or pure agraphia.

The core characteristics of this type of agraphia appear consistently, whether the lesion lies in the temporo-parietal/angular-occipital region or in the frontal part of the central writing area.

The accompanying symptoms, of course, vary accordingly.

It is from the pattern of these additional symptoms and their linkage to the agraphia that the localization of the cerebral lesion can be inferred.

Pötzl and Hermann have thus used the co-occurrence of pure agraphia with finger agnosia, among other findings, as diagnostically decisive for localizing the main lesion in their case to the transitional region between the angular gyrus and the second occipital gyrus.

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The present paper is essentially devoted, on the one hand, to highlighting the apparently consistent co-occurrence of finger agnosia with an agraphia that is isolated from other related disorders, and on the other hand, to discussing the peculiar symptom of finger agnosia itself.

I would now like to turn to the consideration of the nature of this phenomenon.

As has already been stated in my earlier publication, I regard this symptom as a primary loss of the ability to recognize and name the fingers of both hands, both one's own and those of other individuals.

The two cases reported today offer further confirmation of this interpretation.

I have referred to the phenomenon of finger agnosia as a partial disturbance of the ability to recognize and orient oneself with respect to the own and others' bodies, and I have attributed this manifestation to a pathological, selective disturbance of body awareness, specifically of the so-called body schema.

I believe that this conceptual approach offers the clearest path to understanding the nature of finger agnosia.

It therefore seems appropriate to address the question of the body schema briefly at this point.

The theory of body awareness and its disorders due to cortical lesions can be traced back, as is well known, to A. Pick and Head.

Pick referred to the ability to orient oneself to one's own body as "autotopography".

Head, in turn, coined the very apt term "body schema" and introduced the concept of a "postural model" in a characteristically descriptive way.

Nevertheless, this theory has received surprisingly little attention over the years, as reflected by the scarcity of studies addressing this issue.

Most recently, Schilder has undertaken a monographic treatment of this complex of questions, drawing on a series of interesting clinical cases.

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By the body schema is meant the perception or internal representation of our somatic self—the inner image of our bodily domain, the spatial map we carry within us (probably not at the level of full consciousness, but outside of central awareness) of our own body and its parts in their morphological and spatial relationships to one another.

This spatial image of the body is predominantly visual, but impressions and experiences from other sensory domains—especially tactile and kinesthetic sensations—also contribute to its formation, in a manner that reflects the functional importance of the individual body parts.

The body schema represents, in its entirety (in the words of A. Pick), "the essential framework for the awareness of our corporeality."

We may assume that the body schema, or spatial body image, is not a uniform, undifferentiated whole.

Rather, it appears to be structured according to individual regions and subregions of the body, in their sequential and adjacent arrangement and mutual spatial relationships—exhibiting a pronounced somatotopic differentiation.

The body schema can be understood as a complex of closely interconnected sub-schemata (sub-models) that reflect the various bodily experiences, each represented individually,

and differentiated according to the functional value of the parts they represent.
Damage to one such sub-schema would accordingly abolish or alter recognition of the corresponding body part.
There are numerous observations supporting the existence of such a body schema.
Among these, one particularly well-known empirical fact stands out: that after amputation of a body part (such as a limb or part of a limb, a breast, etc.), the missing limb is still perceived by the patient in various forms and for varying durations after the event.
The sensation of continued possession of the amputated body part is often initially so intense that the patient may completely overlook the fact of the amputation.
For instance, a person may fail to recognize the loss of a leg, reach for it, attempt to stand up in the morning—and fall.
Clearly, after removal of a body part, the corresponding spatial image tends to persist for some time, even though it is cut off from peripheral input.
It fades only gradually, as the ongoing sensory stimulation that normally maintains the corresponding schema ceases following amputation.
In cases where amputation occurs in early childhood, or is congenital, such phantom limbs are absent, according to A. Pick—likely because the missing body part was never fully represented in the body schema.
In an observation by Head, a patient's phantom limb disappeared following a cortical lesion localized to the centroparietal region.
This demonstrates that the awareness of corporeality, the spatial image of the self, the body schema, can undergo specific alterations due to cortical brain damage.

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It lies in the nature of the matter that disturbances of the body schema and its utilization are prone to lead to peculiar deficits in the domain of body recognition and orientation with respect to one's own body. The first observations of this kind come from A. Pick, who termed this disorder *autotopagnosia*. Patients who remain oriented in the external world show a loss or impairment of the ability to recognize and locate parts of their own body; they are unable to consistently know where individual body parts are located. In a given moment, they do not know how to correctly point to or name the eyes, ears, mouth, chin, nose, shoulder, elbow, etc., and they appear hesitant and perplexed when faced with this task. In two of A. Pick's cases, the disturbance of orientation also affected body parts such as the head and torso.
Additionally, in such cases, there is a variable degree of disturbance in the orientation,

recognition, and distinction between right and left on one's own body. Rosenberg has referred to this particular aspect as *chiragnosia*. Thus, these patients are agnosic—each to varying degrees—regarding their body parts and their laterality. Together with the deficits in recognition and orientation on their own bodies, there is typically a consequential uncertainty in recognizing and orienting toward the corresponding body parts of others, and in distinguishing and selecting between right and left in external space.

Pick associated this disturbance with cortical disorders in the region of the parietal lobe. A number of clinical observations—especially those by Anton, Hartmann, Pötzl, Bonhoeffer, and Schilder—are available that compellingly support this localization.

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It is to be expected a priori that, apart from disturbances affecting body awareness as a whole due to cortical lesions, the body schema, according to its division into different body regions, can also be affected in a localized, circumscribed manner; thus, isolated disturbances in the recognition and naming of specific body parts, i.e., localized forms of autotopagnosia, may occur.

The observations presented here now demonstrate a special case of such a selective disturbance in the orientation capacity mediated by the body schema, namely in the form of the described finger agnosia.

This symptom should therefore be understood as a loss of the corresponding part of the spatial body image, a damage to or isolation of the area of the body schema assigned to the fingers.

It is as though the optical–tactile–kinaesthetic spatial representation for the individual fingers—what one might call the "finger schema"—has been separated from the overall body schema.

Clinical experience shows that other circumscribed forms of autotopagnosia, different from the one discussed here, are extremely rarely observed in clear form.

Since I began paying attention to these matters, I have only encountered a single relevant case: a brain tumor patient who showed a recognition and orientation disorder restricted primarily to the eyes.

However, I must admit that, upon closer critical examination, the findings in that case cannot be considered definitive.

In contrast, I have observed the phenomenon of finger agnosia in three cases with softening lesions localized to the same cerebral region, clearly manifested within a relatively short time period, and Pötzl and Hermann have likewise observed this

symptom clearly in two brain tumor cases.
(It should be noted that one of these is the often-cited case involving agraphia; the other was recently demonstrated by Hermann and Kerschner from Pötzl's clinic at a meeting of the Prague Medical Society.)
It appears, then, that among the clinical manifestations of isolated recognition and orientation disturbances of the body, finger agnosia represents the main or at least the most frequent variant.
Why this should be the case, I cannot satisfactorily explain.
Perhaps the region of the body schema related to the fingers of both hands occupies a special position compared to other sections, in the sense that localized cortical lesions, depending on their extent and severity, may alter body awareness or the spatial body image either more globally or in the selective form of finger agnosia.
Given the more refined and differentiated structure of finger function and their cortical representation, the hypothetical assumption of such a special status seems permissible.

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For the assessment of the position of finger agnosia within the context of more complex neuropathological phenomena, it appeared necessary to me to conduct examinations in cases with general psychological disturbances—especially those with intellectual and memory deficits, as well as patients presenting with aphasia, pure word blindness, apraxia, object agnosia, and similar conditions.
These examinations focused on the behavior related to recognition, differentiation, naming, and pointing out of the individual fingers of both hands.
Insofar as the patients' overall condition did not prevent them from understanding and responding to verbal instructions, my findings thus far have shown that in none of the relevant cases was there a significant impairment in finger recognition or differentiated finger naming, when compared with physiological norms.
Therefore, finger agnosia must be regarded as a direct symptom independent of those other disorders.
Further investigations on this subject are currently underway.
It is certainly well known from personal experience that even under normal conditions, occasional mistakes in localizing, selecting, naming, or pointing out individual fingers of both hands—as well as in determining their right-left orientation—can occur when attention is lacking. This is such common knowledge that it hardly requires special discussion.
It is also generally known that such occasional errors tend to occur more frequently with

the index, middle, and ring fingers than with the thumb or little finger, and that when such mistakes are pointed out, there is usually immediate recognition and prompt correction.

I have examined a large number of healthy individuals in this regard and would like to counter any possible objections—based on such everyday experiences—by affirming that the aforementioned occasional errors do not exceed a certain physiologically normal range.

With appropriate focus of attention, these errors generally do not occur, and can be easily reduced or completely avoided through practice.

A comparison between normal behavior and the abnormal behavior observed in the phenomenon of finger agnosia reveals that significant quantitative and qualitative differences exist between the two — differences of the kind generally seen between physiological and pathological phenomena.

In the case of the finger agnosia symptom — as evidenced by the case histories — we are dealing with persistent errors, affecting both the patient's own fingers and those of others, which may involve all fingers equally or some more than others, and which often cannot be overcome even with intense directed attention, and may in fact become worse with practice.

Thus, finger agnosia represents a pathological alteration and imbalance of a functional mechanism, which — although already somewhat unstable due to its complexity — is normally well regulated.

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If I now summarize the essential points of my discussion, the following picture emerges:

In four cases — three of which are my own and one from the observations of Pötzl and Hermann — a peculiar symptom could be identified, manifesting as an isolated disturbance in the recognition, naming, selection, and pointing out of the individual fingers on both hands.

For this symptom, I previously chose the term "finger agnosia" based on its main characteristic.

This disturbance occurred independently of the patients' other psychological behavior, and was accompanied by a lack of awareness of the deficit (anosognosia), as well as by an unnoticed impairment in recognizing left-right orientation on the body, particularly of the hands and fingers.

Coinciding with the primary disturbance in the recognition and orientation of the patient's own fingers, there was also a corresponding impairment in the recognition,

differentiation, and naming of the fingers of other people.

In my cases, right-left disorientation on paired parts of the body was associated with uncertainty in maintaining directionality during crossed pointing or grasping movements toward paired body parts of others, and not infrequently toward objects in external space as well.

In cases where a noticeable impairment of individual finger movements occurred — specifically, where the selection and separate use of individual fingers in performing specific tasks, as well as the processing of right-left orientation in praxis, no longer occurred with proper ease — I am of the opinion that these must be considered secondary consequences of the described impairment in the cognitive act itself.

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This symptom of finger agnosia, which I first described more than two years ago, must — as is already evident from my initial as well as subsequent observations — be regarded as an independent disturbance.

In the absence of any significant aphasic, apraxic, or agnostic features to which the symptom might otherwise be attributed, finger agnosia presented in all four of my cases as a cardinal component of the objective clinical picture. It was consistently associated with the predominantly manifest symptom of isolated or pure agraphia, forming a distinct cerebral syndrome that has, in this particular form, not previously been described.

Other focal neurological signs were also present. Among these, a severe and apparently isolated impairment of calculation ability — the so-called acalculia, as termed by Henschen — stood out as the most pronounced disturbance.

It was present in every one of the cases I have observed, and was also found in the case reported by Pötzl and Hermann.

In a more recently reported case (from the Prague psychiatric clinic) by Hermann and Kerschner, involving a cystic occipital brain tumor, acalculia even emerged as the main symptom, alongside finger agnosia.

Whether impairment of arithmetic ability is an obligatory accompanying feature of the syndrome of finger agnosia and isolated agraphia must remain an open question, pending further observations.

In the case described by Pötzl and Hermann, which was distinguished by autopsy findings, the symptom of finger agnosia already acquired localizing significance. Similarly, in the surgically treated case reported by Hermann and Kerschner, the focal diagnosis was confirmed.

It was shown — especially in the former case — that this symptom could be attributed to a lesion in the transitional zone between the angular gyrus and the second occipital gyrus.
Given the close similarity in symptomatology between my cases and that of Pötzl and Hermann, it seems reasonable to assume that in my cases as well, the cerebral lesion was located in an analogous region.

As far as I am able to judge based on the behavior of my cases, the symptom discussed should be understood as a primary agnostic disturbance of a selective nature.
It presents itself as a localized disorder affecting the fingers, and constitutes a circumscribed impairment within the ability to recognize and orient oneself with respect to both one's own and others' bodies.
By applying the concept of the body schema to explain finger agnosia, a deeper insight into the nature of this phenomenon can be gained.
It is self-evident that from this recognition disorder, a certain restriction of individual finger movements, a kind of constraint in their use, may result — thereby giving the symptom of finger agnosia a partially innervatory-apraxic character.
Does there also exist a primarily innervatory-apraxic form of this symptom?
This question must be answered in the affirmative. Just as the body schema itself can undergo changes originating from either the sensory or the motor domain, so too, in addition to the agnostic type, a praxis-related (apraxic) type of finger agnosia may occur. Indeed, Pötzl and Hermann have described the finger agnosia in their tumor case as an isolated innervatory-apraxic disturbance.
Whether we are dealing here with distinct manifestations, or — as Pötzl and Hermann argue — with variants of the same underlying disorder, depending on the gradual extent of the focal lesion, is a question that must be left to be clarified by further clinical experience.

A series of investigations has shown me that it is primarily — or perhaps even exclusively — the peculiar nature of isolated or pure agraphia that tends to be associated with finger agnosia.
At least, I have never yet observed the combination of deficits in recognizing, naming, pointing to, and selecting individual fingers in cases of writing disorders of a different kind, such as those typically occurring in association with aphasia, apraxia, word blindness, etc.

However, this is not to suggest that finger agnosia must necessarily be expected in every case of true agraphia, even though such a possibility cannot be ruled out in advance. What causal mechanism might lead to the picture of isolated or pure agraphia being linked with that of finger agnosia, or vice versa, must for now remain unclear.
Yet, based on current observations, it may be generally assumed that both disturbances are due to a circumscribed lesion of a common functional apparatus located in the region of the parieto-occipital cortical convexity.