Nervous excitability dynamics in a multisensory syndrome and its similitude with a normal state. Scaling laws

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In the context of increased number of works published on multisensory and cross-modal effects, we review a cortical multisensory syndrome (called central syndrome) associated with a unilateral parieto-occipital lesion in a rather unspecific (or multisensory) zone of the cortex.

The patients with this syndrome suffered from bilateral and symmetric multisensory disorders dependent on the extent of nervous mass lost and the intensity of the stimulus. They also presented cross-modal effects. A key point is the similitude of this syndrome with a normal state, since this syndrome would be the result of a scale reduction in brain excitability. The first qualities lost when the nervous excitation diminishes are the most complex ones, following allometric laws proper of a dynamic system.

The inverted perception (visual, tactile, auditive) in this syndrome is compared to other cases of visual inversion reported in the literature. We focus on the capability of improving perception by intensifying the stimulus or by means of another type of stimulus (cross-modal), muscular effort being one of the most efficient and least known means. This capability is greater when nervous excitability deficit (lesion) is greater and when the primary stimulus is weaker. Thus, in a normal subject, this capability is much weaker although perceptible for functions with high excitability demand. We also review the proposed scheme of functional cortical gradients whereby the specificity of the cortex is distributed with a continuous variation leading to a brain dynamics model accounting for multisensory or cross-modal interactions. Perception data (including cross-modal effects) in this syndrome are fitted using Stevens’ power law which we relate to the allometric scaling power laws dependent on the active neural mass, which seem to be the laws governing many biological neural networks.

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I. INTRODUCTION

In the last few years there has been an explosion of interest in multisensory effects such as cross-modal interactions in integrative cerebral processes (see, e.g., [1-13] and of course the general books [14, 16] and their references). Multisensory interactions have been revealed by functional magnetic resonance imaging, positron emission tomography and analysis of blood oxygen level, which have suggested that longstanding notions of cortical organization need to be revised. It was determined that cross-modal interactions can affect activity in cortical regions traditionally regarded as “unimodal”. This occurs, for example, in the contribution of the visual cortex to tactile perception [1, 2, 17]. It was then suggested that multisensory interactions must be considered an inherent component of the functional brain organization (e.g., [2, 6, 18] and references therein).

In this context, we shall analyze here the multisensory syndrome characterized by Gonzalo [19, 21], originated from a unilateral parieto-occipital cortical lesion equidistant from the visual, tactile, and auditory projection areas (the middle of area 19, the anterior part of area 18 and the most posterior of area 39, in Brodmann terminology), as shown in Fig. 1 A. This syndrome was called central syndrome of the cortex to highlight the “central” zone of the lesion in comparison with the primary or projection areas (considered as “peripheral” or “marginal” in this context). The immediate repercussion of part of this research (e.g., [22-24]) is later addressed towards the development of cerebral processing models [25-32], and other directions (e.g., [33-35]).

Among 117 selected patients with cortical lesions, 35 of them presented central syndrome of various intensities. A comparison is made in that research [19, 21] between an acute central syndrome case (patient M) and a less acute one (patient T). The syndrome is characterized by a multisensory and symmetric affection where all the sensory functions are affected, and with symmetric bilaterality. In the visual system for example, in addition to other disorders, there is a bilateral and symmetric concentric reduction of the visual field (Fig. 1 A), with gradation of the involvement from the center to the pe-
FIG. 1: Position of cortical lesions and respective visual fields with their sensibility profile (involved zones are dark). A: central, B: paracentral, C: marginal or peripheral, syndromes. (Modified from Fig. (a) of [37] with permission of the MIT Press).

FIG. 2: Excitation threshold curves for electrical stimulation of the retina (cathode on eyelid) in the acute central syndrome case (patient M), in the less intense central syndrome case (patient T), and for a normal man. For M and T cases, curves for an inactive state (free of facilitation) and for a facilitated state by strong muscular effort. Electrical intensity (indirectly given by volts) versus time (given by microfarads) necessary to obtain minimum luminous sensation. (From Fig. 2 of [38] with permission of Elsevier).

The syndrome presents a functional depression, shown for example in the excitation threshold curves for electrical stimulation of the retina in Fig. 2 [19, 21], where the thresholds for the acute central syndrome case (patient M) are seen to be greater than those for the less intense central syndrome case (patient T), and the latter greater than for a normal man. The functional depression presents dynamic phenomena related to the dynamic conditions of the nervous excitability [19, 21]. In this chapter, we focus mainly on some aspects regarding visual and tactile systems although the involvement is general.

One of the dynamic phenomena is the functional decomposition of perception in the form of a dissociation or desynchronization of sensory qualities normally united in perception. The sensory qualities are lost according to their excitability demands, i.e., the higher functions (with higher excitability demand) are the first lost as the stimulus intensity diminishes (in a given patient) or as the magnitude of the lesion grows (in several patients). Thus, the first function lost is gnosia, then acuity, blue color, etc... In this decomposition, the direction function is manifested. For example, when the illumination of an upright white arrow diminishes, the perceived arrow is at first upright and well-defined, next more and more rotated in the frontal plane at the same time that of a smaller size and losing its form and colors in a well-defined physiological order [19]. These phenomena are treated, together with tactile and auditory inversion, in Section 2.

Among many other dissociation phenomena, the chromatic irradiation or “flat colors” disorder (colors as separate from objects) [36] was interpreted as a dissociation between the chromatic function and the spatial localization function due to their different excitability thresholds. In relation with orientation, the so-called orthogonal disorder, detected in patients M and T, can be considered a dissociation phenomenon in which objects are seen the same independently of their orientation. For example, texts can be read upright or upside-down without noting any difference. Another detected disorder in the most acute case M was the substitution of the halocentric spatial orientation by the egocentric one.

The continuous gradation found between the central syndrome and a syndrome associated to a projection path led Gonzalo [21] to propose a functional gradient scheme in which the specificity of the cortex is distributed with a continuous variation. This model highlights a functional continuity and unity of the cortex. The overlap of several functional specificities would account for multisensory or cross-modal interactions. Different syndromes can then be ordered according to the position and magnitude of the lesion. These ideas are the subject of Section 3.

A key point of the central syndrome is its similitude with a normal man. The sensory system maintains its organization but on a reduced scale of nervous excitability. This fact permits us to apply the allometric laws of dynamical systems that are subjected to a scale change, and to explain the different (allometric) loss of the different sensory functions. These points are exposed in Section 4.

Another dynamic phenomenon is the unusual capability to improve perception not only by increasing the stimulus intensity but also by facilitation by another stimulus of the same or different modality (cross-modal effect). The added stimulus provides an extra excitation that compensates the excitation deficit due to neural mass lost in the “central” lesion. This capability was found to be greater as the nervous excitability deficit (lesion) is greater and as the primary stimulus is weaker. Muscular effort was found to be particularly efficient at improving perception (see the facilitated cases in Fig. 2).

The available data on improvement of perception with
the intensity of the facilitating stimulus, and also with the primary stimulus, are fitted using Stevens’ power law, which in turn is shown to reflect the allometric scaling power laws dependent on the active mass (the biological neural network in this case). These issues are considered in detail in Sections 5 and 6.

It is also noticeable the capability of patients with central syndrome to iterative temporal summation. The slowness of the cerebral system in this syndrome makes the cerebral excitation to a short stimulus to decay slowly. If a second stimulus arrives before the first excitation has completely fallen down, excitations are summed up, making possible to reach the excitation threshold to produce a sensory perception, reducing by this way the pathological dissociation. A simple model has been proposed recently to explain these and others temporal aspects of this syndrome, as the shortening of the perceived duration of a given stimulus \[29, 32\], and will be the subject of further work.

The rich phenomenology observed in this syndrome reveals general aspects of brain dynamics, as the functional unity of the cortex and the continuity from lower to higher sensory functions. The syndrome and its interpretation offer a framework to understand, at a functional level, aspects of the integrative cerebral process on a physiological basis.

II. INVERTED PERCEPTION DISORDER

The functional depression referred to in the previous section is richly illustrated by the striking inverted or tilted perception disorder in central syndrome, associated to left or right unilateral “central” lesions \[19, 21\].

First, we shall describe this phenomenon in the visual system. Gonzalo \[19, 21\] reported on 25 patients with cortical lesions that presented clearly chronic manifestation of tilted or almost inverted vision under conditions of minimum stimulation (illumination): 13 cases between \(2^\circ\)–12\(^\circ\), six between 12\(^\circ\)–30\(^\circ\), and six between 30\(^\circ\)–160\(^\circ\), all in the frontal plane, and a few cases also with small tilt in the sagittal plane. There were 12 with brain injury in the parieto-occipital region, which can be considered as “pure” central syndrome cases.

In the acute central syndrome of case M in an inactive state (free of sensory facilitation), the perception of a vertical upright white arrow suffered from the following dissociation phenomena as the cerebral excitation was diminished. During high enough illumination, the perception of the arrow was upright, well defined and with a slight green tinge. As lighting was reduced, the arrow was perceived as more and more rotated in the frontal plane [see Fig. 3(a)], at the same time it became smaller and with less defined shape and color, following a well-defined physiological order. The first function lost was the meaning of the object, then, blue color, visual acuity, yellow color, red color, luminosity... If the tilt was between 90\(^\circ\) and 180\(^\circ\), the object was perceived as a small shadow. The tilt was measured by rotating the arrow in the opposite direction until it was seen upright. For a test object situated in one side of the visual field, the object was seen to rotate with centripetal deviation, coming to rest inverted and constricted in contralateral position quite close to the center of the visual field, as shown in Fig. 3(b). The more peripheral the vision, the more tilted the arrow was perceived to be. In central vision, the rotation was clockwise for the right eye and counterclockwise for the left eye. In peripheral vision, the rotation was clockwise (counterclockwise) in the right (left) side of the visual field.

The perceived turn was dependent on the size and distance of the objects, i.e., on the subtended angle of vision, and also on the illumination and exposure time. Thus, an object appeared to be more tilted if it was only seen for an instant. As no inclination was perceived in clearly distinguished (well illuminated) objects, many patients were unaware of their anomalies, which were only relevant when provoked in an inactive state and under low intensity of the stimulus.

In the acute case M, the maximum inclination perceived was about 170\(^\circ\) with the left eye and 145\(^\circ\) with the right eye. In the less acute case T, the maximum inclination was only about 25\(^\circ\) with the right eye and 16\(^\circ\) with the left eye, following the same behavior as case M. The disorder was chronic in both cases. It was found in case T that two days after an epileptic attack the maximum inclination was 120\(^\circ\) with the right eye and 70\(^\circ\) with the left one. A slight tendency to rotation in the sagittal plane was also detected in case T and a few other cases. The reversal of vision was discovered in patient M when a moving object was seen by the patient with inverted direction of movement, and perceived as a mere blurred spot moving along a much smaller trajectory, and with an overestimated speed. For a moving object, the time that the object is seen in a place diminishes, and there-
fore the stimulus diminishes. The inversion process was reversible, i.e., the perception was improved by increasing the stimulus intensity or by multisensory facilitation, as explained in Sections 5 and 6.

Concerning the tactile system, it was found that for a mechanical pressure stimulus on one hand, five successive stages of dissociated perception were distinguished successively in case M as the energy of the stimulus was increased \[19,21\] (see Fig. 4 left part): 1, primitive tactile sensation without localization; 2, deviation to the middle with irradiation (spatial diffusion in a similar way to the chromatic irradiation in vision); 3, inversion phase but closer to the middle line of the body than the stimulus; 4, homolateral phase; 5, normal localization, which required intense stimulus, or moderate stimulus and facilitation by muscular effort, for example. The lowest sensory level stages (1, 2, 3) were separated only by very small increments of the stimulus intensity, while the highest stages (4, 5), close to normal localization, were separated by large increments, these being the most perturbed stages by the cerebral excitability deficit in the central syndrome.

When a cutaneous mobile stimulus was perceived in the inversion phase (Fig. 4), the perception was contralateral and close to the middle line of the body, with a very shortened trajectory (approximately 1/10 in case M) and almost inverted direction of movement within three autonomous zones of inversion: head, upper extremities and lower extremities.

Tactile inversion was studied for cutaneous and articular stimulation that includes processes such as walking. In moderate walking, the process showed striking characteristics: the first step was ignored, the second step was felt inverted, the third one as transversal, the fourth oblique, etc. This is a progressive recruitment in the direction of the perceived steps due to accumulation of excitation in the nervous centers by iterative action of steps due to the capability of iterative temporal summation. In slow walking, there was no summation and the direction remained inverted for each step, the steps being felt as very short.

It was found that the general laws for the tactile direction were the same as for the visual direction function. This led Gonzalo \[19,21\] to consider spatial inversion in a general way and as an essential fact in the organization of sensory functions with a spatial character, and to postulate a continuity between lower and higher sensory functions. Together with the turn, there is a diminution of sensorial intensity, space and time, dynamic parameters whose values depend on the active neural mass \[19,21\].

With respect to the auditory system, cases M and T presented also a dissociation phenomenon in analogous way as in vision and touch. For a particular sound, they perceived simple sonorousness if the stimulus is weak enough, and real tone if the stimulus is more intense. Contralateral localization due to spatial inversion of a sound stimulus occurred only in case M when the stimulus was weak and the patient was in an inactive state. The inverted perception always lacked tonal quality \[21\].

Other cases were reported by Gonzalo \[19,21\] that presented a sudden and transitory tilt or reversal of vision, mainly during epileptic auras. In this type of sudden turn, the visual scene was not as deteriorated as in the earlier case. He observed different degrees of tilted vision not only in cases with lesions on parieto-occipital region but on the occipital pole, and also far from the occipital area (e.g., a very anterior parieto-temporal region), showing that the anomaly occurs not only in central syndrome. He stated \[19\] that the disorder in the visual direction function is not an autonomous syndrome, but it is connected with the rest of visual functions; and could be present in cases with cerebral lesions in different locations, provided there exists some involvement of visual functions. Moreover, this disorder could be a rather common affection since the direction function is easily perturbed in different types of lesions, giving place, at least, to small inclinations of the visual image. Tilts in chronic disorder can be only evidenced under convenient exploration since a good illumination of the objects and sensory facilitation make the disorder go unnoticed for the patient very often. In the bibliography revised by Gonzalo up to 1950 he found about 27 cases with permanent disorder of tilted vision (in the sense as already described), and 45 cases which presented transitory turns during attacks. In general, in the bibliographic revisions made until recent years \[95,93,10\] most of the cases are transitory, without loss of shape and size of the perceived object or the visual scene, except in very few cases (e.g., some similar features in the degradation of the image are reported in \[11\]). Both permanent and transitory types are associated to a wide variety of cerebral lesions but with predominance of the parieto-occipital or parieto-occipital-temporal regions, and only very few cases (two or three cases) are associated to the frontal region. There are also some cerebellar cases, apart from other etiologies such as multiple sclerosis and epilepsy.

Concerning the rotation plane, in almost all cases reported in the literature and in those described by Gonzalo, the rotation of the visual image was in the frontal
plane. In one of the patients of River et al. \[42\] the sense of the rotation of the visual scene was specified to be counterclockwise for the left visual field, as described before. In general, few cases (e.g., \[42-45\]) presented rotation in the sagittal plane, and some of them presented in addition rotation in the frontal plane, as in case T. In a few other cases \[e.g., \[46\] (one case), \[47\] (four cases)], the image was rotated in the horizontal plane, leading to left-right visual inversion.

As for the orthogonal disorder in the central syndrome, in which objects upright and upside-down seem the same without noting any difference, a similar phenomenon was reported by Solms et al. \[39\].

To the best of our knowledge, tactile and auditory inversions as described here (with similar laws to those of visual inversion) have not yet been reported by other authors. This type of tactile inversion must be distinguished from the frequently described tactile allochiria (tactile alessthesia). In the latter, analogously to visual allochiria or alessthesia, tactile localization is contralateral to the tactile stimulus, in general symmetric (without centripetal deviation), and from the healthy side to the affected side \([48, 49]\) for example).

III. CORTICAL FUNCTIONAL GRADIENTS

The gradation found between the central syndrome and a syndrome of the projection paths; and between different central syndrome cases of various degrees, led Gonzalo to define two types of continuous functions through the cortex \[21, 38\], shown in Fig. 5. One type describes the specific sensory functional densities, of contralateral character, with a maximum value in the respective projection area and decreasing gradually towards a more “central” zone and beyond so that the final decline must reach other specific areas, including their primary zones. This is illustrated in Fig. 5 where the specific visual function density reaches all the tactile area until its primary zone. This type of function combines the factors of position and magnitude of a lesion. The more “central” the lesion, the greater the lesion must be to originate a specific anomaly of the same intensity. For a given position of the lesion, its magnitude determines the degree of functional depression. The other type of function has an unspecific (or multispecific) character, is maximum in the “central” region (where the decline of the earlier mentioned specific functions overlap) and vanishes towards the projection areas. It represents the multisensory effect in the anomalies and the bilaterality or interhemispheric effect by the action of the corpus callosum. Each point of the cortex is then characterized by a combination of specific contralateral action with unspecific “central” and bilateral action.

The central syndrome refers, as stated earlier, to lesions in the “central” zone, equidistant from the visual, tactile, and auditory projection paths. This syndrome exhibits both maximum multisensory and dynamic effects.

The concentric reduction of the visual field is called antiscotoma by contrast to the scotoma cases, as indicated in Fig. 5. Two central syndrome cases of different magnitude are shown in the lower part of this figure. Syndromes corresponding to lesions in the projection paths suffer from a functional suppression restricted to the contralateral half of the corresponding sensory system and scarcely present dynamic effects. For example, in the visual system there is a loss of the contralateral visual field, as was shown schematically in Fig. 1 C. Intermediate syndromes between these and central syndromes were called “paracentral” syndromes, bilateral involvement being asymmetric. In a visual paracentral syndrome, for example, there is an asymmetric concentric reduction of the visual field. Concerning the two tactile syndrome cases shown in the left part of the Fig. 5, only a very slight visual defect is present in the case where the magnitude of the lesion (in the tactile region in these cases) is high enough.

Fig. 6 shows more specifically the effect of the position and magnitude of the lesion in the visual field, and the corresponding visual gradient. It must be understood that, for the visual function to be normal, the action of the region with greatest visual sensory function density
is not enough, and the whole specific functional density in gradation through the cortex must be involved in the integrative cerebral process, leading to the normal sensory visual function, as shown schematically in Fig. 6 by means of the integration curve. It is noticeable, for example, the significant participation of the traditionally “extravisual” cortex in the maintenance of the visual field. The same applies to other senses and qualities. In a formal and abstract way, it can be said that the specific functional density is related to the derivative of the integration through the cortex (upper curve in Fig. 6), which justifies that the specific function was named gradient. The specific gradient would take into account the density of specific neurons through the cortex and their connections, representing the dynamic aspect of its anatomic basis. A sensory signal in a projection area would be only an inverted and constricted outline that must be magnified and reinverted, i.e., integrated over the whole region of the cortex where the corresponding specific sensory functional density is extended. Magnification would be due to the increase in recruited cerebral mass, and reinversion due to some effect of cerebral plasticity, following a spiral growth, as in Fig. 3 [19–21]. In the visual system, reinversion and bilateralization would occur in the 18 and 19 Brodmann areas, where the sensory representation is already reinverted. We must note that the specific with capacity for adaptation or learning. It is very small in animals, and even in other mammals, but it has a large extension in man.

In this framework, the central syndrome was interpreted as a deficit of cerebral integration due to a deficit of cerebral nervous excitation caused by the loss of a rather unspecific (or multi-specific) neural mass [19]. Thus, it can be considered as the result of a deficiancy nervous excitation of the cerebral system.

To conclude this section, we can say that, contrary to the rigid separation of regions (mosaic type), the functional gradients account for a functional continuity and physiological heterogeneity of the cortex, this one being subjected to a common principle of organization. The gradients scheme is an abstraction of the observed facts and offers a dynamic conception of quantitative localizations which permits an ordering and an interpretation of multiple phenomena and syndromes. A very similar gradients scheme was proposed by Goldberg [54]. The model described here is in close connection with the gradients found in the last years by means of neuroimage techniques [51–53], and with findings and proposals based on a distributed character of the cerebral processing and its adaptive aspects [54-59, 18]. It was suggested that traditional specific cortical domains are separated from one another by transitional multisensory zones and that multisensory interactions occur even in the primary sensory cortices [6, 8, 17]. The functional gradients model also accounts for multisensory interactions, which is a requirement formulated by several authors.

IV. SIMILITUDE AND ALLOMETRIC SCALING POWER LAWS

An essential feature of the central syndrome that makes it of special interest is its similitude with a normal case, since, as we show in the following, the syndrome can be interpreted as the result of a scale reduction in the cerebral nervous excitability with respect to a normal cerebral system.

The functional depression originated in the central syndrome is the result of a new dynamic equilibrium which maintains, nevertheless, the same cerebral organization as in a normal case. This can be appreciated, for example, in the hypoexcitability of the nervous centers in the excitability curves of Fig. 2 which, having the same shape, are shifted with respect to the normal case following the same law. The same occurs for the luminosity threshold curves [19, 21], and also for the concentric reduction of the visual fields (anti-scotoma) and their sensibility profiles, that maintain approximately the same shape as in a normal case but in a reduced size, as seen in Fig. 2. The same can be said for the visual acuity profiles and for the rest of sensory functions or qualities. In fact, the same type of electrical excitability threshold curves as those shown in Fig. 2 were found for the tactile system [19, 21]. The cases of central syndrome
with different degrees due to the different magnitude of the lesions, are then not only similar to a normal case but they are also similar between each case (see Fig. 7), being related between themselves by a scale change.

The concept of dynamic similitude, according to which different parts of a dynamical system change differently under a change in the size of the system, was then applied to the central syndrome. It is known that in the growth of a biological system, the sizes of two parts (say $x$ and $y$) of the system are approximately related by a scaling power law of the type

$$y = Ax^n,$$  
\(n\) being different for different parts ($y_1, y_2, \ldots$) of the system. These parts change then differently, i.e., allometrically \([10]\). This power relation means that the rates of growth of the two parts compared are proportional, i.e., \((1/y)(dy/dt) = n(1/x)(dx/dt)\) [as obtained from Eq. \([1]\) by taking the logarithm and differentiating]. These ideas were applied to the sensorial growth (or reduction), and an allometric variation of the different sensory qualities was then proposed.

In this regard, it is very remarkable the correlation found between the tilt of the perceived image (it could be variable $y$) and the width of the visual field (it could be variable $x$) in 24 cases studied by Gonzalo, with permanent tilted perception disorder, as shown in Fig. \(8\) \([19, 21]\). Among these 24 cases, 12 were considered as “pure” central syndromes of different intensity (those shown in the right part of Fig. \(8\)), and then a power allometric law of the type \([1]\) is expected.

Indeed, we show in Fig. \(9\) \(a\) the allometric power laws of type \([1]\) that fit to the data for these 12 cases. They relate the visual direction function (denoted by $y_1$) to the healthy visual field surface (denoted by $x$), and the acuity function (denoted by $y_2$) to the visual field $x$ \([38]\). In Fig. \(9\) the direction function is considered $0^\circ$ for the total inverted perception of the upright test arrow, and $180^\circ$ for the upright perception (normal), i.e., the maximum value is achieved in a normal integrative process from the inverted signal in the projection path. We consider normalized values with respect to the normal value, i.e., for a normal case $N$ the normalized values $x$, $y_1$ and $y_2$ are the unity, the maximum value. For case $M$, the corresponding values are very small since the non-normalized values are, $6$ degrees for the visual field width, $0.04$ for the acuity and $160^\circ$ for the perceived direction. These normalized values of $y_1$, $y_2$ and $x$ are used in Fig. \(9\) \(a\). The acute case $M$ (with considerable neural mass lost), the less intense case $T$ (with less neural mass lost than $M$) and a normal case $N$ are indicated. Errors are greater in cases with intense central syndrome because of their high sensory degradation and the high capability to facilitate by temporal and multisensory summation. Since $y_1$ and $y_2$ are $1$ for $x = 1$, the value $A$ of Eq. \([1]\) is $1$ and the approximate allometric power laws we found are $y_1 = x^{0.5}$ for the direction function and $y_2 = x^{0.1}$ for the acuity function. If the widths of the visual fields are considered instead of the surface values $x$, there is no evidence of power laws.

In order to better appreciate the allometric loss of the sensory functions in the pathological disgregation of the sensorium in central syndrome, we show the loss of direction function $(1 - y_1)$ and the loss of visual acuity $(1 - y_2)$ versus the loss of visual field $(1 - x)$ in Fig. \(9\) \(b\). A qualitative representation of the loss of an elementary function such as luminosity, and a higher one as gnosis, according to the observed facts, is included. The origin of the graphs corresponds to a normal man $(N)$. We can see that for a particular loss of visual field (due to a given central lesion), a split of the different qualities occurs so that the higher ones (e.g., gnosis) are loss in a higher degree than the lower (elementary) ones (e.g., luminosity). The order of the splitting corresponds to the order of complexity (directly related to the excitability demand) of the sensory functions and then to the order they are lost due to the shifts in their threshold excitabilities.

The most complex qualities, with the greatest nervous excitability (and integration) demand, become lost or delayed in greater degree than the most simple ones (with lower excitability demand). Sensations usually considered as elementary are then seen to be decomposed into several functions, one of them being the direction function, thus revealing up to a certain extent the organization of the sensorium. Very small differences in the excitations of different qualities already occur in the normal individual (in colors for example), and they grow considerably in central syndrome, as the allometric laws show. In normal individuals, there is already a significant dependence of the visual acuity with the luminous intensity, that is much more pronounced in a patient with central syndrome. In this context, it could be said that...
FIG. 8: Correlation between the perceived rotation of a vertical upright test arrow and the amplitude of the visual field, for 24 central (M and T are indicated) and paracentral (asymmetric) syndrome cases with permanent tilted perception disorder. On the right, the visual fields of the 12 pure syndrome cases are indicated with a small number, and the arrows indicate the rotation degree. Although the rotation sense of the arrow is opposite for left and right eyes, the same sense of rotation is drawn in the figure for a better comparison of the absolute values of rotation between the different cases. (Adapted from Fig. 17 of Suplemento II in Ref. [19] with permission of the Red Temática en Tecnologías de Computación Artificial/Natural and the University of Santiago de Compostela, Spain).

the cerebral system of the normal man works like an almost saturated cerebral system, in the sense that a very low stimulus induces cerebral excitation enough to perceive not only the simplest sensory functions but also the most complex ones in a synchronized way.

Concerning the tactile system for a central syndrome, the tactile qualities with the greatest demand of nervous excitation were the first lost as the intensity of the stimulus was diminished. For example, the first quality lost was temperature, then pain, and then pressure.

To illustrate the allometric behavior in the tactile system, Fig. 10 shows the narrowing of the tactile field in six cases studied by Gonzalo [19, 21], corresponding to different degrees of tactile paracentral cases (i.e., asymmetric central syndrome with predominance of tactile involvement in the contralateral side to the lesion). Analogously to the visual field, the so-called tactile anti-scotoma accounts for the progressive growth of the affection towards the periphery, a tendency to bilaterality, and shows dynamic effects. In Fig. 10 the most acute case is 1, and the less acute case is 6. The solid line corresponds to the limit of the perception of the most elementary quality: tactile pressure. In cases 2 and 3 (case 2 more acute than 3), the limits of perception of the different qualities are shown. The similitude of the shape of the limits (pressure and pain in these cases) is remarkable. Each quality is affected in a different degree according to its excitability demand, leading to the allometric dissociation between different qualities (see also the extremity of case F. in Fig. 10). As can be appreciated in the tactile paracentral cases I - IV shown in Fig. 11, the different qualities are lost in a constant order. The involvement decreases from case I to IV, the most intense affection being towards the periphery, indicated by arrows [19]. The sensation of cold is the first quality lost, i.e., the slightest disorder involves loosing cold sensation only, while the loss of pressure sensation implies the loss of other higher tactile qualities. The progressive distance between the limits of the lost qualities is shown in horizontal lines. The same order and relative distances are shown in the facial cases in the lower right of Fig. 11.

The maintenance of the shape of the different boundaries for the loss of different tactile qualities is analogous to the maintenance of the shape of the isopters in the visual fields of central syndromes, i.e., there is an isomorphism between involvements at different degrees, governed by the allometric laws. In summary, the allometric behavior (different exponent for each quality) accounts for the formal and quantitative description for the loss of qualities in the central syndrome, as result of its similitude to a normal cerebral system but on a reduced scale in the nervous excitability.

V. IMPROVING PERCEPTION BY MEANS OF INTERSENSORY OR CROSS-MODAL FACILITATION. POWER LAWS

Next, we analyze the dynamic effect which consists in improving perception through the facilitation of multi-
sensory interactions such as cross-modal effects.

Cross-modal effects were reviewed earlier by Hartmann [61] in a fundamental book where, among several references to different types of multisensory interactions, reference was made to the improvement of visual acuity in subjects with damaged brain [62] and in normals [63, 64] by auditory stimuli, as well as by other stimuli [65]. It is also remarkable the amount of work in the early thirties on intersensory relations in Soviet Union (reviewed by London [63]), and the extensive study of Gonzalo on multisensory interactions including cross-modal effects in visual, tactile, or auditory perception in subjects with lesions in the parieto-occipital cortex [19, 21]. In the last decade, cross-modal effects and multisensory integration has become a highly and increasing active research topic. For a rather recent review of cross-modal influences of sound and touch on visual perception, see, for example, Ref. [66] and references therein. More general reviews are in Refs. [14, 17].

By contrast, there are very few studies on the effects of motor system on perception except the research by Gonzalo [19, 21]. This author performed a wide and quantitative study on improvement of perception by muscular effort, as well as by other stimuli in other sensory modalities, in patients with central syndrome. In these patients, disorders such as tilted or inverted perception, concentric reduction of the visual field, loss of visual acuity, for example, were noticeably reduced by muscular effort or by means of other stimuli. This author also gave an interpretation to the injured brain Schon. case [67], who was able to recognize objects thanks to head movements.

We note in Fig. 2 that the curve for M in the facilitated state by muscular effort descends towards the curve for a normal man. In relation with this experience, it is of particular interest the quantitative equivalence found between stimuli as different as muscular effort and electrical excitation of retina, to produce minimum phosphene [19], as shown in Fig. 12 [68]. Each point in this figure represents the saved voltage \( V_s \) in electrical stimulation of retina to obtain minimum phosphene; e.g., the higher point in this curve (8 volts saved) means that 12 volts instead of 20 volts were needed when the patient M was holding a weight \( W = 80 \) kg (40 in each hand). The logarithmic dependence \( V_s \propto \log W \) means that a given increment in weight is much more efficient at saving voltage for smaller weights.

![FIG. 9: For central vision: (a) Normalized direction function (squares) and normalized visual acuity (circles) versus normalized untouched visual field surface \( x \) of the observing eye. Respective fittings (solid curves) with \( y_1 = x^{0.5} \) and \( y_2 = x^{0.1} \). Cases M, T and normal (N) are indicated. (b) Curves for the loss of direction function, \( 1 - y_1 \), and loss of visual acuity, \( 1 - y_2 \), versus loss of the visual field, \( 1 - x \), for the same conditions and normalizations as in (a). Qualitative indications for the loss of the higher sensory function gnosis and lower sensory function luminosity are shown as examples. (From Fig. 7 (a) and (b) of Ref. [38] with permission of Elsevier).](image)

![FIG. 10: Narrowing of the tactile field in 6 cases of tactile paracentral syndrome with different intensity. Solid line: limit to perceive a given pressure, from the most acute case 1 (very narrow tactile field) to the less acute case 6. Different limits for the loss of different tactile qualities (cold, heat, pain, pressure) for case 2, case 3, and case F (allometric dissociation). Darker parts are more affected (anti-s Scotoma). Similitude between the respective limits of pressure and pain in cases 2 and 3. (Adapted from Fig. 24 of Suplemento II in Ref. [14] with permission of the Red Temática en Tecnologías de Computación Artificial/Natural and the University of Santiago de Compostela, Spain).](image)
FIG. 11: Constant order in the loss of qualities: cold (4), heat (3), pain (2) and pressure (1), for the tactile paracentral cases I, II, III and IV (decreasing affection from I to IV). Limits for the loss of the qualities are indicated. The most intense affection is towards the periphery (indicated by arrows). The progressive distance between the limits is shown in horizontal lines for these four cases. The same order is shown in facial zone for two different cases. (Adapted from Fig. 25 of Supplements II in Ref. [19] with permission of the Red Temática en Tecnologías de Computación Artificial/Natural and the University of Santiago de Compostela, Spain).

FIG. 12: Saved voltage $V_s$ (volts) in electrical stimulation of retina versus static muscular effort made by holding weights $W$ (kg in log scale), to obtain minimum phosphene, for the acute central syndrome case M [10]. (From Fig. 1 of Ref. [68] with permission of Elsevier).

It was determined that muscular effort was one of the most efficient ways to improve the perception for any of the sensory systems. This study revealed that the greater the muscular innervation involved the greater facilitation was obtained. A remarkable general fact was that the improvement by cross-modal effects was greater as the primary stimulus to be perceived was weaker, and as the cerebral lesion was greater, i.e., as the deficit in the cerebral excitation was greater [19]. Thus, the conclusion for a normal individual was that facilitation cross-modal effects should be much more weaker or even negligible, and difficult to put in evidence.

By using available data from the patient M [19], we show in Fig. 13 some examples of improving perception versus the intensity of a facilitating stimulus, whereas the intensity of the primary stimulus was maintained at a constant low value. All data correspond to a stationary regime (the facilitating stimulus was acting until a stationary perception was reached). The well-known Stevens’ power law [69] that establishes a relationship between stimulus, $S$, and perception $P$, 

$$P = p S^m$$  \hspace{1cm} (2) 

(considered an improvement to the Fechner law) was used for fitting the data [38], the slope of the straight line in a log-log representation being the exponent $m$. Fig. 13 (a) shows, in log-log representation, the visual field amplitude of the right eye of patient M, versus facilitation by muscular effort when holding in his hands increasing weights [19, 21]. The data fit to straight lines with slopes 1/2 and 1/3 for the respective diameters, 1 cm and 0.5 cm, of the white circular test object. A greater test object implies greater stimulus, and we can see that it leads to a lower slope, i.e., to a lower effect of the facilitation according to what was said above. Figure 13 (b) shows data under similar conditions as in (a) but the sensory function measured is the recovery of the upright direction (180°) of an upright test white arrow that the patient perceived tilted or almost inverted (0°) under low illumination. The data fit to a power law of exponent $m = 1/4$. The novelty in Fig. 13 (c) is that facilitation is supplied by illuminating the left eye which is not observing the object, the power obtained from the fitting being $m = 1/8$ [38, 70].

There are also some cases reported in the literature [40] in which certain stimuli as eye closure, changes in body position, moving one hand, seeing a flame or holding on to a fix object, corrected and reinverted the vision [39] (one case), [44] (one case), [43] (three cases), [71] (one case), [72] (one case), [73] (one case), [42] (one case). Facilitation cross-modal effects have also been observed in patients with other visual deficits, e.g., [4, 12], and also in normals (e.g., [4, 7, 11, 74, 76] and general reviews [13, 16]).

The feature of central syndrome that the improvement of perception by cross-modal effects was greater as the primary stimulus to be perceived was weaker, is in relation to what is observed later by other authors [12, 77, 78]. Also, the fact this type of facilitation is greater as the cerebral lesion (excitability deficit) is greater, is in agreement with subsequent observations [7, 54, 76]. In a previous work [30], we suggested that the multisensory interaction contributes nonlinearly with the stimuli to the cerebral excitation, which is related to other suggestions [79].

Concerning the facilitating effect of muscular effort in normal subjects, a slight improvement of postural equilibrium by clenching fists was reported in Ref. [80]. Also,
we have recently observed [68, 81] improvement of visual Vernier acuity (capability to discerning misalignment between two segments) by moderate static muscular effort (males holding 14 kg and females 9 kg) in 10 tested normal subjects (age range 21–61). This acuity is called also hyperacuity because it is a higher function, then it easily degradable according to what said in the previous section. The maximum improvement was found in the subject of older age while for the youngest there was no conclusive improvement. This is in agreement with the assumption of some deficit of cerebral excitability in the oldest subject for which facilitating effects would be then more pronounced [74].

The improvement of perception by facilitation can be considered a sensory “growth” by means of an increase in the cerebral excitation which compensates in part for the neural mass lost in the central lesion, making the cerebral system to become more rapid and excitable [19–21].

This consideration is of special interest in relation to the following discussion on Stevens’ power law. First, we note that the validity of the Stevens’s power law is assumed to be restricted to limited ranges of stimuli, and it is not exempt from criticism. However, it is remarkable that many biological observable quantities are statistically seen to scale with the mass $M$ of the organism, according to power laws $Y = kM^r$, where most of the exponents $r$ are surprisingly found to be multiples of $1/4$ such as the metabolic rate ($r \approx 3/4$), lifespan ($r \approx 1/4$), growth rate ($r \approx -1/4$), height of trees ($r \approx 1/4$), cerebral gray matter ($r \approx 5/4$), etc. These scaling laws are supposed to arise from universal mechanisms in all biological systems as the optimization to regulate the activity of its subunits, as cells [82, 83] and references therein).

Under the assumption that a stimulus $S$ activates a neural mass $M_{neur} = \alpha S^\beta$ [70, 84], the power law of perception becomes $P = k(M_{neur})^m = k(M_{neur})^m$ with $r = m/\beta$, i.e., we recover a biological scaling power law of growth which would be the basis of the Stevens’ power law. Note that the exponent, and hence the slope, is independent of the units used and other proportionality factors. In cases where $\beta$ would be close to unity [84], then $r \approx m$, and Stevens’ law would exhibit quarter powers as seen in some of the cases analyzed here. Thus, the sensory growth by means of intersensory or cross-modal facilitation would be a particular case of the universal biological growth laws.

VI. IMPROVING PERCEPTION OF A STIMULUS BY INCREASING ITS INTENSITY. MORE POWER LAWS

As stated previously, the anomalies in central syndrome are clearly manifested under low stimulation, giving place to a decomposition or dissociation of the perception in which the functions are lost according to allometric laws (see section 4). Now, let us consider the improvement of perception in central syndrome by increasing the intensity of the stimulus to be perceived. We use available data from the central syndrome cases, which are exceptional examples of quantification of perception [19], and again we use Stevens’ power law given by Eq. (2) in the fitting to data [38, 85].

We show in Fig. 14 (a), in log-log representation, the visual field amplitude of right eye versus illumination intensity of a test white disk of 1 cm diameter. The exponents of the fitting are $m \approx 1/4$ for case M inactive (free of facilitation), $m \approx 1/4$ for M facilitated by constant muscular effort and $m \approx 1/8$ for the less acute case T. For visual acuity, we show in Fig. 14 (b) the data and the fitting curves for direct vision of the right eye in case M inactive, M facilitated, T inactive and a normal subject, versus light intensity of the stimulus. The respective power exponents are given.

However, the fitting corresponding to the lower data of visual acuity of case M facilitated is not good. Contrary to the improvement of perception versus facilitating stimulus, we found that improvement of perception with the intensity of the primary stimulus is well-described in some cases by two power laws, each one in the limits of very low and very high stimulus intensity. Although two branches can be also appreciated under other represen-
For the data of visual acuity of case M facilitated, a good fitting is obtained with the function given by Eq. (4), with exponent $m_1 = 6/4$ for low intensity and $m_2 = 1/4$ for high intensity, as shown in Fig. 15 (a).

The next example deals with the perception of a vertical upright white arrow by the right eye of the patient M in direct vision and for an inactive state. The tilt was measured by rotating the arrow in the opposite direction until it was seen upright ($180^\circ$). The direction perceived by M as a function of the light intensity that illuminated the arrow is shown in Fig. 15 (b). For the case of M inactive, the fitting with Eq. (3) gives $m_1 = 1$ for low light intensity and $m_2 = 1/8$ for high intensity. For M facilitated by muscular effort, a good fitting is possible with a single power law with slope $m = 1/8$.

In the tactile system, we first analyze the deviation of the perception of a punctual pressure stimulus on one hand towards the middle line of the body. As shown in Fig. 14 the perception in phase 2 is highly delocalized and centered at the middle line of the body (completely reduced tactile field). The perception in phase 3 is contralateral and at a certain distance from the middle line (a more developed tactile field). In phase 4, the perception becomes homolateral and still farther from the middle line, until it reaches the normal localization on the hand in phase 5. Measurements of the deviation versus
would suggest a similar neural network acting under high intensity of a facilitating stimulus (Fig. 13). This noticeable fact is that the exponents 1/4 and 1/8 also appear in the power laws that relate the perception to the intensity of a facilitating stimulus (Fig. 15), for high enough stimulus. An even more striking observation is that the perception of the tactile direction of 180° (pressure of the line stimulus measured in kg) is affected, as in inverted vision, that a tactile direction of 180° means a correct (restored) perception of the line direction, as a function of the intensity (weight in g). The experimental data and the fittings using a double-power-law are shown in Fig. 16 (a) for M inactive and M facilitated. The exponents \( m_1, m_2 \) are indicated.

Now, we consider the case in which the stimulus is a pressure on a 6 cm long line on one hand. In this situation, there is also the tilted perception of the orientation of the line stimulus, depending on the stimulus intensity (pressure of the line stimulus measured in kg). We consider, as in inverted vision, that a tactile direction of 180° means a correct (restored) perception of the line direction. The experimental data and the fittings with Eq. (3) are shown in Fig. 16 (b), where the exponents are indicated.

In general, it is noticeable that the exponents 1/4 and 1/8 appear equally in the examples for the tactile system (Fig. 16a) and for the visual system (Fig. 13b) (c) and Fig. 15, for high enough stimulus. An even more noticeable fact is that the exponents 1/4 and 1/8 also appear in the power laws that relate the perception to the intensity of a facilitating stimulus (Fig. 13). This would suggest a similar neural network acting under high stimulus intensity and under a facilitating stimulus in intensity of the stimulus were made with the arms separated from the body and perpendicular to the middle line. The data and the fitting using a double-power-law are shown in Fig. 16(a) for M inactive and M facilitated. The exponents \( m_1, m_2 \) are indicated.

We have analyzed in a current context the central syndrome characterized and interpreted by Gonzalo [19–21]. The first remarkable feature of this syndrome is the multisensory involvement with symmetric bilaterality, notwithstanding the unilateral lesion in a rather unspecific zone also referred to as the associative zone. The proposed model of functional cortical gradients, which is an abstraction based on observed facts, accounts for the great variety of different syndromes according to the position and magnitude of the lesion, and explains the acute multisensory disorder in the anomalies of the central syndrome. The model of gradients suggests a functional continuity and unity of the cortex, as well as an integrative process through the whole gradient extended over the cortex for a specific sensory function to be normal. Multisensory integration would be involved in a greater degree in regions where the specific functional gradients overlap. This scheme responds to requirements formulated recently, and to experimental findings in the last few years.

The central syndrome corresponds to a loss of neural mass in the region where the gradients overlap, producing a deficit of cerebral excitability. In the new equilibrium reached, the syndrome is considered to be a scale reduction of the cerebral system with respect to the normal one, exhibiting the same basic laws of nervous excitability but on a smaller scale. A qualitative and quantitative interpretation of a second relevant feature in the central syndrome: the dynamic decomposition or disgregation of perception into its components as the nervous excitation diminishes (lower intensity of stimulus or greater lesion). Higher, more complex functions, requiring greater nervous excitation, are lost before less complex functions, following a well-defined physiological order. In this disgregation, the perception function can be discerned from others. It can be lost in different degrees, giving place to the striking inverted perception disorder, which we have related to other cases reported in the literature. The corresponding tactile and auditory inversion disorders have not been described in the literature in the terms described in this research.

The progressive loss of different qualities as the neural mass lost is greater would be governed by allometric scaling power laws. They are indeed seen to account quite accurately for the continuous variation of a quality (e.g., visual direction, acuity, etc.) with the variation of another quality (e.g., visual field). These continuous variations are governed by power laws with different power exponent for each quality. This quantification

VII. CONCLUSION

FIG. 16: In the log-log representation: (a) Distance in cm from the middle line of the body to the localization of perception, as a function of the intensity (weight in g) of a punctual stimulus on the hand of patient M, in inactive and facilitated states. (b) Direction perceived of the line versus the pressure intensity (weight in g) on a line 6 cm long on one hand of patient M, in inactive and facilitated states.
of the splitting of the perception in different qualities has not yet been reported aside from this research, and stresses a functional unity and continuity between the sensory functions (lower and higher) according to their excitability demands.

The functional unity of the cortex is also manifested in a third remarkable dynamic feature in the central syndrome: the capability to improve perception by means of another stimulus in the same or different (cross-modal) sensory modality. This secondary stimulus supplies an extra excitation that compensates in part the excitability deficit. The permeability to cross-modal effects increases as the excitability deficit is higher, e.g., as the lesion is greater, and as the primary stimulus is weaker. Similar statements can be found in more recent studies on the highly active field of multisensory and cross-modal effects. This capability is then very low in normals, though perceptible in some high functions under threshold stimulus. Facilitation by muscular effort, scarcely reported in perceptible in some high functions under threshold stimulus. Facilitation by muscular effort, scarcely reported in literature, is one of the most efficient ways to improve perception in the central syndrome. We have evidenced that moderate muscular effort is also efficient in normals to improve Vernier acuity. The growth of the sensory level as a function of the facilitating stimulus is found to follow approximately scaling power laws, which are supposed to emerge from the dynamics of biological neural networks.

In the same way, the strong dependence of perception on the intensity of the primary stimulus to be perceived is another pathway for sensory growth. We have found that in some situations, perception versus stimulus can be described by two successive power laws with different exponents. The exponent values for the range of high stimulus intensity are very similar for visual and tactile qualities, and also similar to those found for a facilitating stimulus. It would suggest that for high intensity of the stimulus, the involved neural network would be the same as for a facilitating stimulus, i.e., the unspecific or multisensory location-congruence rather than task-relevance. Neuroimage 26 (2005) 414-425.

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