THE PIONEERING RESEARCH OF JUSTO GONZALO (1910–1986)
ON BRAIN DYNAMICS

Isabel Gonzalo Fonrodona, Departamento de Óptica, Facultad de Ciencias Físicas,
Universidad Complutense de Madrid, 28040-Madrid, Spain. igonzalo@fis.ucm.es
June 18, 2015

Abstract

This work is the English translation of an extract of the pioneering research on brain dynamics by Justo Gonzalo (1910–1986). This is an article published in Spanish in the scientific journal ‘Trabajos del Instituto Cajal de Investigaciones Biológicas’, Vol. XLIV, pp. 95-157 (1952), and entitled ‘Human brain functions according to new data and physiological bases. An introduction to the studies of brain dynamics’ in its English version. As his author pointed out there, the article is a brief summary of the extensive preceding work ‘DINÁMICA CEREBRAL’, Volume I (1945) Volume II (1950), but also includes original work performed later, as the cortical gradients model. From the study of patients with unilateral lesion in the parieto-occipital cortex, J. Gonzalo characterized what he called the central syndrome of the cortex, a multisensory, bilateral and symmetric affection presenting dynamic phenomena dependent on the intensity of the stimulus, such as the separation of sensory qualities united in normal perception, noticeable facilitation by motor and cross-modal effects (that he named “refuerzo” = reinforcement), and tilted or inverted perception, among other disorders. He interpreted these phenomena under a dynamic physiological concept, and from a model based on functional gradients through the cortex and scaling laws of dynamical systems, thus highlighting the functional unity of the cortex and offering a dynamic solution to the traditional cerebral localizations. In the 2000’s decade, phenomena (related to multisensory integration and inverted vision) have been reported and models have been proposed, similar to, or in close relation with this research, which then continues to constitute nowadays a highly active research area.

A preface introduces some aspects of the translated article, its author and his research. The English translation of the indexes of volumes I and II of the book ‘DINÁMICA CEREBRAL’, to which frequent reference is made in the article, are included after the article.

Key words: Brain dynamics, Neurophysiology, Multisensory, Cross-modal, Facilitation, Inverted vision, Tilted vision, Inverted perception, Visual perception, Tactile perception, Brain injury, Central and paracentral syndromes, Cortical lesions, Parieto-occipital cortex, Sensory field, Cortical gradients
PREFACE


In spite that several works on the research of Justo Gonzalo have been published in English in the last years (see, for example, [5] [6] [7]) it is of particular interest the English translation of his original works.

The small portion here translated is the article published by Justo Gonzalo in 1952 in the scientific journal ‘Trabajos del Instituto Cajal de Investigaciones Biológicas’ [4]. As his author expressed there, it is a brief summary of the extensive preceding works (Vol. I and II [1, 2, 3]) to which he makes frequent reference, and also includes original work performed later, as the proposed model of the cortical gradients. I acknowledge the permission of the publisher [‘Consejo Superior de Investigaciones Científicas’ (CSIC)] of that journal to reproduce the article. A reduced and modified version of the present translation was distributed in the Workshop on Alpha Processes in the Brain, in 1994 in Lübeck (Germany). In order to inform about the contents of Vol. I and II, the English translation of their respective detailed indexes are also include at the end of this work.

Justo Gonzalo Rodríguez-Leal (Barcelona 1910 – Madrid 1986) was a Spanish neuroscientist. After finishing his degree in medicine, he specialized in neurology at the University of Vienna (1933-34) with H. Hoff and O. Pötzl, and in Frankfurt (1934-35) with K. Kleist. During the Spanish Civil War (1936-39), he studied a large number of brain injured patients and then described what he called brain dynamic phenomena. From 1941 he was a full-time researcher exploring patients with brain lesions and holding PhD courses on Physiopathology of the Brain at the University of Madrid. He belonged to the ‘Instituto Cajal’ and to the Official Research Council, also in Madrid. A list of his published works, works on his research and references to his works, as well as other data on his biography and research are in: http://en.wikipedia.org/wiki/Justo%20Gonzalo

From the study of patients with unilateral lesion in the parieto-occipital cortex, he characterized what he called the central syndrome of the cortex. This syndrome, with multisensory, bilateral and symmetric affection, presented dynamic phenomena dependent on the stimulus intensity, such as the separation of sensory qualities (united in the normal perception), noticeable facilitation by motor and cross-modal effects (that he named “refuerzo” = reinforcement), and tilted or inverted perception, among other disorders. The pathological sensory stages in perception can be synchronized by reinforcement, whose first detailed study was carried out in this research. In this syndrome a strong muscular contraction, for example, can improve the perception in any sensory system. Particularly, the dynamic phenomena manifested themselves in the inverted perception process, whose first profound experimental study and interpretation belongs also to this research. The author interpreted these phenomena under a dynamic physiological concept, and from a model based on functional gradients through the cortex and scaling laws of dynamical systems, thus highlighting the functional unity of the cortex and offering a dynamic
solution to the traditional cerebral localizations. He developed the concepts of similarity and allometry based on the biological principles of development and growth, applying them to the brain dynamics, and extended the model to the auditory system and language. This extension is summarized and published for the first time in ‘Suplemento II’ of the recent edition of the book [1].

The details about data and methods that firmly support what is established in this article, are extensively presented in the mentioned book, in which Vol. I [1 2] covers general findings and the systematic study of visual functions, while Vol. II [1 3] is devoted to tactile functions and to expand on the principles introduced in Vol. I.

The first edition of the book [2 3] had a significative reception by the international neurologist community at that time. To our best knowledge, this is the first time that the term ‘brain dynamics’ (‘dinánica cerebral’) was used to describe brain mechanisms in relation to sensory organization. More recently, this research awaked special interest in the field of Artificial Intelligence (see e.g., [8]). In the 2000’ s decade, some phenomena have been reported which are similar to those that J. Gonzalo had described, related to the tilted or inverted vision, multisensory interactions, cross-modal and the multisensory integration. Also, modelling of the cortex has been proposed that are closely related to J. Gonzalo’s model (see [5, 6, 7] and references therein). The phenomena and model described by J. Gonzalo are then closely related to highly active research nowadays.

As is usual in texts written many decades ago, some aspects of the original title, contents, and the way of expressing it, reflects the context of that time. The original work whose translation we present here does not contain references, the author making always reference to Vol. I and II in which the author refers to 350 works of international specialized literature. These references were included in the facsimil edition [1] with the indication, for each reference, of the pages of Vol I and II where it is cited. As an sample, let us mention a few of those references studied by the author on experimentation with animals by Flourens [9], the isochronisme of Lapicque [10], physiological activity of the cerebral cortex by Pavlov [11], the theory of Jackson [12] on the unity and functional levels of the brain, the diaschisis phenomena of Monakow [13], the mass action of Lashley [14], precedents on visual agnosia [15]-[23], in particular the much-discussed Schn. case of Goldstein and Gelb [17], other works on brain injuries and brain pathology [24, 25, 26], nerve crossings [27], cerebral organization [28], apraxia [29], pathology of the sensitivity [30] and of sensory perceptions [31, 32], body perception [33], aphasia [34], gestalt theory and psychophysical isomorphism [35, 36], etc.

References


ENGLISH TRANSLATION OF THE ARTICLE

LAS FUNCIONES CEREBRALES HUMANAS SEGÚN NUEVOS DATOS Y BASES FISIOLÓGICAS

Una introducción a los estudios de Dinámica Cerebral

Autor: Justo Gonzalo

Published in Spanish in:
Trabajos del Instituto Cajal de Investigaciones Biológicas, XLIV (1952) 95–157 [1]
and in:
DINÁMICA CEREBRAL, Suplemento I. Universidad de Santiago de Compostela, Red Temática en Tecnologías de Computación Artificial/Natural, 2010 [1]
Open Access from [http://hdl.handle.net/10347/4341](http://hdl.handle.net/10347/4341)

English translation by Jamie Benyei and Isabel Gonzalo Fonrodona
HUMAN BRAIN FUNCTIONS ACCORDING TO NEW DATA AND PHYSIOLOGICAL BASES

An introduction to the studies of brain dynamics

Author: JUSTO GONZALO (1910 - 1986)

INDEX

Introduction ................................................................. 9
I. Phenomena of dynamic action .......................................... 10
II. Magnitude and position. The problem of localizations .................. 11
III. Optic functions. The problem of sensory organization .............. 14
IV. Spiral development ..................................................... 18
V. Tactile functions ....................................................... 25
VI. Dimensions of the sensory field ..................................... 28
VII. Cerebral gradients ..................................................... 29
VIII. Brain lesions .......................................................... 37
IX. Dynamic reduction and new cases of the reversal of vision process .... 38
X. Tactile bilateral involvement in unilateral lesions ..................... 41
XI. Concluding remarks .................................................... 43
I have judged that man’s knowledge of nature must be taken from the principles of geometry and mechanics, for all other notions we have of sensitive matters, being confused and obscure, cannot provide us with knowledge of anything.

**Descartes**

Until a physical-chemical explanation is found for a biological phenomena, it will be commonly incomprehensible. If the veil is ever lifted, we shall be surprised at not having guessed what was hidden from the outset.

**J. Loeb**

If one can measure what one speaks about and express it as a number, something is known about it. If not, the knowledge is a poor and quite unsatisfactory thing, whatever it is that one is dealing with.

**Lord Kelvin**

**Introduction**

These quotes are doubtlessly a difficult demand to be placed on cerebral pathology, and may seem to some to be utopian. Nevertheless, they refer to the general nature of our work and avoid many explanations from the outset. This orientation is not at all a criticism of more conventional paths, but rather points towards a subsequent stage which may be reached more easily.

The study many years ago of a multitude of cases of brain injury has given rise to new data which help to determine human cerebral functions according to much more physiological principles than those established to date. The present report is restricted to highlighting the most demonstrative aspects of the research, and refers the reader to the original publications for more details. Firstly, I shall present a summary of my work of brain dynamics (1941-45-49)\footnote{2, 3}, which will be followed by indications of complementary studies carried out between 1950 and 1952.

The progress may come from the use of other methods, and also from an unbiased examination of simple observed facts. Both approaches have a place in our study. Many matters have been advanced here thanks to a more physiological and experimental analysis. The natural advantages of quantitative procedures providing observations with precision may be worth objective control in sensory phenomena and provide quantitative laws of the process.

The two published volumes of *Dinámica Cerebral*\footnote{2, 3} study two brain war injuries, cases M and T, with a high degree of topographic similarity in their left parieto-occipital cortex lesion, although the magnitude of the lesion was greater in M. The studies cover an observation period between 1938 and 1948. The innovative nature of this research appeared in 1939 with the discovery of dynamic action phenomena. In 1941, a memo on the most important experiences
and arguments covering the whole work was released. The first volume was published in 1945 [2]. It covers general findings and the systematic study of visual functions. The second volume, published in 1950 [3], covers tactile functions and expands on the principles introduced in volume I.

I. Phenomena of dynamic action

This type of phenomena is treated in the first place due to its general nature, because it clarified different agnosic disorders in prototype cases M and T, and above all, because it indicated a physiological direction from the outset of this research in 1939. The dynamic action phenomena (desynchronization or dephasing, reinforcement and repercussion) consist of transformations of central nervous excitability.

Desynchronization or dephasing involves the excision of sensory phenomena which are normally united and non-dissociated, in such a way that pathological sensorial stages appear and the normal process of all or nothing is broken down into phases or partial reactions. This sensory desynchronization, originated by a nervous asynchronism, permits an analysis of the sensory complex by penetrating its structure: A very weak tactile stimulus is only perceived as a mere sensation of contact lacking the possibility of spatial localization, whereas localization is normal when the stimulus is stronger. Small, distant or weakly illuminated objects appear to be turned or even inverted although when stimulation is much greater (large visual angle, more intense illumination etc.), the object is perceived as being upright, and so on with other examples. The abnormal stage is then understood to be recruited or surpassed by the intensification of stimulus. The amplitude of such a pathological stage is dependent on the magnitude of the cortical lesion, then being quite pronounced in the case of M. It must be pointed out that the most striking abnormal phenomena (inversion) only occur under minimal stimulus and are absent after a certain intensity of stimulus. Subjects may thus behave normally in everyday life and even be unaware of a great part of their anomalies.

The reinforcement or multisensory facilitation phenomenon is very significant in excitability disorders. It corresponds neurophysiologically to the “spatial or simultaneous summation”. It was found that intense muscular contraction in the subject was able to replace the loss of elementary excitability and thus reduce the desynchronization or dephasing. When the objects appear to be greatly turned, this reinforcement straightens them instantly and simultaneously clears the vision, dilates the visual field and is similarly shown in touch or hearing. A distinction must thus be made between the “inactive” state of the subject and the state of “reinforcement” which is much more favourable. Regardless of the intensity of the reinforcement, however, it is unable to annul the entire deficit. Together with this muscular effect there are other types of spatial summation such as the “bi effect”, i.e. the effect of binocular summation in which one eye reinforces the other, i.e., all visual functions being better when both eyes are used instead of one. The same occurs with the other senses: the combination of muscular effort and the bi effect results in increased action.

Experiences of inter-sensory or multisensory summation are also described: A certain class of tactile stimulation, for example, may assist or increase the visual function. This reinforcement has nil effect in a normal subject, and while it is quite clear in case M, it is scarcely notable in T.
As well as abnormal sensitivity to spatial summation, there is also sensitivity to “temporal or successive summation”, i.e. to the accumulation of successive stimuli, a phenomenon which is also absent in a normal subject. It is highly significant in case M and only quite clear in case T.

Finally, the phenomenon of cerebral repercussion of the lesion is the most contrasted concept to the traditional ideas of specific anatomic localizations. In M and T, the cortical brain lesion is located in a “central zone” which is equidistant from the visual, tactile and auditory zones their projection areas being not directly implicated although it can be demonstrated that all these sensory systems are clearly affected as their activity is reduced in all types of function from simple excitability to the most complex ones. Furthermore, both sides of the body are affected almost equally in spite of it being a unilateral lesion. This form of repercussion gives rise to the new “central syndrome” of the cortex.

These phenomena of dynamic action may be said to initiate the transition from the traditional or static cerebral concept (clinical empiricism) to a dynamic one (physiological causal analysis), proven not only in cases M and T, until now models of this research of brain dynamics, but also in many others.

II. Magnitude and position (the problem of localizations)

The examination of cases M, T and many others (1941) established that the effect of cortical lesions may depend on just two factors: the magnitude and the position of the lesion. Position conditions the type of disorder distribution in the cerebral system, i.e., the topography of the above mentioned “repercussion”. The magnitude or extension of cortical destruction determines the intensity of the disorder.

Position

Three general cortical syndromes must be distinguished with respect to position: central, paracentral and marginal (Fig.1).

The central syndrome is the new syndrome mentioned in the description of repercussion, and is characteristic of this research of brain dynamics: The site of the lesion is geometrically “central” or equidistant from the visual, tactile and auditory projection areas. These sensory systems are equally affected on both sides and in all their aspects. It is thus a homogeneous repercussion in the whole sensory brain in both hemispheres. The sensory affection thus has maximum “symmetry”. For example, concerning the visual system, a “symmetric concentric reduction” of the visual field is found.

The paracentral syndrome is somewhat similar to the central syndrome. It is a form of transition as its name indicates, and the repercussion or distribution of the disorder is “asymmetric”. The lesion is in an intermediate site between the “central zone” and the projection area, or closer to the latter. There are thus three paracentral syndromes: visual, tactile and auditory. Functional involvement may be somewhat general while naturally predominating over the nearest projection with the contralateral side predominating more than the homolateral side of the lesion. The type of alteration in the visual field is “asymmetric concentric reduction”, corresponding to a hemianopsia, with varying degrees of macula conservation and homolateral
Figure 1: Scheme of positions of cortical lesions (x) and corresponding types of visual field. a: symmetric concentric reduction in the central syndrome; b: asymmetric concentric reduction in the paracentral visual syndrome; c: hemianopsia in the projection path syndrome (marginal or peripheral disorder). Lesion magnitude is not considered, although the more central the syndrome, the greater the lesion must be to provoke a deficit in the visual field.

Figure 2: The pathological forms of each visual field (above) and the respective visual profile intensity. In symmetric concentric reduction (central syndrome), there is a depression in the sensitivity profile with the form more or less maintained. In paracentral syndrome, the depression is less homogeneous or asymmetrical. In the projection path syndrome, there is a visual suppression or interruption for one half, according to the anatomical disposition of the cortical projection.
constriction: the two halves of the field undergo restriction by the identical mechanism but much more so on the contralateral side. In touch and hearing, a quite small deficit may be shown contralaterally, while it is practically null homolaterally.

The *marginal syndrome* lacks repercussion. The lesion is in the projection area (visual, tactile or auditory) with the affection completely restricted to the contralateral half of only one sensory system. In the visual field, the defect is a simple hemianopsia lacking other components.

Thus, when the lesion moves from the “central” zone of the cortex to the “marginal” zone (Fig.1), the repercussion is reduced and the general disorder (bilateral and extended to all systems) becomes restricted to only one system on its contralateral half. The central syndrome is something totally new. The marginal syndrome traditionally refers to the projection areas. The paracentral syndrome is also known but not correctly interpreted and now, considered involved in the asymmetrical repercussion effect, it is presented as justly interpreted. Furthermore, it is worthwhile noting that while the marginal syndrome is more a syndrome of projection paths, the other two syndromes correspond to the activity of the centres. In a more physiological consideration, the marginal syndrome is a functional *suppression* or interruption while the others should be regarded as syndromes of functional *depression* or descent (Fig.2) which present the above mentioned phenomena of dynamic action.

**Magnitude**

The intensity of affectation, i.e. the degree of functional descent or depression depends on the quantity of cortex destroyed. The best examples of this in the whole work are cases M and T, which, as with their central syndrome, are distinguished by the intensity of the symptoms in close concordance with the different magnitude of their respective cortical lesions. The concept of magnitude is characteristic of brain dynamics given its extreme *quantitative* nature, radically opposed to the traditional, somewhat *qualitative* doctrines. The parallel between the magnitude of the lesion and the degree of affection leads to the establishment of continuous transitions in the various abnormal phenomena and the exclusion of independent or genuine qualitative defects. This is a problem which was raised long ago in cerebral pathology although generally avoided by most authors. Thus, the difference between paralysis and paresis, anesthesia and hypoesthesia, hemianopsia and hemiambliopia, etc., may only lie in different degrees of involvement of the same function.

The simplification of the thorny problem of localizations with the two factors here analyzed (magnitude and position) gives place to a dynamic solution to localizations. The repercussion phenomenon, particularly in the central syndrome, may seem to put an end to the traditional doctrine of the specific centres. It would lead to the postulation of a dynamic unit of the whole cerebral system whose degree of involvement only depends on the magnitude of the lesion. The position factor, on the other hand, determines diverse paracentral syndromes and firmly supports the heterogeneity of the cerebral system. It may thus be concluded that *instead of specific centres there are dynamic effects on the cerebral system according to the magnitude and position of the lesion*. This concept was the “first principle” of this research on brain dynamics.
III. Visual functions. The problem of sensory organization

A further fundamental problem is that of functional organization which, while related to the problem of localizations, has its own character. In the present research we shall only analyze the facet of sensory functions which offer a wealth of manifestations difficult to match in motor functions. Given the central syndrome in M and T, a structural analysis is possible in any of the three primary sensory systems—vision, touch and hearing. Apart from their individual peculiarities, they show a common organizational plan. Furthermore, a great number of manifestations may be united under a general principle which is dealt with in a further section.

The structure of the functions are analyzed here under the activity of the sensory field suffering a functional “depression” in our subjects, derived from a deficit in cerebral excitability. The most elementary excitability is reduced and there is an abnormal sensitivity to spatial and temporal summation. The normal sensorial steps of differential sensitivity are greatly dilated and further steps arise where they are normally absent, as set out in the paragraph on desynchronization in section I.

Within each system, the excitability disorder covers all types of activity and increases with rises in physiological demand. This gives rise to the typical exclusion of more complex activities according to a well defined physiological order, constituting the so-called “dynamic reduction”.

In synthesis, the depression or reduction of the sensory field does not imply a mere smaller field but a field of residual function in which every activity is touched (affected), whereas the general scheme of organization is conserved, while functioning on a different numerical scale from the normal. In fact, the magnitude of cortical lesion brings on a change in the magnitude of the sensory scale, i.e., the “sensory dimensions” become different.

The monographic study of visual functions on the basis of cases M and T occupies a large part of the precedent book “Dinámica Cerebral” [2, 3]. More attention is placed on the case M due to its more intense disorders, and using the case T as a complement. Many phenomena are new or almost unknown and there is an effort to provide a physiological and quantitative basis of them even in the details. An study was made [“Dinámica Cerebral” Vol. I (1945) [2], pp 96-392] on the following issues.


iii) Visual forms: Concentric reduction of the visual field, relationships of excitability in the visual field, organization of the visual field (monocular polyopia, pseudofovea, etc.). Flat colours or chromatic irradiation (distorted chromatic contour perception). Visual acuity. Vision of movement. Vision of figures and objects (metamorphopsia, etc.)

iv) Perceived direction of the visual image: Various cases of perceived inverted vision, characteristics of this disorder, fundamental experience, synchronization by reinforcement (variation according to the state of the central nervous system), perceived direction of the image accord-
Table 1: Visual functions according to three types of physiological level (M inactive, M under reinforcement, T inactive). R. E.: right eye; L. E.: left eye; Chr. cap.: Chronaxia capacity; “mono”: monocular vision; “bi”: binocular vision.

<table>
<thead>
<tr>
<th></th>
<th>Case M inactive</th>
<th>Case M reinforced</th>
<th>Case T inactive</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Chr. cap. 3.5 µF</td>
<td>Chr. cap. 2.7 µF</td>
<td>Chr. cap. 1.4 µF</td>
</tr>
<tr>
<td><strong>Colours</strong></td>
<td>Medium light: very weak yellow-blue, etc.</td>
<td>Medium light: colour vision practically normal.</td>
<td>Very low light: Tri-tanomaly (weakness in blue).</td>
</tr>
<tr>
<td></td>
<td>Intense chromatopsia.</td>
<td></td>
<td>Low and fleeting chromatopsia.</td>
</tr>
<tr>
<td></td>
<td>High disorder of chromatic induction.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Forms</strong></td>
<td>Visual field reaching 6°.</td>
<td>Visual field reaching 40°.</td>
<td>Visual field reaching 50° or more.</td>
</tr>
<tr>
<td></td>
<td>“bi”, 1/10.</td>
<td>Irradiation only in red (with medium light).</td>
<td>Weak irradiation in red only.</td>
</tr>
<tr>
<td></td>
<td>Severe decrease of the movement perceived.</td>
<td>Coherent and faster perception than inactive state.</td>
<td>Unstable forms; somewhat slow perception in very low light.</td>
</tr>
<tr>
<td></td>
<td>Very unstable &amp; diffuse form, very slow &amp; successive perception.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Perceived Direction</strong></td>
<td>R. E.: max. rotated vision 145°.</td>
<td>Starting from inactive state and the same stimulus: strong reinversion from 145° to 30° or 20°.</td>
<td>R. E.: max. rotated vision 25°.</td>
</tr>
<tr>
<td><strong>Schemes</strong></td>
<td>“Bi” &amp; strong light: Illusions, disgregation, concretist behaviour.</td>
<td>“Bi” &amp; strong light: Generally good, with serious defects in complex tests.</td>
<td>Failure only in complex texts. Some indications of weakness for all of them.</td>
</tr>
<tr>
<td></td>
<td>In low light, again disorders of the inactive state.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
ing to receptor (variation by receptor), various complementary experiences, mechanism in the perceived direction of the visual image.

v) **Gnosis or schemes:** Agnosic defect, visual behaviour. Structure of visual agnosia. Alteration of orthogonal orientation. Halocentric orientation disorder.

Table 1 is intended to be a rapid orientation in the study of cases M and T, their disorders and the effect of reinforcement on M. In general terms, chronaxia and visual rheobase are ten times greater than normal in M and four times in T. The phenomena of summation are highly apparent in the former and much less so in the latter. M’s visual field is considerably reduced but recoverable or recruitable through intense stimulation and reinforcement whereas reduction is moderate in T. Similar occurrences are found in the reduction of visual acuity, in the abnormal chromatic irradiation due to weakness of specific spatial localization, in the colour alteration where blue-violet suffers more as a result of being the slowest or that of greatest chronaxia, in the instability of forms or figures and in the visual perception of movement.

The directional disorder in case M results in a turn of the perceived direction of objects that is almost an inversion of direction. Exact measurements seem to indicate a turn of up to $150^\circ$, while only reaching $20^\circ$ in case T. Visual agnosia phenomena are extreme in case M and moderate in T. The former was able to interpret very few drawings of objects and failed completely in mixed or superimposed drawings, while the latter only had difficulties with the second problem.

The following brief remarks refer to the five sections of the table:

1. There is a highly objective basis for the experiences in **general excitability.** The electrical stimulation of the retina and the pathological summation curves (Figs. 3 and 4) are highly accurate. Similar results were obtained on luminous stimulation, with slower and narrower luminous adaptation than in normal cases. Flicker fusion threshold was measured.

2. The study of **colours** is somewhat difficult in these subjects due to their chromatic sensorial defects and also to their perceptive, spatial and agnosic defects. In the first aspect, chromatic weakness occurs in the whole spectrum but is stronger in the yellow-blue pair, particularly in the latter colour (tritanopia-tritanomaly). The corresponding singular interval or step is greatly increased and complicated in some colours. The sensitivity for the chromatic discrimination is highly diminished. White has a green tinge (chromatopsia) due to a deficit of chromatic ingredients etc., with each phenomenon fitting the known rules of chromatic blindness.

3. Defects in the **visual field, acuity** etc. (Fig.5) become highly useful numerical data for brain dynamics. We focus on concentric reduction as it is generally an obscure and overlooked matter. Here it is defended in its organic nature in accordance with the central syndrome. **Chromatic irradiation** or “flat colours” described by Gelb, is studied physiologically according to the relationships of excitability. The peculiarity of the disorders in visual perception of movement lies in the phenomena of apparent acceleration and reduction of distance covered when the stimulus is weak. It is a spatial and temporal contraction. In addition, there is an inversion of mobile direction. It is the starting point for fundamental matters.

4. **Directional disorder** in relation to inverted-upright vision has weak but numerous precedents, while the first objective observation of such an unusual phenomenon and the profound experimental study places it entirely within the scope of this research of brain dynamics. It is treated separately for many reasons (see next section **Spiral development**).
Figure 3: Intensity-duration curves (volts-microfarad.) obtained by retina stimulation, with condenser discharge (cathode on eyelid) to obtain minimum phosphene. Vertical arrows indicate chronaxia capacity. Note comparative values to normal in cases M and T, the change of the curve in state of reinforcement (facilitation) in M and the smallness of the reinforcement effect in T.

Figure 4: Iterative aptitude in retina in case M, which is null in normal man. Curves for inactive and reinforced (facilitated) states (volts versus number of stimuli) by stimulation as in Fig. 3, maintaining the interval $\epsilon$ at 1/12 second and duration of stimulation or condenser capacity (1.5 $\mu$F) fixed. The intensity of summation is the economy of the voltage, in %, between one stimulus and an undefined number.
Electric excitability of the retina

Visual Field

Inactive

Reinf.

Normal

Acuity in the visual field

Figure 5: Case M: correspondence between curves of excitability, visual field amplitude and acuity in the visual field, for inactive and reinforced states. Acuity in central vision descends to 1/20 in the inactive case and 1/10 in the reinforced state.

5. **Gnosis disorder**, covered here according to the concept of the scheme, is caused by a disgregation (or disintegration) and diffuse conception of objects and their schematic representations. More importantly, while it is the defect of a peculiar stage in sensory organization, it is by no means admitted as an independent phenomenon; contrary to conventional doctrine, it conforms with the remaining sensorial defects of the system as set out in table 1. The most outstanding contribution of the new phenomena to this field is probably the discovery of the **orthogonal disorder**, a singular defect of spatial orientation in which objects and figures are recognized independently of their orientation, i.e., inverted or upright portraits seem the same; written texts in particular can be read both upside down and upright without the subject noting any difference. The modifying effect of summations (bi effect, muscular reinforcement) on this disorder is very strong. For several months after being injured, case T showed clear signs of this disorder. Afterwards it disappeared completely with the involution of the cerebral disorder, and only occurred fleetingly in a rudimentary form as a consequence of a strong epileptic shock. Another original contribution is the description of the substitution of **hallocentric** spatial orientation in M by mere **egocentric** orientation (see the book “Dinámica Cerebral” [2, 1]).

IV. Spiral development

This final part on the visual functions refers to the phenomenon of inverted vision. Through this phenomenon it can be amply demonstrated the experimental and quantitative analysis which characterizes this research of cerebral dynamics. The spatial inversion process is connected to other sensory manifestations already analyzed (visual field, colours, acuity, movement, etc.) It is a functional complex which develops in accordance with the inversion-reinversion process, highlighting the intimate structure of the sensory field in spiral development and in accordance with physiological relationships of cerebral excitability and anatomic textures of the cortex. [See Vol I (1945) and II (1950) of the book “Dinámica Cerebral” [2, 3, 1].

Inverted vision was discovered by accident in case M (1938), although on this subject it was
Figure 6: Inclination and fading of the configuration, seen by patient M, according to size of letters and states of summation. Left column is the model to look at. The model is situated at a convenient and fixed distance, so that the patient M, with the right eye and in inactive state obtains completely upright vision only for the tallest letter (N). Then, the smaller letters seem tilted and are illegible beyond a turn of 40° (fourth column). For the states of summation: right eye and reinforcement (third column) and binocular and reinforcement (second column), an analogous effect is obtained, although with the corresponding “scale shift” because of greater sensitivity.
not complete. While its exact extent was difficult to determine, there seemed to be a turn of 160°, a highly notable disorder in accordance with his other characteristics. In T there was only a slight inclination of some 20°. Other cases of brain injuries with small turns were observed by the author prior to M. The phenomenon in M consisted in seeing objects greatly inclined and even inverted, turning on the frontal plane, with the turn found to be dependent on the size and distance of the objects, i.e. on the subtended angle of vision. There was also a dependence on the intensity of illumination and exposure time, as a nearby object appeared to be greatly turned if it was only seen for an instant. Case T was included in the same process and interpreted as an attenuating manifestation or frustrated inversion. It is not merely an exchange between above and below, but essentially a gradual turn according to the energy of stimulation and in any direction (vertical, horizontal). In fact, the phenomenon was discovered in objects with horizontal movement which were perceived as moving in the opposite direction.

In case M, the directional disorder does not disturb everyday life and, perhaps paradoxically, almost goes unnoticed as no turn is perceived in clearly distinguished objects. A notable turn of 50° corresponds to hazy vision which impedes the recognition of the object, and beyond 90° and nearing inversion, the object is reduced to a shadow that is ignored, so that great turns are practically excluded. Normally, an upright image with small inclinations which do not cause disturbance is perceived. Thus, large inclinations and inversion should be provoked, but without having to using any special instruments. It is sufficient for the subject to concentrate on peripheral vision in an elongated object moving away or from one side to another or from above to below etc. Such simple tests lead to the discovery of the previously mentioned factors of stimulation.

In order to give an idea of the questions arising from spatial inversion, in this section we analyze 1) the phenomenological aspect, 2) the quantitative aspect, 3) spiral development of the field, 4) cerebral mechanisms and 5) nervous crossings.

1) Several examples serve to give a phenomenological idea of the process. Once beyond a perceived turn of 40°, letters appear to be unrecognizably blurred (Fig. 6). A more pronounced turn may be studied in more elongated objects. A vertical fork moving away from the subject,
may be taken for a spoon when turned subjectively $30^\circ$. At $90^\circ$ it looks like an smaller, elongated diffuse object; at $140^\circ$, it is on the verge of becoming a shapeless spot which is hardly distinguished from the background. The experimental conditions were: M in an inactive state at 1.5 m from the object, using one eye and under medium lighting (Fig. 7). A similar perception occurs for human figures but at 5 m and with low illumination. If a moving vehicle is seen in an inverted direction of movement, it appears as a mere blurred spot moving over a much smaller trajectory and at an overestimated speed. Thus, together with the change of direction, there is a parallel alteration of sensorial intensity, space and time.

2) More extensive experimental study shows that the process matches certain quantitative relationships perfectly, as suggested in 1941 and developed in 1943. Desynchronization (or dephasing) causes an abnormal sensory step or interval in the visual perception of direction between pathological inversion and the normal perception, a step or interval which must be recruited or overcome by an increment in the stimulus. The basic relationship to be determined is simple (direction perceived versus stimulus intensity), although it may show a high diversity of aspects due to the involvement of many excitation factors (stimulus, receptor, nervous central state) and other experimental circumstances (se details in the book “Dinámica Cerebral”, Vol. I and II).

Here we shall focus on the most simple and principal aspects (Figs. 8–10). Fig. 8 illustrates the device used to obtain the curves shown in Fig. 9. The perceived rotation by the patient of the vertical test arrow depends on the illumination of the arrow at a given subject-arrow distance. The process of sensorial recruitment of the direction is shown in Fig. 9 in function of the intensity of illumination. It can be observed that from the upright or normal perception onwards, light must be reduced significantly to obtain a perceived inclination of $45^\circ$. At this point, however, the turn increases quickly when the illumination is reduced very little more. In the “state of reinforcement”, all occurs identically but with less light than in the inactive state. The sensorial recruitment curves for direction may also be obtained by varying the visual angle, either by distancing the object or using different sized objects at the same distance (as in a previous test with letters or optotypes). When the curves are drawn taking the logarithm of light intensity (Fig. 10), they are converted into sygmoideas that approximate straight lines. It may thus be stated that the sensorial growth of direction is proportional to the logarithm...
Figure 9: Sensory recruitment curves for the direction (orientation) perceived of the visual image in function of the light intensity, for case M in inactive and reinforced (facilitated) states. Experimental device of Fig. 8).

Figure 10: Relationship between angle of the perceived turn and logarithm of the stimulus intensity (Fechner law).
of the stimulus. This conclusion falls within the Fechner law, which is therefore still valid for pathological stages. In case T, a similar but much higher recruitment curve than in case M reinforced is found. It means that in the three levels (M inactive, M reinforced, T inactive) the law is the same but with a different constant or parameter, whose value depends on the magnitude of the lesion or on the state of the centres due to the effect of summation.

3) We are aware that the perceived turn embodies a complex process of sensory degradation. For example, when the vertical test arrow is perceived to turn, it narrows, shortens among other changes. Several measurements which may only be extremely indirect seem to indicate that this reduction in the size of the arrow, i.e., in the visual field, is quite progressive (Fig. 11). Following the perceived trajectory of one extreme of the arrow during the turn, we obtain a quite open spiral branch which expresses the actual spatial field disorder (assembling turn and constriction), constituting what we call a spiral development of the sensory field.

4) Concerning the mechanism of the disorder, the problem lies in the determination of the origin of the turn. In Vol. I of “Dinámica Cerebral”, after referring to the historical controversy over how upright vision is possible given optical inversion in the ocular globe, it is admitted that the retina factor is a spatial reality which can become independent or “isolated” in patients with brain injuries when it is not corrected by some brain mechanism, the problem being thus restricted to a mere particular question of the vision system. However, on the discovery of tactile and auditory inverted perception in M in 1946, the process was extended to all the sensory systems with a spatial character.

There had to be a structural factor responsible for the spatial inversion. This effect was soon attributed to the respective cortical projection areas with an inverted and contralateral disposition. The anatomical configuration thus acquired an unexpected protagonism in this research on brain dynamics. Within this concept, the spiral development has the following cerebral base: inversion and constriction extremes correspond to individual action of the projection (or “marginal”) area; magnification and reinversion are linked to the activity of more “central” areas (magnification due to an increase in recruited neural mass, reinversion due to some effect of brain plasticity). There is then a “cerebral growth or recruitment” where the spiral trajectory represents a series of successive balances between “marginal” (projection area) and “central” action, which may also be expressed indicating that in magnitude and direction there is a sensory-cerebral correspondence, i.e., a psychophysical isomorphism. Due to the important consequences of this formulation in the sensory organization, it is the “second principle” in this research on brain dynamics (1947): Spiral development of the sensory field due to a psychophysical isomorphism. However, this left a gap with regard to the reinversion process, which appears to be filled in this research (1950) by ascribing the recently discovered “secondary areas” (of unknown significance), to this final functional process. Such areas represent the sensory field in the reinverted position and have a bilateral action.

In 1951, the problems of spiral development were the subject of a specific study. In relation to previous findings on spatial inversion in tactile system (see “Dinámica Cerebral”, Vol. II), spiral development was specifically investigated for the case of the test object being situated in peripheral vision to one side of the visual field. As sensory degradation was produced according to the circumstances of excitation, the test object underwent turn, centripetal deviation, reduction in size and intensity and finally came to rest in a contralateral and inverted position, quite close to the point of fixing or centre of the field. It is as if on turning around the centre of the visual field, one half of the field had been progressively constricted and dulled in visual intensity (Fig.
Figure 11: Reduction of arrow size (or reduction of the field) during the turn. The extreme of the arrow thus describes a spiral. There is a spiral field development.

Figure 12: Experiences in visual spiral development in case M. The test object is placed 10° from the central point of fixation. As visual excitation is conveniently reduced, the image of the object wanes in size and intensity, turns and ends contralaterally, close to the centre. The drawn images represent phases obtained during the process. The trajectories correspond to the type of logarithmic spiral.

Figure 13: Dynamic characters of the spiral in the field development. The two vectors in which the tangent to a point on the curve is decomposed signify the two “forces” of growth causing the spiral: “dimension” for amplification of the field, “direction” to reinvert it.
12). The spiral trajectory obtained is like a field “force line” representing the *physiogenesis of the local sign*. Geometrically, it has the aspect of a logarithmic spiral and may be expressed dynamically according to the “forces” it is subjected to. It can be broken down into two vectors of dimension and direction (Fig. 13).

5) Finally, the problem of inversion-reinversion is taken to its anatomical roots and related to the celebrate Cajal theory of nerve crossings (quiasma, crossing of long paths) where three processes occur:

i. The ocular inversion caused by the presence of the crystalline makes the quiasmatic cross necessary to achieve binocular congruence in the nervous centres (Cajal).

ii. Cerebral visual projection remains inverted and crossed with respect to the exterior. According to Cajal, however, a crossing of the pathways of the other sensory and motor systems is functionally an uncrossing of the vision so that a visual stimulus may have a reflective discharge in a motor response on the same side, i.e. congruently from a spatial point of view.

iii. However, in the sensorium, the crossing and inversion of the projection or primary areas still holds. In accordance with this order of ideas on crossings, I have proposed that reinversion and bilateralization of the image occurs in the secondary areas. This fact does away with all spatial incongruency in the latter process since the cerebral centres could obtain a copy or image of the external world in the same order and direction.

The pathological turning process in our subjects will thus be produced by an asynchronism between the primary and secondary areas (see details in the book “Dinámica Cerebral”).

V. Tactile functions. Other functions

Touch and vision have a remarkable structural similarity. However, the former is less objetivable, making its analysis more difficult. The visual-tactile similarity has already generated much comment and the exposition here will thus be very brief. [See details in “Dinámica Cerebral” Vol. II (1950), pp 393–823].

1. *General tactile excitability* behaves in the same way as visual excitability with a similar quantitative deficit, due to the homogeneous repercussion of the central syndrome. *Vibratory sensitivity* presents a similar loss of high frequencies analogously as in flicker fusion threshold in vision. The study is made on electrical, mechanical and vibratory excitability (intensity versus duration curves) in inactive state, under reinforcement or facilitation and iteration. Pathological bilateral tactile cases with unilateral lesions, unexplained by other authors, are interpreted in this research.

2. *Tactile qualities*, much simpler than colours, have also a heterotactile interval or step due to desynchronization. Pressure or contact appears to be the primitive tactile activity (similar to luminosity in vision). Pain and temperature are differentiations. Clinical dissociations are relative and not absolute.

3. *Tactile space* shows many important phenomena. There is a large abnormal interval or step in cutaneous spatial localization of an stimulus between simple contact sensation and specific localization, with five phases distinguished in relation to the energy of the stimulus: I, primitive projection or contact sensation without localization; II, deviation towards the middle line of the
Figure 14: Phases of tactile localization in M in inactive state for mechanical pressure stimulus on a hand. Sensory threshold intensity originates tactile sensation lacking localization (phase I, which is not in the figure); slight raise in intensity leads to phase II or middle deviation, and to phase III or inversion phase. Phase IV is the homolateral phase corresponding to moderate stimulus. Phase V is a normal sensation of localization, requiring intense stimulus or moderate stimulus and reinforcement.

body; III, inversion; IV, proximal homolateral deviation; V, specific or normal localization (see Fig. 14). These phases are a consequence of general factors such as proximal deviation, spatial inversion and irradiation, becoming more manifest as the intensity of the stimulus decreases since the delay in the localization phase increases.

provides (according to intensity or energy): centripetal deviation, contralaterality, and irradiation.

Tactile irradiation (spatial diffusion instead of a normal puntiform sensation) is similar to chromatic irradiation. The quantitative relationships are determined by the curves of logarithmic recruitment (proximal deviation in function of stimulus intensity) and, more precisely, intensity-duration curves for each phase of localization. The higher levels, closer to normal localization, are perturbed to a much greater extent than the lower levels. The lower phases (I, II and III) are very close, making them difficult to distinguish at the start of the study.

It should be noted that in the theory of the local sign, the abnormal phenomena (deviation, irradiation, inversion) are an expression of a field with residual function in correspondence with the central nervous substratum, and there is no local sign as genuine individuality, but there are multiple gradations according to the functional state of the organization of the nervous centres.

Acuity (Weber), movement, tactile figures, undergo similar defects to the corresponding visual functions.

4. Tactile direction is closely linked to the process of localization and inversion was discovered when the third phase was identified. The study of all these phenomena is much less accessible than in vision and more difficult to objectify, leading potentially to many errors in superficial examination. The general laws are the same as in visual direction. The phenomena of tactile inversion are studied in cutaneous, articular or deep stimulation, as well as in complex processes
such as walking. Fig. 15 illustrates the phenomonic aspect of inversion in *cutaneous movement*. A mobile stimulus running down the side is perceived (with the function in the inversion phase) contralaterally and close to the middle line of the body, with a very shortened trajectory and opposed movement direction. If such a stimulus is along an arm, the inverted movement remains on the contralateral shoulder, and; if it is on the face, the third phase (or inversion) will correspond to the cranial tegmentum. In summary, a distal sector undergoing inversion is contralateral, in the opposite direction, highly constricted and thus close to the middle line of the body, verifying the conditions of a field of residual function. The turn or inversion trajectory is not for the whole field as in vision but is more regional according to three autonomous zones of turn (head and the two extremities). The quantitative results are shown in the curves of direction recruitment (turn and deviation of the line in function of pressure applied on the skin) although the same precision as in vision cannot be obtained.

In *walking*, the process shows unique characteristics: in moderate walking, the first step is ignored, the second feels inverted, the third transversal, the fourth oblique, etc. This is a progressive recruitment of direction of perceived steps due to accumulation in the centres or iterative action of steps. On the other hand, in slow walking there is no accumulation and the direction remains inverted for each step. The inverted steps feel very short and fast in conformity with the residual field. The energy of the step modify the result according to the rules of excitation.

The *mechanism* and *theory* of spatial direction are amply covered in the vision study, where they are presented as a general process for all spatial sensory systems. Tactile research has promoted the union between the constriction of the field (proximal deviation) and inversion, leading to the spiral development described above.

5. *Tactile schemes* involve several functions. There are two fundamental aspects: the corporal model or body scheme and the tactile scheme or tactile recognition. The former may be considered gradually according to the *somatic, postural* and *praxic* models, which show notable anomalies when studied in detail. Tactile recognition shows defects such as reduction and esterognosic transformation, and the defect of the tactile scheme or asymbolia in a higher phase. Recognition is indirect and the typical fragmentation or discontinuity of agnosia appears, which is only an effect of the reduction of scheme dimensions (small scheme which includes juxtaposed parts but not groups).
With respect to *Auditory functions*, their dynamic phenomena and the other manifestations appear in cases M and T in the same way as with vision and touch except for the peculiarities of each system. Auditive excitability is in deficit and is sensitive to summations (reinforcement, bi effect, iteration). There is a certain hypoacousis, perhaps stronger in high tones. Acoustic quality (musical tone) has an abnormal step, as in vision and touch. It is a singular interval between simple “sonorousness” and real “tone”. Contralateral localization or inversion of a sound stimulus only occurs in M when the intensity of the stimulus is weak and the subject is in an inactive state. The inverted perception always lacks tonal quality (residual function).

In language, diverse aphasic aspects occur depending on stimulation, this being in fact a semantic defect.

**VI. Parameters of the sensory field**

At this point, several further observations will be made on the residual field and sensory dimensions or parameters.

Firstly, the clinical aspect of the two types of cortical syndromes will be indicated. In the “marginal” syndrome, the subjects have a quite ostensible defect because the lesion of a determined sensory system is massive. There is an anatomic “suppression” of the system which prevents any type of study of the functional structure and we are reduced to outlining a “gap” in the sensory organization, on which the specific localizations are built. On the contrary, in the more “central” syndromes (central and paracentral) even in extreme magnitude, the individuals show few symptoms at first sight and sometimes are unaware of them themselves. However, examination reveals certain gradual alterations in a series of functions (physiological “depression”). Under minimum stimulus or under a situation of sensorial threshold, anomalies of utmost importance then appear. Only in these cases may we penetrate the sensory structures. The anodize appearance of these cases is derived from the fact that the essential disorder consists in a *scale shift of sensory magnitudes* and thus, if the stimuli are significant there are no functional deficits or failures.

Such a systematic alteration of multiple functions in “central” affection, even in the case of being limited to only one sensory system (paracentral syndrome), immediately runs up against several nosological entities admitted as independent and specifically localized, which in classic cerebral pathology are ordered into *sensory, perceptive* and *intellectual* disorders (anesthesia or blindness, astereognosis, agnosia), classification system more or less derived from the doctrine of mental faculties. An examination of the table 1 on visual functions highlights the great regularity of the involvement of the whole system, a regularity which permits, from just one pathological datum, to presume the alteration of the other functions. Contrary to traditional ideas, there is no isolated effect on any of the functions, nor do they recognize genuine nature (see desynchronization).

At this point we merely approach these problems with respect to the cases at hand, in which we only deal with a sensory field governed by other values of *sensory dimensions or parameters*. These parameters may be reduced to intensity, space and time, simple physical concepts common to natural sciences and with a quantitative character. *Intensity* refers to both the most simple sensory or absolute threshold, rheobase, and to the changes in intensity in the
differential threshold. *Space* or spatial dimension covers place or local sign, spatial acuity, extent and size, shape or figure, etc. *Sensorytime* includes reaction speed, chronaxia, rhythm frequency (fluttering, vibration etc.), and kinetic or movement processes. In a highly residual field, the values of these parameters tend towards zero, while they increase as the sensory field develops until the normal field value is reached. They are thus field parameters and depend on the active neural mass, i.e., they are dynamic parameters. All of this responds to physiopathological needs and considerably simplifies the sensory problem, avoiding bothersome classifications.

These parameters permit a natural description of the state of the sensory field, although they could be believed to only have applications in “lower” sensory functions, while the “higher” or intellectualized functions (figures, gnosis, etc.) would be outside. However, experience shows that the system is disturbed as a whole (see Table 1 on visual functions) and the scheme function is reduced according to the other activities of the field. It is thus possible to postulate that *between mere sensory function and gnosis activity there is a continuity via the one functional pattern*, even when there are successive stages of increasing complexity. The traditional separation and even opposition between higher and lower functions, as well as the notable preponderance of the former in classical cerebral pathology, are questionable criteria after this study. As mentioned, both types of functions have the same basis, and only the lower functions permit its elucidation.

Concerning the agnosia, it should be pointed out that while traditional theory remarks the amnesic defect or reproduction disorder (recognition), here the remark is made on the “production” defect, as shown clearly in orthogonal and halo-centric disorders, and very simply in the mixed figures, an aspect which facilitates its link with simpler functions. The gnosis is thus basically a mere ability of the field to gather information. It would be most useful to analyze the agnostic process in accordance with the concept of *information quantity* according to the recent theory of communication.

**VII. Cerebral gradients**

Accepting the traditional anatomic-clinical method, three syndromes are distinguished: marginal, paracentral and central, which in Fig. 1 refer to the visual system, taking the visual field as an index. This schematic illustration is the starting point for the concept of cerebral gradients which are based on a continuous variation.

When the visual field is used as a general pattern for all syndromes, the problem of localizations is simplified substantially and offers new perspectives so that the question seems to flow along its natural course. In traditional localizations, the three above mentioned syndromes, marginal, paracentral and central (Fig.1) would be referred respectively to, the field, perceptive defects and agnosia; completely heterogeneous terms which are useless for mutual comparison. Nevertheless, the syndromes are expressed by the same factor in the brain dynamics here developed: *the field*, whose form and extension may vary from one syndrome to another according to defined rules. The common denominator (the field) provides homogeneity and transition from one to another, i.e., it makes possible a continuity of appreciation. It would seem difficult to find anything better than the *field* to comply with the strictest requisites of functional localization since this is a spatial effect and it is better covered when an entity of an essentially spatial nature such as the visual field is used. Thus, the problem of cerebral localizations is covered here according to cerebral gradients thanks to the prior work of determining a) what is *localizable*,

29
and b) the *transition* between the syndromes.

First, we shall present several cases to adequately demonstrate the three visual syndromes illustrated in Fig 1. From a great number of first-hand observations of war injuries, I have selected the following three series of “visual cases”. Fig. 16 presents cases of *central scotoma* and incisures due to occipital pole lesion (macular projection in the striate area), examples of a marginal syndrome which is now partial. Fig. 17 illustrates cases of *hemianopsia with homolateral constriction*, i.e., of the paracentral visual syndrome due to unilateral lesion in the occipital convexity at a certain distance from the posterior mid-line. Fig. 18 refers to cases of central syndrome, with *concentric reduction* from unilateral lesion in a more “central” position than the previous type.

These are not equally common types. Cases of central scotoma require a highly circumscribed lesion in both occipital poles. Even after numerous civilian and war cases are studied, they are still somewhat rare. Cases of hemianopsia with some homolateral constriction and quite moderate concentric reductions are quite common in war injuries. On the contrary, significant constrictions are rare, both in the paracentral and central syndromes. In the figures we have designated our most affected cases but none reach the intensity of case M although case 1 in Fig. 18 is close. Each case is a permanent disorder continuing many years after the time of injury. It should be pointed out that the visual defect is purely cortical, in the scotomas and in cases of constriction, so that any peripheral cause such as papillary stasis is completely rejected.

Cases of *scotoma* have an anatomic explanation (macular projection), although given the diverse degree of visual acuity and density of the scotoma, the defect should be understood somewhat functionally, and rather referred to the number of destroyed neurons in the macular area. These purely marginal cases are closest to ocular defects. There is no reduction in visual organization and less in other sensory systems.

Contralateral hemianopsia of the paracentral syndrome would seem to be explained by purely anatomic causes, in accordance with the traditional homonymous cortical lateral hemianopsia. However, this could be excessively inexact since the lesion may lie *outside the calcarine* in many cases and the contralateral hemianopsia is then only a hemiambliopia with the defect disappearing under intense stimulus, thus being only a large lateral constriction of the same nature as the small homolateral constriction. It would then be an asymmetrical concentric reduction which is a transition towards the symmetrical concentric reduction due to more central lesion, or even further from the calcarine, and completely unadaptable to traditional anatomic explanation. Concerning the transitions, the field in paracentral cases winds up taking a more rounded or symmetrical form when using a highly intense visual stimulus or test, while one finds a trend towards hemianopsia in some symmetrical concentric reductions when the central isopters are determined. Case 1 in Fig. 18 shows an intense constriction in the 3/300 ordinary isopter, with asymmetry and incisure in the upper quadrant corresponding to the type of cortical lesion. But, under a very bright stimulus, the field widens and tends to be more regular although with a certain lateral asymmetry. This is a typical intermediate case between the paracentral and central syndromes.

The paracentral and central syndromes are satisfactorily explained by the physiological depression we have seen and, being dynamic syndromes, are fully ascribed to the system of gradients. The depression is accompanied by phenomena of dynamic action, reduction of functions etc. as set out for cases M and T. In case 1 of Fig. 17, the constriction is significant and the acuity descends proportionally, the dynamism being evident since this case showed a pathological
Figure 16: Series of *central scotoma* cases (first four) and central notches (final two), due to war lesion in both occipital poles. Note considerable deficit of acuity due to cortical affection of the macula. These cases have an incomplete *marginal* syndrome.
Figure 17: Various cases of lateral cortical hemianopsia due to occipital war lesion on one side of the mid line. In all cases there is a reduction of the conserved field. The more the reduction the greater the deficit of visual acuity and the more accused desynchronization (case 1 has a turn of 40°, case 2 only 6°). In this research, all these cases correspond to the paracentral visual syndrome with intensity varying from one to another.
Figure 18: Cases of *concentric reduction* of the visual field due to parieto-occipital lesions which may be very distant from the striate area. An extensive lesion which is close to the visual area produces much greater reduction (case 1) than when further away (case 6). All are cases of *central* syndrome, with the corresponding dynamic phenomena (first case presents a $90^\circ$ turn, case 6 has a $12^\circ$ turn).
Figure 19: Cranium-clinical correlation showing a synthesis of the three series of above cases: 1 concentric reduction; 2 asymmetrical reduction; 3 central scotoma. Note the ample participation of the parietal zone in the visual field.

visual turn of 40°. Furthermore, the patient showed a certain tactile and auditory repercussion in the contralateral side. In contrast, case 2 (Fig. 17) only reaches a turn of some 6°. In Fig. 18, all cases have a significant dynamic defect. The first one reaches a turn of 90° - 100° and the sixth reaches 12° with the worse eye, i.e. that contralateral to the lesion.

The symmetrical and asymmetrical constrictions, in accordance with the position of the lesions indicated, are found in both war and civilian injury cases of other authors, and although some authors had pointed out it, this question remains generally ignored.

An outlined image of the syndromes according to the double craneo-clinical aspect is offered in Fig. 19, which has a meaning similar to that of Fig. 1. One of the most notable results is the important participation of the extravisual cortex (occipitoparietal, parietal, temporal, etc.) in the “maintenance” of the visual field. Such a result is also valid for touch and the rest of the senses. Thus, the traditional separation between “projection” and “association” areas is in serious crisis, as here it is demonstrated that the “central” zone participates to some extent in the formation of the field. This situation should also lead to refute the distinction between “higher” and “lower” functions as set out in section IV. The idea of functional continuity through the cortical areas is thus imposed, in spite of a certain variation, leading to the system of gradients.

In the sense used here, a gradient is defined as “the proportion in which a magnitude varies with distance”. Since 1951 (doctorate courses), I have stood by the schematic representation shown in Fig. 20, where the different syndromes can be interpreted according to two types of gradients: specific gradient and integration gradient:
1. The specific gradient referred to vision, means that, beside area 17 (visual projection area), the density or intensity of the visual function is maximum (there is a critical zone at this point) and decreases progressively in more central areas and beyond. This corresponds to the curve (see Fig. 20) descending from the visual extreme towards the central zone (in the simplest form, intensity would decrease with the square of distance). This specific gradient only has contralateral action. The same may be said for the specific tactile gradient, and we could also add an auditory gradient, omitted to simplify the diagram. Such a type of gradient involves all sensory activity, so that for the visual field to be normal, acuity with a value of 1, etc., the action of the critical zone or that of greatest density is not enough: the whole visual gradation through the cortex must be involved. This gradient is essential and it is understood that it involves and assembles the factors of magnitude and position in this research on brain dynamics.

2. The integration gradient is more complex. On the one hand it is the consequence of the crossing between the specific gradients, given the extent they occupy. It is therefore reasonable that in the central zone where they interfere, there must be an action of mutual integration which is nonspecific, let us say, equipotential. This action is maximum in the central zone and minimal towards the projection areas (bell curve). In addition, this gradient contains bilaterality or interhemispheric effect due to the action of the callous, the projection zones being excluded as is known from anatomical and neuronographic studies.

In general terms, in lesions towards the extremes of the gradient functions, the defect is predominantly contralateral and unisensory. In “central” lesions, the defect is bilateral and multisensory, i.e. universal. With respect to the intensity of the defect, what is achieved in the extremes with a small lesion requires extensive lesions in the central area as indicated in Fig.
The gradients system is one of quantitative localizations according to action fields, offering an eminently dynamic conception. However this representation of gradients is no special hypothesis but an abbreviated formulation of the syndromes described in this research of brain dynamics. It is a mere abstraction of the observed facts and is an attempt to place them in a certain order. As opposed to the rigid separation into areas or centres according to traditional theory, here a functional continuity with regional variation is offered. For each point of the cortex, the combination of a specific action with a central action leads to characterize that point by a determinate value. Each point acquire then different properties from the neighboring points. In spite of this diversity, even in the most specific areas, there seem to be a certain unity with the rest of the cortex. For example, in a shrapnel injury in the tactile area which produced a complete permanent hemianaesthesia (a rare occurrence), in addition to anartria, apraxia of the mouth, etc., there was a clear constriction of the more internal isopters in the visual fields, to a greater degree in the contralateral eye. On the other hand, in a similar case but with less acute tactile symptoms, the visual fields were completely normal. This leads one to think that even the tactile projection area affects vision to some extent. This influence is quite small, and the tactile loss must thus be very great for the influence to become manifest. Similarly, there is a reciprocal action of the visual zone on touch, hence the final decline of the specific gradient must reach the opposite extreme of the horizontal axis Fig. 20.

These gradients come into play in the topographical disposition of a particular sensory system. The corresponding specific gradient evidently plays a role, but also counting on the bilaterality provided by the other gradient so that the system tends to be elaborated and integrated towards the central zone. In some cases, a gradient with a hemispherical dominance also has to be added. A further aspect is that of certain complex functions which could arise from the fusion of gradients of different systems. Thus, the genuine or primary alexia which would have its own characteristic field obtained from visual and acoustic fusion, leading to a “lexical” bell gradient between the two systems. It is clear that local fields and gradients with more restricted effects must arise along with the general types.

With respect to the effect of lesions on the gradients, it should be admitted that in the central syndrome at least, the proportions are maintained while the values are reduced. It is questionable whether this occurs or the distribution also varies in paracentral syndromes, but in whatever case it is essential that the change affects the whole system whose potential is reduced.

Finally, it must be noted that the gradients have an anatomic base (terminal paths, contralaterality, corpus callosum) and represent its dynamic aspect. The whole cortex is subjected to a common principle of organization (action field and its gradient), signifying a “desintellectualization” of the cortex. The sensory field projected on the marginal area (projection area) is only an outline which must be magnified and elaborated (integrated) towards the central zone. The ample activation of territory with strychnine, the extensive diffusion of the EEG response without anaesthetic, the series of “accessory” projection areas (supplementary, secondary, mixed) with characteristic physiological properties (greater threshold and latency, easy exclusion with anaesthesia, etc.) would lead, through statistical results, to the gradients.
VIII. Brain lesions

Brain lesions deserve several comments, particularly in the basic aspect of the so-called lesion magnitude. In this research of brain dynamics, the exclusively quantitative differences between cases M and T are perfectly explained by the varying quantity of cortical destruction on the same site, showing the same syndrome in different intensities.

In M it is a grazing cortical destruction, with entry and exit orifices of the projectile in the parieto-occipital convexity. In T there is a notable cranial opening consecutive to fracture, and a sinking in the upper zone of the same convexity. However the surgical operations revealed the dura mater intact and pulsating. A cortical contusion of considerable extension but without profound functional abolition must therefore be admitted. The involution of the disorders was marked in T and quite small in M (see details in “Dinámica Cerebral”).

At this point it is worth providing some indication of the types of cranial injuries and the corresponding cerebral disorder, thus we will have an adequate criterion to judge the magnitude of the lesion. Within our aims and as a mere empirical orientation, gaps should be distinguished from injuries with an entry and exit point. Contrary to what could be expected, large openings or gaps only cause weak, but possibly numerous or varied symptoms. These gaps are bone losses due to limited fractures and sinkings which only cause contusions in the brain and the functions generally recover well. Case T may belong to this group although his gap has a medium extension. There are also small gaps resulting from large fragments of shrapnel located at varying depths. In this case the subjects at times show symptoms of great intensity. Cases of projectile “entry and exit” are more appropriate for comparative study. In these cases, biparietal or bilateral shots must be discarded as they do not result in lasting disorders, as is the case of shots in one hemisphere with distant orifices or very long entirely medullary trajectory. On the other hand, in those with a short trajectory and a grazing tendency, the destruction of the cortex tends to be quite large with intense and permanent symptoms such as in case M. Several of the cases in Fig. 18 fall in this category. Apart from these ordinary circumstances, complicated cases may occur due to cicatricial sclerosis, secondary infections etc. in which it is impossible to foresee the lesion magnitude. There are also cases of small but deep injuries, such as the first case in Fig. 17, which initially showed a disorder of low intensity but with time worsened without any apparent cause.

Brain war injuries constitute the most favourable material for brain research, given the immense variety of injuries, the youth of the subjects, and the well defined nature of the lesions, complementing, and at times overrunning, civilian material. Brain pathology caused by war is more a “polio-pathology” or grey matter pathology, while brain pathology in peacetime is a “leuco-pathology” or white matter pathology (P. Marie). The first one thus has a particular character and is quite important with respect to the cerebral cortex.

The groups of cases in Figs. 16, 17 and 18, grouped according to similarity of lesion position and to the corresponding type of sensory defect, are what we may call families of lesions, the differences within the same group or family being in the magnitude of the involvement. Results may be obtained from the more homogeneous and easily compared cases of concentric reduction to confirm the thesis that the degree of constriction or reduction of the field parameters is more remarkable as more extensive is the lesion determined determined by the trajectory of the projectile.
If the question of the lesion magnitude is still considered insufficiently proven in brain injuries, more direct and precise data may be provided by neurosurgical cases which fit our thesis perfectly. Results from extensive experimentation on animals also favours this.

With respect to the surgical excisions, certain aspects should be mentioned which may be far-reaching doctrinally and provide new information on the functional coupling and dynamism of the brain hemispheres. Thus, a small parastriate lesion may be more perturbing functionally than an occipital lobectomy which suppresses a larger quantity of cerebral mass than in the first case. In the first case, both occipital lobes may be admitted to be perturbed, one significantly reduced in function (hemianopsia) and the other somewhat depressed (homolateral constriction) due to the interoccipital correlation, while in the second case, the complete excision of a lobe (even striate area) suppresses the function on one side while the function of the other side remains intact since the corresponding lobe is untouched and isolated or free from superimposed links. This result leads to the interesting suggestion that a paracentral visual syndrome may benefit functionally through an occipital lobectomy in spite of the greater loss of cerebral mass, as a new state of dynamic equilibrium is promoted in the interhemispheric correlation.

Many occipital lobectomies do not cause the repercussion in the other hemisphere we are used to observing in the paracentral syndrome due to parastriate lesion. However, when dealing with broad lobectomies, this effect is presented anew, probably due to a more central action. The question may pass on to a broader problem, and thus we may think that a central syndrome (bilateral disturbance or depression due to unilateral central lesion) could be “cured” to a degree through the hemispherectomy of the side holding the organic lesion. Absolute defects would remain (in vision at least), but intact functions without the least desynchronization nor depression would result, as shown in cases of hemispherectomy I have observed. We thus appear to be dealing with the quantity of energy to be distributed between the two hemispheres according to the functional unit. It should be noted that the suppression of the repercussion may be achieved more simply through the section of the corpus callosum.

We should also indicate that the “magnitude of lesion-magnitude of disorder” relationship has long been expressed by outstanding authors in both human pathology and animal experimentation. It contributes to the criticism of nosological units, to the problem of functional restitution, and to neurophysiological matters. However, such a relationship has not been highlighted enough due to the restriction imposed by the classical doctrine of specific centers. The classical doctrine of localizations still could be used in small lesions as an approximation, but in large lesions, as is the case M and others, the viewpoint of this research of brain dynamics is imposed in a natural way. Perhaps it could be said that the transition from de the classical to the dynamic conception derives from the magnitude of the lesion.

IX. Dynamic reduction and new cases of the visual inversion process

Although cases of intense disorders, where the dynamic phenomena may be examined conveniently, are not common because large lesions are rare, we will refer briefly to a more extensive casuistry in order to consolidate the concepts of this research of brain dynamics. We focus on the visual inversion process.

Fig. 21 covers more than twenty cases with visual disorders presenting a chronic manifestation
Figure 21: The curve illustrates the order of a series of new cases in relation to the reduction of the visual field and the perceived turn in the visual image. Cases M and T are included for comparison. The corresponding visual acuity is indicated in parenthesis.

of the pathological visual inversion process in varying degrees. The cases are ordered along the curve according to the degree of the perceived turn of a vertical text arrow and to the width (constriction) of the visual field. The majority were injuries with more or less intense concentric reduction, a few others with hemianopsia and homolateral constriction (paracentral visual syndrome). In comparison with cases M and T, we see that there were few cases with considerable turn none has yet reached the intensity of M. Only twelve cases have a manifest turn of over 10°, half of which are moderate and even less have a sufficiently marked turn to be used in an extensive study of spiral development. On the other hand, many cases with little inclination are found. Some of which are easy to identify, while others are less accessible, almost within the observation error.

Several injuries with a large turn are included in Fig. 18 (the first case presented 90° turn, the sixth almost 15° others near 15° are in Fig. 17 (the first case reaching 40°, the second only 6° ). From these figures and the curve in Fig. 21 we see that the greater the constriction of the visual field, i.e. the more reduced the sensory dimensions, the greater the turn under minimum stimulus, and the greater the dynamic reduction, i.e. the greater the trend towards the residual function field (as seen in the corresponding visual acuity). The curve in Fig. 21, which is only an approximation, shows a functional continuity between the reduction of the visual field and the width of the turn, highlighting the non-linear quantitative relationship between them. The field may be constricted significantly up to 45°, and the turn may only reach 10°. But for greater constrictions of the field, the width of the turn, i.e. the desynchronization, becomes highly significant. As a practical rule, one cannot expect to find cases with significantly marked turns of up to 100°.

In the most acute cases, curves of directional recruitment were determined in function of the visual angle subtended by a vertical test arrow, leading to results similar to those found in
case M. A well fitting of the respective parameters of the curves (see IV) is obtained from the comparison between the various cases. The most acute cases also showed considerable agnostic defects and accentuated chromatic weakness, visual fatigability, phenomena of summation and other disorders. The characteristics of the inversion process comply with that established in this research of brain dynamics: the greater the blurredness, the greater the inclination, accompanied by micropsia. At times, even subjects with a large turn were unaware of their disorder.

In some cases, as well as the turn in the frontal plane, there was a combination with turn on the sagittal plane, which has already been observed partially in case T. The majority of the important cases showed several peculiarities besides the turn, especially in the most extreme case (case 1 in Fig. 18). In this case, in addition to suffered from a significant constriction of the visual field and acuity deficit, he showed a complex and quite accentuated syndrome of visual and chromatic agnosia, amnestic aphasia and particularly primary alexia of the verbal-literal type. Furthermore, he was close to the orthogonal disorder since in upright and inverted numbers the subject noticed something strange but he was unable to pinpoint the change in the orientation.

As already said, all cases in Fig. 21 belong to the central or paracentral syndrome, whose localization in the parieto-occipital convexity is indicated in Fig. 19. It should be pointed out that the turn is obtained in both left and right lesions.

Besides these cases of permanent disorder in the visual direction, we have observed transitory cases, primarily during epileptic auras. Naturally these cases are not as important as the permanent ones, although they do contribute to some extent to our knowledge of the inversion process. Apart from the attack, the subjects (old brain injuries) show no visual disorder worth taking into consideration. Permanent cases with small turns which intensify during the auras exist, but other subjects only show the turn during the attack, whether this results in grand mal or in simple absences, during which the real visual scene seems to be brusquely tilted and at other times almost inverted. In this type of sudden turn, visual forms undergo less deterioration than in the turn due to permanent desynchronization, although they present always some blurredness. The subjects with this type of attack are aware of their disorder in visual direction and tend to refer to it spontaneously. A quite curious case is that of a subject with an old injury with shrapnel lodged in the medullary region or parieto-occipital subcortical zone, entering through the frontal region. He suffered for a long time and in a identical way absences during which he had hallucinations in an inverted position: shortly after entering the trance, he visualized a door appearing near the roof which opened and soldiers filed through upside down for a certain time.

We have collected more than 100 cases of visual turn in the bibliographic research covering a period of about 50 years. However, this phenomenon is practically unknown and goes unnoticed even in the specialized works. The vast majority of the cases collected present sporadic manifestations of tilted or even inversion visual in diverse attacks. Permanent cases are less abundant and a moderate visual inclination is then more common. In general, all these case are referred in publications of simple isolated notes or of common clinical data. It is interesting to note that the cases where it was possible to determine a lesion, showed a clear predominance of the parieto-occipital-temporal zone.

Finally, we make simple reference to the auditory system concerning the dynamic reduction, i.e. the involvement of a whole sensory system and according to a physiological order. In effect, in many cases of aphasia, particularly sensory aphasia, it is demonstrated that the aphasic
defect is not an isolated disorder, in such a way that the greater the disturbance in language, the greater the deficit shown in the audiogram curve which expresses the simple excitability of the scale of sounds. The “semantic” defect thus shows much less deficit of simple hearing than the “amnestic” defect, residual to a sensory aphasia.

X. Bilateral tactile involvement in unilateral lesions

Some comments on the paracentral tactile syndrome will be made here. Fig. 22 illustrates a case with an injury in the right cortical tactile zone in its upper part, i.e., towards the region of the lower extremity, offering a residual disorder consisting in a slight contralateral hemiparesia predominating in the lower extremity, and also in a hypoesthesia not only on such contralateral side but partially on the homolateral side, as shown in the figure.

In accordance with the localization of the cortical lesion, the greatest tactile defect corresponds to the lower contralateral extremity, a defect of distal predominance which reaches its maximum intensity in the toes and extends more weakly until the mid-thigh and further up. On the other hand, it is much less intense and extensive in the upper contralateral extremity, where the degree of disturbance is close to or somewhat above that of the lower homolateral extremity of the lesion. The contralateral face showed a light tactile deficiency, as does the homolateral hand. In accordance with the dynamic reduction or physiological depression, the defect includes each tactile quality in a defined order (temperature > pain > pressure), the vibratory sensitivity, the sensation of passive articular movement, etc., and with an intensity of the involvement in relation to the topography described above.

We are basically facing an asymmetrical concentric reduction of the tactile field, similar to that of the visual field, or expressed otherwise, this is an “unequal bilateral disorder in unilateral cortical lesion”, with the maximum defect or greatest constriction of the sensorial field on the
Figure 23: Asymmetric bilateral tactile disorder expressed according to the thresholds of vibratory sensitivity. Normal value: 900–1000 vibrations per second.

Such tactile cases are more abundant than one might believe. If the exploration of the patients were sufficiently detailed and complete, the more or less acute bilateral defect could be generalized in at least some types of lesions. Fig. 23 presents a case of smaller tactile cortical lesion than the previous one, with a more paracentral situation and close to the area of the hand. This subject had certain paresis in the fingers of the contralateral (left) hand. Tactile examination revealed a hypoestesia in that hand expressed by the correlative deficit in functions (sensitivity, passive articular movement, Weber, pain threshold, vibratory threshold, a certain stereognosis), but there were also indications of disturbance in the homolateral hand. This is an asymmetric bilateral disorder which could be exemplified better using the corresponding thresholds of vibratory sensitivity (with electric vibrator of regulable frequency) as indicated in Fig. 23. The distribution of the disorder is typical, with the defect predominating peripherally, i.e., in a cubital and distal direction. The defect is thus maximum in the contralateral little finger, although the homolateral little finger is affected in a similar manner to the contralateral thumb, and the homolateral thumb may not be completely intact. A similar result was obtained when determining the Weber thresholds.

The other case in Fig. 24 is of the same type, but presents a disorder which is more extended to the whole body in a similar manner to the first subject (Fig. 22), although the defect in this case (Fig. 24) is much less intense. These two cases (Figs. 22 and 24) show bilateral asymmetry and also asymmetry along the axis of the body, the maximum defect corresponding to a lower extremity. According to traditional doctrine, the latter defect (that of the lower extremity) is the only one that could be explained anatomically, but the disorder is really more dynamic and reaches the whole tactile field, although heterogeneously, i.e., according to a doubly asymmetric
Figure 24: Bilateral tactile disorder with greater area than in the previous figure 23. It is determined by the same method of thresholds of vibratory sensitivity. Normal value: 900–1000. Note the predominance of cubital-peroneal disorder and the double asymmetry in the lateral direction and from below to upwards.

While there are some clinical observations on this type of affection in the bibliography, it appears as an obscure matter. Foerster in particular insists on the bilateral action of area 5a and 5b (according to other authors, area 7) or “supplementary tactile area”. Many of our cases fit well this paracentral localization. It can be said that the bilateral defect does not exist in strictly “peripheral” (projection paths) lesions. The asymmetric bilateral tactile defect also occurs in other paracentral zones, and even in a more symmetric manner in the central zone. One cannot ignore the fact that an important aspect of the paracentral syndrome is its transitional character to the central syndrome. Finally, it must be recalled that there are several very important classical tests on the bilateral tactile effect from a cerebral hemisphere in animal physiology.

XI. Concluding remarks

The most outstanding feature of these studies on cerebral dynamics is its physiological basis, which is manifest from the outset through the phenomena of dynamic action in cerebral excitability, instituting far-reaching principles which lead to simpler and more systematic notions than those of the traditional doctrine. To pass from a mere empiricism to a more causal theory signifies the transition from an anatomoclinic or “static” conception, to a physiologic or dynamic anatomoclinic conception, common evolution to all types of knowledge. Dynamism alludes to
transformation, development, etc., and in an abstract sense to continuity. Here, dynamism involves a physical and quantitative “mechanism”, without resorting to other principles, and is expressed by the value of excitability parameters in function of neural mass, spiral development and the vectors which determine it, in the cerebral gradients, etc.

The place of this research of brain dynamics (see “Dinámica Cerebral”) with respect to the “localist” and “antilocalist” theories may be of practical interest. The former, a more traditional direction, is accepted here within the general criteria of functional regional heterogeneity, which should figure as a definitive acquisition, but is relegated when innumerable specific anatomic centres are to be detailed, as well as the series of nosologic categorical entities it is derived from. It may be said that the localism, being starting point, it is surpassed. Functional or antilocalist orientation, at times brighter than the former but much more diffuse, as well as being in opposition to the excesses of localism, tends towards the functional analysis of syndromes and complexes which naturally must be assumed. However, it is quite imprecise in all matters, as the analysis is primarily aimed at the higher or upper functions, ignoring the pillars of sensory organization, and is far from all physiological basis. Furthermore, the problem of localizations is unattended and a completely confused and indeterminate action of “totality” is proposed.

Each orientation represents an approximation and plays a role within certain aims. Traditional doctrine thus continues to accomplish its function in one form or another in clinical diagnosis, becoming progressively, through the theoretical approach, into an attenuated localism, while badly defined, as a result of the conflict between opposed theories which tend naturally to complement each other.

In this research of brain dynamics, the subject of localizations is clearly focussed according to the two factors of the lesion, magnitude and position, where localism (position) and antilocalism (magnitude) can be assembled. It is even more correctly approached in the cerebral gradients, where the separation between projection and association areas seem to be erased. In this way, the divergence between the higher (or upper) and lower functions also disappears. If we had to define brain activity according to these results, we would simply assert that it consists of the organization of the sensory field.

Finally, it may be said that this research of brain dynamics is a new stage connected to previous ones. As mentioned, as well as being physiological it is still anatomoclinical. In fact, it includes anatomic concepts (hemispheres, corpus callosum, primary and secondary areas, crossings, etc.), physiological concepts (excitability, summation, desynchronization or dephasing, sensory parameters, cerebral gradients, etc.) and physiopathological concepts (central, paracentral and marginal syndromes, residual field, spiral development, etc.). It provides new signification and interpretation to known facts, and supplies more original notions. It is related to well established knowledge but is directly supported by its own data.

In summary: According to the gradients system, the more “peripheral” the cortical lesion, the greater the predominance of unisensory and contralateral defects, and conversely, the more “central” the lesion, the greater the tendency towards the bilateral and multisensory defect. The lesion should be more substantial the more centrally it is located to produce a functional depression, expressed in the corresponding sensory field by the reduction of the sensory parameters (intensity, space and time) and by the turn in direction (crossings), i.e., it will show a spiral development, whose amplitude will be a function of the magnitude of the lesion.
ENGLISH TRANSLATION OF THE INDEXES
OF VOLUMES I AND II OF THE BOOK

Dinámica Cerebral, Vol. I (1945) [1, 2], Vol. II (1950) [1, 3]

Author: Justo Gonzalo

English translation by John Vo Medina and Isabel Gonzalo Fonrodona

VOLUME I

INTRODUCTION .................................................................1

First Part
OVERVIEW
(Fundamental findings and the conception of brain dynamics)

Introduction .................................................................15

1. FUNDAMENTAL FINDINGS

Development of the research ...........................................16
New agnosic syndromes. Physiological basis of the brain activity.

Singular precedents about the agnosia research ....................20
The singular Schn. case of GOLDSTEIN and GELB. Various considerations.

The finding of dynamic action phenomena .............................25

Rebuttal and experimental extension of GOLDSTEIN and GELB agnosia researches .................................................................39
2. THE NEW CENTRAL SYNDROME OF THE BRAIN

The M and T cases ................................................................. 48

The central syndrome ............................................................. 57

Questions of brain dynamics arising from the central syndrome ............... 63
The central syndrome as a crucial experiment for how the brain works. 1. On physiological level (the mass action and the nervous subordination) 2. On sensory structures 3. On cerebral organization (the two extreme types of cerebral syndrome; effects of lesion position and magnitude in the brain dynamic system).

3. DYNAMIC ANALYSIS

Dynamic action and experimental analysis. Physiological levels ............... 70
Fundamental disorder (slow reaction and asynchronism). Physiological levels (excitability curves, types of level).

Basic experiment of dephasing or asynchronism ............................. 74
Set of asynchronous curves. Recruitment of delayed (dephased or lagged) levels.

Synchronization by means of reinforcement ................................. 79
Sensory variation by means of reinforcement. Recruitment by means of iteration.

Sensory “dephasing” ............................................................... 83
Increase of sensory intervals and creation of new intervals (effects of all or nothing, and partial reaction). New functions and sensory analysis.

Dynamic reduction ............................................................... 86
Functional disintegration and exclusion of complex factors (or higher functions). Reversibility of the functional reduction.

**Sensory structures** ....................................................... 89
Sensory differentiation according to nervous synchronization. Functional unity. Degree of alteration of the sensory system. Qualities, shapes and schema.

Second Part

**SENSORY DYNAMICS**

(Sensory structures according to cerebral synchronization)

Introduction ................................................................. 95

(I). VISUAL FUNCTIONS

Introduction ................................................................. 96

I. GENERAL EXCITABILITY

1. ELECTRIC EXCITABILITY

**Intensity-duration relationship (HOORWEG’s law)** ....................... 97

The reinforcement phenomenon .............................................. 107
Change of excitability by means of reinforcement. Saving the stimulus by the reinforcement. Action of several types of reinforcement (intersensory summation). Peripheral subordination.

Iterative excitability or summation by latency ............................... 113

Conclusions on electric excitability ........................................ 125
Several types of excitability. Permeability to reinforcement and to iteration. Excitability and nervous summation.
2. LUMINOUS EXCITABILITY

Excitation by means of appropriate stimulus ........................................ 126
Features of luminous excitability in M and T cases. Effective time for the different types. Evolution of luminous sensation (increased latency and perseveration). Factors involved in the sensation.

Luminous adaptation ................................................................. 133
Slowness in accommodation and sensitivity deficit. Hemeralopia and nyctalopia

Intermittent stimulation (fusion frequency) ................................. 140
Excitability for fluttering and fusion of stimuli. Modification of the curves due to increased persistence. Central disorder.

II. COLOR VISION

1. SPECTRUM EVOLUTION

Chromatic research ................................................................. 145
Complexity of the chromatic disorder. General features of M and T cases (chromatopsia, dyschromatopsia, tritanopia).

Spectrum vision ................................................................. 149
Chromatic limits in the spectrum. Influence of illumination and reinforcement. Other chromatic tests.

Chromatic discrimination sensitivity ......................................... 154
Curves of spectrum discrimination sensitivity for M case. Great deficit and alteration of maxima.

2. CHROMATIC DYNAMICS

Photochromatic and photoheterochromatic intervals .........................157
Increase and disintegration of the photochromatic interval depending on colour. Disintegration (or splitting) of white colour. Thresholds of chromatic excitation.

Dyschromatopsia and chromatopsia. Simple and compound colours ......166
colour, lack of chromatic ingredients).

**Inversion of chromatic isopters** .................................................. 179
Inversion of chromatic isopters (chromatic order in the visual field according to excitability characteristics). Change in dyschromatopsia along the visual meridian. Considerations about the theory of functional duplicity of the retina.

**Alteration of chromatic induction phenomena** .......................... 187
Increase of marginal contrast. Abolition of the negative after image and persistence of the positive one. Eidetism.

3. **THEORY OF CHROMATIC DISCRIMINATION**

**Chromatic discrimination** ................................................................. 190
Tritanomaly and tritanopia. The observed facts according to the two classical chromatic theories. Chromatic differentiation; development of colours.

**Cerebral problem of colours** ............................................................... 196
There is not anatomical localization of colours, but a general disorder affecting the chromatic organization.

III. **VISUAL SHAPES**

1. **VISUAL FIELD**

**Concentric reduction** ................................................................. 198
Dynamic reduction of the visual field in M, T and Schn. cases. Different types and changes induced by reinforcement.

**Functional relationships of excitability in the visual field** ............... 207
Experimental research of concentric reduction. Variation of excitability in the visual field according to different functional types. Enlargement of the visual field by increasing the stimulus intensity and by reinforcement action.

**Organization of the visual field** .................................................. 216
Functional unity of the visual field. Dynamic phenomena of the visual field (monocular polyopia, macular deviation or pseudo-fovea, autokinetic effect, metamorphopsia).
2. FLAT COLOURS (VISUAL SPATIAL LOCALIZATION)

Vision of flat colours (distorted chromatic contour perception) .......... 219
Flat colours and surface colours. Precedents. Physiological basis of the disorder.

The spatial localization disorder ......................................................222
1. Properties of the pathological vision of flat colours (flat orientation, thick and thin colours). 2. Irradiated localization (frontal and lateral irradiation, different irradiation for different colours, irradiation in T case, influence of illumination and reinforcement, degrees of chromatic spatiality, totalization of the shape due to irradiation). 3. Asynchronism or dissociation between simple sensation and spatial localization.

3. VISUAL SHAPES

Visual acuity .................................................................232
Visual acuity in M case according to different vision types (monocular and inactive, binocular and reinforcement, etc.). Idem in T case. Curves of acuity as a function of illumination. Acuity according to physiological level.

Visual disorder in the perception of movement ......................... 240
Precedents. Disorder in M and T cases. Disintegration stages of movement sensation. Phenomenological peculiarities (acceleration, path reduction, inversion, etc.). Genesis of the disorder.

Vision of shapes (figures and body elements) ......................... 249

IV. VISUAL DIRECTION (ORIENTATION)

1. VISUAL DIRECTION DISORDER

Precedents on the visual direction problem ............................. 259
Historic precedents. Clinical precedents.

Diversely inverted vision (M, T and other cases) ..................... 264
The direction disorder in M and T cases. Other cases of personal examination. Diverse cases as different degrees of the same disorder.
General features of the disorder ............................................. 271

Direction according to the stimulus intensity (clear vision, straight up direction, indistinct vision, inverted direction). Factors to be considered in the altered direction (stimulus, receptor and nervous central state). Inclination limits depending on the different cases. Re-inversion by means of reinforcement (diverse reinforcements). Types of turn. Binocular duplication of direction. Compensation method.

2. VISUAL DIRECTION DYNAMICS

Basic experiment in visual direction (variation by the stimulus intensity) 276

Direction as a function of the visual angle (apparent size) in inactive state and under reinforcement. Experiments about direction as a function of the luminous stimulation: 1. Recruitment of the different stages according to the stimulus intensity in M and T cases (curves of variation according to Fechner’s law; correspondence between apparent direction, shape and color; types of plots in T case; alcohol influence in T case; cooling tests in T case; increase of functional deficit after epileptic attacks). 2. Asynchronous set of delayed functions. 3. Development rate of visual direction.

Visual direction according to “types of vision” ............................. 297

Types of excitation and binocular reinforcement in visual direction. Curves of visual direction for different types of vision in M case. Comparison between cases M and T for different types of vision. Maximum turn for the different types of vision in M case. Idem for T case.

Synchronization by means of reinforcements (variation of the central nervous state) ............................................................. 305

Synchronization by means of muscular reinforcement in M case (agreement with Fechner’s law; incomplete re-inversion). Synchronization by the bi-effect (considerable change in the direction perceived by one eye by means of luminous stimulation of the other one which is not seeing the test object). (The same law as in muscular reinforcement). Other ocular reinforcements. General features of the reinforcement.

Visual direction according to the receptor state ............................ 312

Direction deviation in the visual field (central and peripheral vision; curves for the three types: inactive M case, M case under reinforcement and T case). Changes in the perceived direction by glare (light adaptation and variation in the direction).

Various complementary experiences ...................................... 319

a) Drawing and apparent direction. b) Direction during movement (influence of mobil velocity, deviation according to different type of vision) c) Lability of direction even without deviation. Tubular vision. d) Influence of head posture on the perceived direction (various cases, spontaneous compensation of the apparent deviation, Aubert phenomenon).
3. THEORY ABOUT THE PERCEIVED VISUAL DIRECTION (ORIENTATION)

Functional complexity of visual direction ........................................... 328
Visual-haptic combination according to empirical authors. Rehabilitation of the importance of the retina factor (inversion of direction by independence of the retina).

Disintegration and degradation of the visual direction function ............ 330
Disintegration of the complex direction function (exclusion due to the asynchronism of the secondary or haptic factor). The abnormal interval (partial reaction, interval amplitude, variation of the direction as a function of the stimulus intensity according to the normal sensory growth law). Altered direction according to the deficit in the visual cerebral organization.

V. VISUAL SCHEMA

1. THE SCHEMA FUNCTION IN VISUAL SHAPES

The ‘schema’ function ........................................................................ 335
Meaning of schema. Disintegration and diffuse concepts (various tests in inactive state, and change by the reinforcement). Change of vision and constructive deficit of the schema (illusions, disintegration, etc.). Pregnancy reduction in the organization of shapes.

Visual behaviour ................................................................. 348
Intelligence is solidary of perception. Various alterations in ordinary lifetime and in usual tests. Disorder behaviour concerning schema and abstraction (simultaneous agnosia, chromatic agnosia).

Structure of the visual agnosia (schema dissolution) ....................... 357

2. SCHEMA FUNCTION IN THE SPATIAL ORIENTATION

Alteration of the orthogonal orientation ........................................... 365
1) Orthogonal property of a figure: figures with orthogonal meaning and indifferent figures; orthogonal orientation disorder in M and T cases; connection with other visual orientation disorders.
2) Loss of orthogonal orientation: figure appearance is independent on its orientation (various tests with figures and body elements), experiments about perception of numbers.
and writing, orthogonal function and types of vision (change by means of reinforcement, etc.), stages of the orthogonal disorder, behaviour of T case.

3) Structure of the orthogonal orientation (spatial level): ontogenetic and phylogenic parallelism (orientation of figures in the child and in primitives), the spatial level and the persistence of the figure, functional overload and dissolution of the spatial schema.

**Allocentric orientation disorder** ................................................. 383


**COMPILED VISUAL FUNCTIONS** ................................................. 388

**VOLUME II**
(Following the second part)

**(II). TACTILE FUNCTIONS**

**Introduction** ................................................................. 393

**I. GENERAL EXCITABILITY**

1. **ELECTRIC EXCITABILITY**

   **Intensity-duration relationship (HOORWEG’s law)** ................. 395
   Excitation curves for elementary sensorial activity. Rheobase and chronaxia tables, etc.

   **Reinforcement action** .................................................. 400
   Permeability to the summation effect according to the excitability deficit. Functional growth as a function of the logarithm of the reinforcement.

   **Iterative excitability** .................................................. 402
   Normal iteration for the sense of touch. Laws of numbers and intervals in relation to the excitability deficit (plots and tables), summation amplitude. Homogeneous repercussion in the central syndrome.

2. **MECHANICAL EXCITABILITY**

   **Generalities about the appropriate stimulus** ....................... 407
Useful time. Modalities of cutaneous stimulus. Temporal development (evolution) of tactile sensation (perseveration, reactivation, etc.). Sensory fatigue. Adaptation. Weight or force sensation (underestimation, sensory comparison between inactive and reinforcement cases, etc.)

**Vibratory sensitivity (intermittent stimulation)** .......................... 413
Precedents about vibratory sensitivity. Evolution of the process (different stages by excitation accumulation: latency, fusion and trembling). Overestimation or trembling acceleration. Exclusion of high frequencies (optimum frequency and refractory period). Vibratory comparison between inactive and reinforcement cases. Frequency discrimination. Interpretation of the supposed clinical dissociations of the vibratory sensation.

Appendix

**Bilateral tactile disorder in unilateral cortical lesion (central syndrome)** ........... 422
Interpretation of the cases of OPPENHEIM, GOLDSHTEIN and FOIX as ascribed to the central syndrome. Another cases, cases of cortical extirpation. The Schn. case of GOLDSHTEIN and GELB. Quantitative examination, degree of the disorder.

II. TACTILE QUALITIES

1. **DYNAMICS OF THE TACTILE QUALITIES**

   **Generalities. The heterotactile interval** ........................... 433
   Tactile qualities (pressure, pain, temperature), different level of excitation. Heterotactile interval. The physiological non-individuality of qualities (different degrees of differentiation).

   **Pressure and pain** ....................................................... 435
   Pressure as elementary activity of the tactile function. The pain-tactile interval, enlargement of the interval depending on the cases and excitability state (tables of values, etc.). Prick sensation. Pain by iteration.

   **Thermal sensation** ...................................................... 443
   The thermal-tactile interval (graphs for cold and hot). Stimulation modalities. Dilation of the temperature neutral zone, pronounced deficit of thermal sensation, thermal differential sensitivity.

2. **ORGANIZATION OF THE TACTILE QUALITIES**

   **Criticism of the so-called “tactile dissociations”** .......................... 451
   Reviewing of anatomical and clinical ideas and evolution to physiological conceptions. Apparent dissociation due to lack of quantitative research of the functional whole (relative
dissociation, non-absolute dissociation).

**Organization of the qualities ........................................... 455**

New concept about tactile organization; elementary tactile activity (pressure), true qualities (pain and temperature), dynamic classification (tangibleness and qualities).

III. TACTILE SPACE

1. TACTILE LOCALIZATION

**Spatial disorder ............................................................... 460**

Perturbation order in tactile spatial localization (stages or gradations). Difficulties of Schn. case and its interpretation according to new research results. Development of the present research.

**Phenomenology of dephasing (or delay) in tactile localization ............. 472**

Five spatial stages:


II. *Deviation towards the anterior middle line of the body*: Middle line localization of stimulus (deviation towards the anterior middle line as a maximum effect of proximal deviation. Predilection regions (the three medial sectors). Summary.


IV. *Proximal deviation*: Homolateral localization with moderate deviation. Regional localization, oval of irradiation, etc. Various tactile discriminations. Summary.

V. *Specific or normal localization*.

General summary: Evolutive process (factors of the irradiation and proximal deviation). Synoptical table of the tactile spatial development.

**Meaning of the spatial disorder (structural change of sensory field) ...... 502**

1) Analysis of the *relative* disorder of localization (irradiation); comparison with chromatic irradiation (flat colours), shape of the irradiation in relation to shape of the sensory field.

2) Analysis of the *absolute* disorder of localization (deviation towards the middle line); concentric reduction of the body schema, incongruence of fields (sensitive and spatial fields), privilege of the middle zone.

3) Structural change of tactile sensory field; residual field (constricted by the middle deviation and irradiation).
2. LOCALIZATION DYNAMICS

Recruitment of stages in the localization .............................................. 511
Quantitative relationships in the functional dephasing (delay, lag or destaging):
a) Recruitment with a single stimulus: Tactile irradiation as a function of the stimulus intensity (logarithmic law). Deviation towards the middle line as a function of the stimulus intensity (the extremity case), curves for inactive and reinforcement cases, tables of values, etc.
b) Recruitment with iterative stimulus: Medial deviation according to number of stimuli. Idem according to the amplitude of the interval between two stimuli (influence of the relative refractory period). Mechanical vibratory stimulation with tuning forks (vibration frequency and recruitment, etc.).

Asynchronism in the localization .......................................................... 524
Asynchronous set of curves for different spatial sensory levels; greater perturbation of higher levels. Abnormal intervals or stages in relation to cerebral deficit, partial reactions instead of unitary action. Various intensity-duration plots, specially for the five phases (stages) of the localization.

Temporal development ................................................................. 533
a) Temporal development of tactile localization process. Temporal evolution of sensory degradation (fatigue, residual faint), sequence of stages.
b) Posthumous summation (reinforcement action on the residual mark). Effect dependent on the reinforcement delay. Temporal summation and “spatial” summation.

Theory of tactile localization (spatial organization) .............................. 538
Spatial organization, empiricism and nativism. Criticism of normal and automatic localization (specially concerning the Schn. case, various conceptions, etc.). Refutation of the sign of complex associative nature. Spatiality as an elementary attribute; abnormal spatial localization in the residual function field, innate correspondence, the functional individual entity is the result of the organization.

3. SPATIAL DISCRIMINATION

Spatial acuity (Weber) ................................................................. 549
The motion on the cutaneous surface ..............................................557
The motion and the spatial stages (static and kinetic stages). Phenomenology (apparent acceleration and shortening of the path). Origin of the disorder (temporal and spatial constriction). Identity of the kinetic disorder between vision and sense of touch.

Figures or shapes on the cutaneous surface ......................................561
Static shapes. Kinetic figures. Longitudinal dysmorphism. Mistakes or diverse illusions due to the tactile field instability, change by reinforcement.

Deep kinetic sensitivity or articular sensitivity .................................567
Quantitative value of the functional reduction, intrinsic alteration of the movement (constriction and acceleration).

Passive movement:
1) Iterative stimulation or repeated movements: different stages in the localization and kinetic perception, various cases (extremities, head, etc).

Active or voluntary movement, incongruities in the perception.

Schema of the body and touch with hand ........................................578

a) Reduction of the schema of the own body: Fragmentation of the model, abnormal flexibility, weight and strength, body size.
b) Reduction of the sense of touch with hand: Flat touch (flexibility and hardness of the objects), reduced stereognosis.

IV. TACTILE DIRECTION

1. TACTILE DIRECTION DISORDER

Overview .........................................................................................585
Discovery of the spatial inversion in the sense of touch. Difficulty to be objective in tactile phenomena. Tactile inversion and the stage III of spatial localization. General laws similar to optical inversion. Generalization of spatial inversion to all the other sensory systems, and extension of the dynamic postulates (spiral development, psycho-physical correspondence).

Tactile direction in the cutaneous stimulation (surface sensitivity) ...... 592

a) Inversion of a point: On the head (the cranial vault as inversion region, trajectory of the inversion process, centripetal contralateral translation or spiral development). Extremities. Trunk. The three independent areas of inversion. Polyesthesia phenomenon.
b) *Inversion of a line*: Distal stimulation in a extremity. Correspondence between the localization and the direction of the line, graphs about the recruitment of direction (direction depending on the line pressure, etc.). Direction of the perceived turn.

c) *Inversion of movement*: Stimulation conditions. Sensory dimension of space and time. Spiral deviation or spiral deformation of the trajectory.

**Direction in articular movement (deep sensitivity)** ............................... 614

a) *Inversion in passive movements of the extremities*: Localization deviation and consequent change of direction according to the articular oscillation intensity.

b) *Inversion in passive movements of head*: Change of direction dependent on the intensity of the oscillation movement. Clockwise rotation.

c) *Inversion in active or voluntary movement* (incongruities): Sensory-motion incongruence of direction. Automatic change of references and pseudo-active movement. Another types of incongruities by change of the kinetic shape.

**Direction in the body schema** .............................................................. 627

Deviation of the entire body schema, perceptible inclination limit. Spiral rotation of the body. Body schema during rotation (size, apparent height, etc.). Tests of imitation of the inclination under reinforcement, and tests of compensation of the inclination in inactive state.

2. **COMPLEX PROCESSES OF TACTILE DIRECTION**

**Inversion and deviation on walking** .................................................. 633

Subjective changes in walking due to latent addition (summation) of the steps.

1) *Iterative excitation by the steps*: Subjective reduction of step number. Recruitment of the direction of walk according to the number of steps, influence of step frequency (walking with summation effect and without it), influence of the energy of the step.

2) *Space and time during walking*: Spatial-temporal constriction; apparent path, apparent acceleration.

3) *Walking trajectory*: Apparent trajectory. Spiral development of walking (tables of numerical values about steps and direction). Graphs about iterative recruitment of direction as a function of number of steps. Similarity with the visual phenomenon of the same kind (perceived visual orientation of an oscillating movement dependent on the number of oscillations).

4) *Localization of the movement on the body*: Size of the body schema, translation over the floor, etc. Evolution of the localization of the movement and of the size of the body schema. Sensory illusion of translation.

5) *Direction of the body schema during walking*: Direction of body schema in space (rotations, subjective slope). Residual phenomena of the inversion of body schema. Manifestations in everyday life.
Summary. Size and direction, spiral evolution in space.

Motor deviation of the body (postural deviation) by induced action ..... 659
Objective motor deviations and sensomotility. Postural deviation of head, postural deviation of body (or Romberg), idem for index fingers, idem for walking. Postural deviation of the body schema (new comfortable position). Functional complex of the sensomotility, induced postural deviation (motor deviation induced by tactile deviation).

3. THEORY ABOUT TACTILE DIRECTION

Direction and localization in the spiral development ..................670
1) Differences from allochiria (first-hand observations and other ones, other similar states, visual alloesthesia, etc.).
2) Inversion and deviation in spiral development for vision and touch senses (similarities and differences).
3) Spiral development of the sensory field.
4) The mentioned spiral development as ‘sensory organization law’ (second principle of cerebral dynamics).

Mechanism and structure of the spatial direction ..................683
The spatial direction as a general sensory problem.
1) Origin of the inversion in the primary cortical area (contralateral and inverted cortical projection, path decussation, autonomous zones of the sensitive area and independent zones for inversion). Perturbation degrees according to the different syndromes, anatomical and physiological factors.
2) Cerebral mechanism for the direction [cortical area (marginal) and central action], neurosensory correspondence (reduced brain and residual field, normal brain and full field). The continuity of the field and direction.
3) Revision of theories about spatial direction: objection to empirical or associative theories.
4) Basic involved principles: Mass action principle, anatomical configuration principle (static or inverted localization, dynamic or reorganized congruent localization), plasticity of the ‘central’ neural mass. Psychophysical isomorphism.

V. TACTILE SCHEMES

1. BODY SCHEMA

Degrees of the body schema ..................................................698
Precedents about body schema. Degrees of the body schema (somatic schema, postural schema, spatial or praxis schema).
Somatic schema ................................................................. 700
1) Somatic development: somatic size and somatic configuration (reduced or constricted
model, embryonic model, filiform model). Emergence ways of the schema.

2) Praxic inervation: Abolition of the voluntary movement due to lack of schema (impos-
sibility of inserting the action in the own body). Innervation tests in an initial schema
(latency, frustrated movements, under-estimation of time).

3) Muscular reinforcement, its implantation and activation of the body schema: sponta-
eous occurrence of muscular reinforcement in the tactile system. Automatic or quasi-reflex
implantation, diffused implantation in the trunk. Body activation according to reinforce-
ment intensity, and ways to distribute the reinforcement.

4) Theoretical summary (somatic or tactile field, expansion and development of the body
schema). Asomatognosia.

Postural schema ............................................................... 714
Meaning of posture in body schema. Types of postures.
1) Tactile localization of an stimulus in relation to the postural schema: Localization levels
(in relation to postural schema and to somatic schema), diffused activation of postural
schema by cutaneous stimulus.

2) Body posture: Deviation or distortion of the somatic schema. Posture as spatial local-
ization. Another cases of pathological distortion of postural schema.

3) Segmentary postures: Pathological degradation and neutral or habitual posture ( neu-
tral posture deviation). Postural illusion, various tests.

4) Theory about posture: Precedents on plastic schema and sensory influences, posture
as localization. Neutral deviation in the normal case and transition to pathological case,
reduction of body dimension (constriction of the schema).

Praxis schema ................................................................. 729
Justification of the praxis schema.

1) Dyspraxia in the rudimentary schema.
a) Asomatognosia and motor action: phantom body, automatic action and anosognosia.
Another pathological cases.

b) Praxic inversion: autotopagnosia due to inversion and pseudo-active movement. Con-
tralateral canalization of the impulse reaction to obey the order to select fingers (special
features of the phenomenon).

2) Dyspraxia in the coupling of the body with the exterior space.
a) Orientation and left-right actions: praxic spatial orientation and types of alteration.
Left-right differentiation in diverse states, specular writing, etc., general nature of the
disorder (tactile, visual, etc). Other pathological cases (order in praxis destruction). Praxic
neutralization.
b) Egocentric-allocentric orientation and action: egocentric prevalence (tests and diverse phenomena, allocentric change by means of reinforcement). Interpretation of the egocentrism by reference to the neutral or habitual posture. Vestibular orientation in pulsions of the body (contrary sensation of impulse by vestibular relationship with inverted schema). Conclusions (egocentric reduction, “spatial neutralization”, etc.).

3) Dyspraxia due to the instability of the body schema.
   a) Defect in the initial of motor planning: Praxic inervation, the Schn. case, brusque impulsion, etc.
   b) Defect in carrying out the action: Types of action according to its automatization. Various tests. Praxic discontinuity (fragmentation), effect of the reinforcement, reduction of praxic behaviour.

4) Theoretical summary.

Apractognosia series and functional growth ............... 768
   Apratognosic nosology and functional unity (apraxia and agnosia of the body schema, Gerstmann syndrome, etc.). Continuity of phenomena (continuity between sensory and gnosis functions). Functional growth or sensory field growth (the three types of schema as growth stages). Difference between cortical and subcortical lesions.

2. TACTILE SCHEMA IN HAND TOUCH

   Tactile object recognition ........................................ 780
   Body schema and tactile schema in hand touch:
   b) Object recognition: Reduction and stereognosic transformation (object - hand). Tactile asymbolia in the injured patients (M and Schn.). Phenomenology in habitual state (analytic and deductive stages) and change by means of reinforcement. Special tests (mixed objects, artificial tactile reduction, rapid tests, etc.). Conclusions.

   Critical review of tactile agnosia .................................. 796
   Precedents: Wernicke-Dejerine controversy. The so-called pure-cases, disproportion in the alteration. The “Funktionswandel”. The agnosic mind.
   Interpretation by the cerebral dynamics: Insufficiency of the “Funktionswandel” for interpreting the disproportion, explanation by the logarithmic dephasing (delay), the problem of dissociations, sensory dimensions or parameters. Gnosic fragmentation explained by dimension reduction in the schema, consequences.

Acknowledgements: I am very grateful for the continuous help and advice of Miguel A. Porras.