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20 Pathological Experience
A Challenge for Transcendental Constitution Theory?

INTRODUCTION

Husserl's theory of transcendental constitution relies on the idea that the intentional subject – particularly the subject of motor acts orientated towards a goal – has within itself the resources needed to make sense of objects and, in general, of all salient aspects, all configurations and formations invested with meaning in his lifeworld (*Lebenswelt*). Already anticipating the naturalization of phenomenology as embodied cognition (Petitot 1999; Petit 2015), transcendental constitution places special emphasis on the role of kinaesthesia, the body's intimate feeling of doing, and in so doing, it makes of the kinaesthetic system the source of the meaning structures of the *Lebenswelt*, and the guide to its systematic description. From a narrowly naturalistic point of view one might object to this programme on the grounds that it remains dependent upon the transcendental idealism of the Cartesian tradition, to the extent that it seems to suppose that the subject of motor acts necessarily enjoys an optimal control of the use of its motor system, as if it were exempt from disabilities. Hence the objection: surely the programme of transcendental constitution simply makes of successful voluntary movement the rule for all of human experience without regard to the pathological experience? This despite the fact that the empirical approach of biomedical sciences shows how very precarious and limited our capabilities to make sense are using only the normal resources of the body.

[1] Translation by Christopher Macann.

In this contribution I am going to try and defuse just this kind of objection and, more precisely work out a defence against a possible critique of the Husserlian kinaesthetic theory of transcendental constitution, focusing on the example of Parkinson's disease as a selective impairment of the kinaesthetic system. My key idea is that the empirical approach cannot afford to completely eviscerate any normative dimension of the description of the physiological foundations of motivation and action without creating difficulties for itself in the attempt to move back up from the fundamental level of physiological systems and functions (and dysfunctions) towards the phenomenological description of normal and abnormal behaviour. This claim will be supported by scientific literature on Parkinson's, which will be taken up at two levels. 1) At the level of clinical diagnosis, I note that if the symptom differs from normal motor behaviour, it is not on account of some deviation from an average but rather of the deficient realization of an ideal norm: from which I infer that the statistical analysis upon which the diagnosis depends cannot replace, at least in clinical practice, the phenomenological description of lived experience to the extent that the latter is centred on intentionality. 2) At the level of etiological research, the mechanistic explanation seems to be engaged in an infinite regression, where the postulation of the existence of certain physiologically dysfunctional mechanisms cascades down towards multiply embedded, and always more elementary mechanisms, while postponing the integration of all mechanisms in a mutually compatible way in the unity of behaviour, whether normal or abnormal. Here again, the mechanisms assumed to explain anomalies implicitly refer to an ideal of normality that eludes any explanation by mechanisms.

Interpreting pathological experience is a challenge for phenomenology, to the extent that the latter seeks to retain this experience within the field of meaning, the normal world of perception and action, while medical statistics tend to classify it under the laws of chance, and biological research envisages it in the perspective of the chaos of elementary particles. My thesis is the following: that however reticent the empirical approach might be with regard to the admission of any kind of normativity, the concepts of Parkinson's syndrome and motor neurodegeneration cannot be understood without referring to the transcendental norm

of a kinaesthetic activity underlying the sense conferred by the subject to its lived experience.

My claim is that a satisfactory compatibility has to be found between phenomenology and the *practice* of biomedical science – if not its standard ideology, a physicalism averse to phenomenology. Thus, at the same time as the researcher officially has to do with level after level of blind, automatic subpersonal mechanisms, he or she cannot but entertain some tentative interpretation of the possible function of such mechanisms. In field parlance one might well say: 'Such and such mechanism is normally (or fails to be in case of anomaly) *for* such and such function – a function which is in turn *for* such and such component of behaviour, which in turn is *for* the living being to sustain its normal relationships in the *Lebenswelt*.' So the way the scientist thinks of the mechanisms as being *for* something ultimately refers us to normal human life and the functional norms that are part and parcel of it – including the norms of voluntary, free movement. Thus, the entire chain of references is ultimately anchored in the intentionality of the lifeworld – the phenomenologist's home ground.

A KINAESTHETIC SUBJECT?

If the return to the thinking subject as foundation of the sense of experience is a familiar theme in traditional transcendental philosophy, it might, on the other hand, seem hopeless to want to derive from so disembodied a principle as the thinking subject the sense of being of anything whatsoever. Could we credit Husserl with having found a way out of this dilemma by opting for an incarnate conception of the foundation of subjectivity? His theory of transcendental constitution would then be freed from the idealist premises of a constituting subject surveying the entire domain of sense from an unconditionally elevated position inconsistent with our existence as natural beings. Sense constitution would then become a process for which the responsible subjective instance would no longer be an 'I think' (*cogito*) but an 'I act', an 'I' activating the movements of its body. By transferring responsibility for sense constitution from a thinking subject to an acting subject, the requirement that there should be a subjective foundation for transcendental constitution would be met, without detriment to

naturalism. As an acting subject, the pole of a kinaesthetic system capable of feeling itself doing, this subjective activity would no longer be disassociated from the deployment by a living being of a sensorimotor apparatus. 'Constantly kinaesthetic', whether engaged in activating its body and so learning from its success or failure in moving its limbs, or remaining in a posture of rest, the principle governing motor activity would be granted an irreproachable anchorage in the body.

In this article I hope to show that transcendental idealism has effectively been surmounted through a kinaesthetic theory of constitution; or at least that whatever element of idealism cannot be eliminated from this theory remains compatible with a naturalistic approach. But if we think of the acting subject as the pole of a kinaesthetic system rooted in an organism, we still need to ask: will it normally be exposed to the vicissitudes of the sensorimotor system, in just the same way as the anatomical and psychophysiological functions are so exposed? Or will its status as a transcendental source confer upon the acting subject a mysterious exemption from the stigmatism of its living incarnation? Surely, the dysfunctioning of the sensorimotor apparatus, underlying the working of the kinaesthetic system, would disturb the constitution of the sense of anything encompassed by the horizon of the *Lebenswelt* if the sense in question resulted from the investment of a subjective activity into the kinaesthetic animation of a living body?

In reflections found in his manuscript materials on the phenomenological status of a sensory anomaly (Daltonism), Husserl only manages to save the unity of the *Lebenswelt* at the cost of tracing back the abnormal experience to a variation of normal visual experience. For him, abnormal experience necessarily derives its sense from normal experience, whether we are talking about the child, the animal, or even of madness. If one wants to explore the origin of sense, one always has to go back to what sense *is* for the normal subject. The constitutive privilege of normal experience, in turn, relies upon the fact that the ordinary resources of the kinaesthetic system of an incarnate subject (its intimate sense of 'moving itself' in voluntary action) suffice for the constitution of one's own body as a permanent formation in the experiential flux. One's own body is that which can be activated at will by the subject through constituting operations making use of its kinaesthetic

system: one hand touching another and so on. This kind of self-constitution of the own body appeals to a relation of absolute immediacy as between the subject of the voluntary intention and the corporeal movement. The sense of having or of being a body is born of *doing* something with one's body, actualizing a 'can do', whether innate or acquired.

But constitution theory faces a challenge here, since it needs to overcome its persistent idealism in order to be able to account for pathological experience. That this is a real challenge is indicated by a certain ambiguity in the transcendental status of kinaesthesia, acknowledged in the perplexities (quoted below) expressed by Husserl himself. It is the constituting role of kinaesthesia that anchors the subjective activity in the own body, even though this anchorage is not devoid of ambiguity: incarnation or naturalization? The ambiguity is due to the fact that kinaesthesia can be traced back to a phase of motivation which precedes voluntary action and consciousness, a phase in which desire is mingled with intention and volition:

> So there is no distinction to be understood between desire and will, no more than there is between will and acting in general. But let's not lose sight of the kinaesthesiae. As an active Ego – and as conscious I am always active, therefore continually affected – I am in a constant 'I move', I am 'kinetic'. It is the latter (or any originary sphere in the same perspective), which is the kinaesthesia (the problem being to determine whether kinaesthesia is foundational for all moving oneself – all subjective process).
>
> (Husserl 2006: 320)

Along the same lines, one is tempted to confer a dimension of intentionality upon the instinctual impulse of desire as preceding voluntary action, thereby finishing up with a questionable transfer of constitutive functions to the instinctive processes of the organism:

> What we should say is that the instinct effective in kinaesthesia leads right up to the constitution of the mastered system as the unity of one possible mode of access, the possibility of freely reproducing each posture … each kinaesthetic system being an instinctive connection which can be actualized for itself.
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> (Ibid.: 328)

The result is an unresolved tension between kinaesthetic automatism, that aspect of the motor system which is actually operative, and the subjective, *a priori* dimension of the intentional activity and voluntary action of the Ego:

> What then are these kinaesthesia in themselves and what relation is there between them and the leading acts of an I that persists right through them throughout their unfolding? Could it be that this way of understanding acts as leading kinaesthesia is not already misleading?
>
> (Husserl: D10, 60a)[2]

Between a complete immersion of the subject and the overseeing of the kinaesthetic flux by the latter, Husserl's hesitation is patent, as shown in the following quote:

> So what is the special affinity of these kinaesthetic processes to the I in its activity? … But is the 'I' something alongside its concrete acts in the actualization of life, and can the concrete acts be thought in any other way than as a process through which something is actively produced, something that could even have passively happened of itself, and, in the end, as a core that immediately and actively moves out from itself and can be activated with equal immediacy[?]
>
> (Husserl: D10, 62a)

In the very fact that certain kinaesthesiae are privileged as 'kinaesthesiae of the I' – which suggests that the subject surveys those kinaesthesiae from above, as it were – one notes the temptation to treat the constituting subject as self-constituting. The implied idea is that all sense giving has to originate unconditionally – and so un-motivationally – in the I itself. But if we think about the deterioration of the neurophysiological conditions of voluntary movement, we are forced to reconsider the hidden presuppositions of the theory of the kinaesthesiae of the Ego as a refuge for the self-constitution of the constituting subject. The slow movement (so-called bradykinesia) and loss of movement (akinesia) characteristic of Parkinson's disease attest to the relativity of voluntary movement with regard to an unstable equilibrium between activation and inhibition of the neuronal circuits responsible for the initiation

[2] Quoted with permission from the Husserl Archives Leuven.

of motor activity. This state of activation itself relates less to the auto-activity of the subject than to the final result of a series of inhibitions exerted by subcortical inhibitory interneurones upon the innervation of pyramidal neurones of the corticospinal pathways responsible for the motor command sent out to the muscles. In this condition movements are difficult to initiate because motor commands are interfered with by inhibitory internal signals. Since one cannot attribute the activation of the neuronal circuits to 'the subject', the question arises how the subject might uphold its claim to be originally constituting. In order to understand how the intentionality underlying the motor intention can actually come to grips with the motor organs, and through them with the environment, one has to recur to an endless circularity of motivational feedback loops, thereby reducing the constituting subject to redundancy. After all, surely an incarnate subject is an empirical subject, and as such remains quite incapable of upholding the claim to being the constituting instance with regard to the sense of whatever happens?

We should however be careful not to allow our understanding of the kinaesthetic theory of transcendental constitution to be dictated by an empirical model drawn from biological sciences, as if it were a matter of projecting the functioning of the motor system upon the effective behaviour of the individual. Even though it does make an important move in the direction of physiology, the kinaesthetic theory of transcendental constitution remains an explicitly phenomenological approach and, as such, one that is regulated by the norm of intentional directedness within the horizon of a relation of the subject to the world. The pathways opened up by kinaesthetic initiatives do not simply result from a clearing of the pathways of nerve conduction thanks to the alternation of muscular tension and relaxation as the organism departs from and regains its rest posture. From within the immanence of subjective experience, the only paths opened up are those that are continually cleared by a goal-orientated intentionality. Both form of percept and aim of action feature a *telos* whose ideal character is isolated from the vicissitudes of the organism's functioning. The ocular movements are orientated in such a way as to make it possible for the subject to focus on the object of interest in the most convenient way. A triviality in any other context, that a normally sighted person sees *better* than a visually impaired person is an observation worth making here.

Kinaesthetic activity cannot be reduced to the actual state of the body, whether moving or at rest. In the same way, a perceived thing cannot be reduced to its lateral presentation in the visual field. That side of a thing which is actually visible is inscribed in the intentional horizon of a continuous variety of its other aspects, anticipated in advance as possible modes of presentation, and as a function of a constant subjective-objective correlation between the field of vision and the postures and possible movements (felt from within) of the sensorial and motor organs of a subject who is both perceiving and acting. As a result, what is taken to be real acquires its sense for the subject from something that is not observable, if only because it is virtual and not actual, and comprehends all the other possible modes of presentation that might result from a change in the position of the body or of its members. The complete kinaesthetic system, including all its kinaesthetic initiatives and transitional rest postures, subjects to a transcendental schematization the sensorial material which, without the kinaesthesia in question, would be indistinguishable from a fugitive impression. It would have to figure as a sensory illusion, to the extent that the relevant impression would be withdrawn from the framework of an experience allowing for something that remains the same across the variety of its modes of presentation. So the idea is that it is the kinaesthetic system that accounts for the sense I have of seeing a thing with a rear side, and so on, since my sense of the rear side is the sense of something that I *would* experience if I moved in such-and-such ways.

That the physiological concept of kinaesthesia is integrated in the phenomenological description of subjective experience means that the world, as kinaesthetically constituted, is a fully intentional world, endowed with all the layers of sense that it can acquire for an inhabitant. As a result, the incarnation of the theory of constitution made possible by enlisting the kinaesthetic system safeguards some elements of transcendental idealism. For the sense of things is enfolded within the horizon of a world common to many subjects whose sensorial and motor equipment is taken to be variable from one to the other, proceeding as it does from an individual development and personal history, all this within limits which are also those of mutual understanding, and which include, amongst other things, those who are handicapped, not just from the

standpoint of sensory experience but also from that of motricity. From here it is also possible to reach up towards the dimension of the 'Spirit' of the constituted world, inasmuch as the sense of the configurations it displays is not limited to correlations between the effective functioning of the organs of perception and the actual bodily movements of the biological individual. For this world is common to all and so cannot be conceived except as intersubjective, and so cannot be understood from the incommensurable standpoints of subjects locked up in the solipsistic prisons of their own material body, whether these organisms are physiologically functional or not.

In other words, the transcendental idealism that persists in Husserl's thinking is less Cartesian than it is Augustinian. It is not the subject dominating empirical reality from a superior stance who is the constituting subject but rather the subject 'who rises all of one piece to the call of truth' (Augustine 2009: VIII, §9).

PHENOMENON AND SYMPTOM

When I am no longer able to reach out with my hand towards something, grasp it, take hold of and manipulate it as I please; when I can no longer go up to it and walk around it; when I have lost the tempo of fluidly linked movements and tire of trying to command each elementary segment separately; when tracing out the letter I am writing blocks any anticipation of the movement needed to pass on to the next letter; when I become unable to lift my foot off the ground without freezing, then, both locomotion and the displacement of members of my body lose the familiarity, fluidity, and elegance of a gesture. My inability to take part in the movements going on all around me, to intervene appropriately in a general conversation, alienate me from the world – a condition known as *akinetic mutism*. Depressed, apathetic, nothing motivates me any more. Immobile in my chair, the world, having ceased to be the locus of my intervention, ceases even to interest me as a spectacle. The effort needed to adjust my extraocular muscles with a view to looking at people and things directly demands an expenditure of energy I am unwilling to commit. My four limbs paralysed in a hypertonic contraction, my life remains fixed in a 'now' lacking any protentional horizon. Disarmed by the arrival of anything unknown, I anxiously hold on to an indefinitely repeated 'now'.

The contrast with phenomenology, the kingdom of sense, whose horizon takes in the *Lebenswelt*, could not be greater. Phenomena make sense at their own level: shapes, a face, a melody, a sentence, the aim of an action are all given to the perceiving and acting subject with a meaningfulness that is directly evident. On the other hand, the symptom is ambiguous even for the subject whose symptom is in question. 'What's happening to me?': loss of control over my hand, reluctance to extend my hand towards an object, difficulty in bringing my hand to my face, inability to change the constantly flexed position of my arm, an unsteady gait … The symptoms only make sense within the etiological perspective of the doctor, who explains what the patient says in terms of underlying causal mechanisms. A given set of symptoms may only make sense in the framework of a syndrome without any other unity to hold them together except that of characterizing the majority of observed cases. As an emphasis on the fuzziness of that condition, let's only remember that diagnosing Parkinson's disease, for example, can never be more selective than the detection of a 'Parkinsonian syndrome', covering similar behavioural traits whose pathologies may be quite different.

A paradoxical relation obtains between the subjective aspect of the description of experience given by the patient and the medical typology of the expression in terms of symptoms of underlying causes of dysfunction. Afar from the solitary drama of a self betrayed by itself in its frustrating, because ineffectual, efforts to make movements; the identification of symptoms adopts the detour of medical statistics on numerous patients selected on the basis of the usual diagnostic questionnaires. Husserl tells that our subjective certainty of the coexistence of the *Lebenswelt* and of our own body is *apodictic*. For it functions as the continuous background against which the coherence of appearances constantly gets re-established in the further course of experience, in spite of any locally encountered disruption. Cut off from this ground of certainty, the interpretation of symptoms across the detour of a medical hermeneutics moves in the sphere of the probable, relying on the fact that the body of subjects considered remains sufficiently representative with regard to the usual statistical criteria. But even the experts recognize that these routine thresholds of significance afford no guarantee for the possibility of reproducing the published results (Johnson 2013; Ioannidis 2005). A symptom only makes sense if it is rooted

in a multidimensional representational substrate-space whose dimensions have to be differentiated empirically – by comparing competing scales of measurement, reflecting the different aspects of the illness, and the possible approaches – but which also reflect the sociological spectrum of the institutions who promote these scales of measurement.[3]

After 200 years of diagnosis and fifty years of treating Parkinson's disease, the relevant nosological category has still not acquired the strict definition of a Fregean concept, without, for all that, collapsing into a Wittgensteinian family resemblance. Taken as an 'umbrella concept', its extension is stretched between regrouping and/or dividing tendencies and so raises the question: 'What is Parkinson's?' (Jenner 2013). The symptoms only have the approximate unity of a flexible whole distributed across time. Ranging over varying degrees of gravity and successive steps in the development of the disease, the traits in common between Parkinson's, Lewy body dementia and Alzheimer's disease argue in favour of the existence of a continuum of degenerative neuropathologies running from Parkinson's to Alzheimer's, without excluding the possibility of an accumulation of the pathologies with age. Not all cases present all the symptoms, even those most representative of the disease, if not of its gravity (the trembling at rest at the root of its description as *shaking palsy* or *paralysis agitans* by James Parkinson (1817)). While traditional clinical classifications talk of a disorder that is basically motor, further progress in research now tends to classify it with neuropsychiatry. The animal taken as a model (transgenetic mouse) does not exhibit the behavioural symptoms of the Parkinson's shown by humans. The usual remedy – doses of dopamine, a provisional palliative without protective implications for the neurones – does improve bradykinesia for a while, but ends up causing abnormal movements (*dyskinesia*) and hallucinations, and is unable to stop, or even slow down, the deterioration of neurones. Kinesitherapy, finally, relies on brain plasticity. But by the time the disease takes

[3] *NMSS*: Non-Motor Symptoms Scale in Parkinson's disease; *CISI-PD*: Clinical Impression of Severity Index for Parkinson's Disease; *SCOPA-M*: Scales for Outcomes in Parkinson's Disease – Motor; *UPDRS*: Unified Parkinson's Disease Rating Scale; *PDQ-39*: Parkinson's Disease Quality of Life Questionnaire, and so on.

hold, brain plasticity has perhaps already reached its limits. The late appearance of motor symptoms in the evolution of the disease might just be recording the exhaustion of the resources upon which neurones of parallel motor circuits of a brain deprived of dopamine afferents relied to draw the energy needed to compensate the deficit over the asymptomatic period of time.

In sum, the occurrence of a symptom diminishes subjects' capabilities to make sense of their own experiences. Nevertheless and even while recognizing the opacity it introduces into experience, the symptom, in its turn, retains an intermediary status between phenomenology and mechanism, one which indicates that in spite of everything it still belongs on the phenomenal plane. The contrast between the divergence of the paths of research on mechanisms responsible for cellular death and the integration of symptoms within the unity of behaviour attests to a gap in explanation, and makes it difficult to discern any smooth transition from given dysfunctions of systems to such and such pathological symptoms, implying a failure in the self-confidence of an objectivity cut off from subjective experience. Between phenomenology and symptomatology, the to-and-fro might well turn out in favour of the former, by making it possible to fill the gaps in our usual thinking about the phenomena due to a disregard for pathological experience, and so to open the way to an exhaustive and systematic phenomenology extended across the borders of normality. If one does not want to have to deal with an overly idealized constituting subject, the pathologies in question, together with their motor disorders, have to be reintegrated into the kinaesthetic theory as constitutional operators. But, how is this possible, given that the symptom does not disclose – as does the phenomenon – the sources of its own significance?

CROSSING THE PHENO-ONTIC FRONTIER

'My brain', Paul Ricoeur affirmed, 'cannot be fitted into my bodily experience. It's an object of science' (Ricoeur in Changeux and Ricoeur 1998: 64). In other words, a pheno-ontical frontier separates any description developed in the light of reflective evidence and the ontic domain, in this case, that of statistics bearing on populations of individuals and hypotheses on the underlying mechanisms.

Nevertheless, the explanatory narrow dependency of pathological experience on dysfunctional brain systems looks like a denial of that descriptive closure of normal experience, requiring that we try to cross one way or another this pheno-ontical frontier.

Kinaesthetic experience is the place where the physiological constraints of motivation and action are brought to conscious awareness – or rather to that horizon of feeling enveloping the attentional focus of consciousness. Ideally, an immanent description of the kinaesthetic experience would be called upon to accompany the process of motivation and realization of action in all its stages. But no one thinks of attributing to the intuition and reflection of the phenomenologist an imaginary power of insight into the underlying mechanisms. The pheno-ontic frontier is not a transparent window behind which the subject might be able to observe its own organism. And in any case, the reality of the mechanisms remains that of models under discussion in the present state of research, a condition far removed from anything like a visionary contemplation. Nevertheless, there should be an alternative to the confrontation between a Cartesianism of the subject shut up in the citadel of consciousness and an imperialism of neurosciences in search of subjective territory to take over.

But in following this route, the *a priori* character of the transcendental theory of kinaesthetic constitution will have to come to terms with certain *events* in the history of medicine: so many empirico-transcendental precipitates in the process of objectifying pathological experience. The description of shaking palsy by James Parkinson (1817), the discovery of proteinaceous inclusions in the nerve cells of patients by Franz Lewy (1912), the description of Parkinson's syndrome in accidentally intoxicated drug addicts (Langston *et al.* 1983), the discovery of a mutation of the gene α-synuclein associated with Parkinson phenotype in the genotype of an Italian family (Polymeropoulos *et al.* 1997), discoveries of this kind prolong the long shadow of Galileo, 'this revealing-concealing genius' whose intervention shattered the *Lebenswelt*, 'where the earth does not move', precipitating it onto a physico-geometrical space (Husserl 1976: §9). These are the phases of objectification of my ill-being in a world that has become too constrained, in the form of a clinical syndrome currently being explained by the dysfunctioning of mechanisms of the motor system, the causal outcome of the

neurodegeneration of a brain, itself no more than a society of cells in neuronic man.

The pathology of voluntary activity represented by Parkinson's motor disorders highlights the exceptional character of the normal functioning of living beings and the uniqueness of the emergence of a sense-giving activity at the very heart of a Nature, otherwise indifferent to sense. And this because it is the multiplicity of ways in which organisms can fail to function, the greater probability of disorder and nonsense over order, which is precisely what confers value upon sense. When drawn from the resources of the kinaesthetic system of the agent, transcendental constitution prolongs 'the effort after meaning' (Barlow 1985), from cellular metabolism to behaviour. Moving in the reverse direction, complementary to that of constitution, let us embark upon the limitless descent towards those levels of organization (and risk factors of disorganization) which make it possible to envisage a constitutive role for kinaesthesia. Given the impossibility of retracing all levels distinguished by neurosciences, I will make do with two complementary approaches, the integrative neurophysiology of the motor system and molecular and cellular biology – with regard to which it should be added that the causal connection is far from having been established between the molecular alterations in nerve cells, the dysfunctioning of the motor system and the behavioural disturbances (Caviness 2014).

The causes of Parkinson's disease are multiple and their impact can be either divergent or convergent. Exogenous causes: the intoxication of addicts by an adulterated drug; the exposure of agricultural workers to herbicides and pesticides; the exposure of the population at large to environmental pollutants; cerebral traumatism and so on. Endogenous causes: the mutation of certain genes predisposes the organism to produce proteins of an abnormal kind whose toxic accumulation in nerve cells induces an irreversible process of degeneration and death. The best identified causes only take account of the most infrequent cases (Polymeropoulos 1997; Gasser 2011). For the common cases of Parkinson's known as sporadic or idiopathic, the recognition of risk factors (to the extent they are plurigenetic) will depend on further progress in sequencing the coding sequences of genes, or even on sequencing the complete genome. Regarding these heterogeneous causes, a synergy of influences is probable, but has yet to be established. Similar processes can be found in distinct

pathologies (Lewy bodies in brain tissues of Parkinson's patients have their homologues in the senile amyloidal plaques found in Alzheimer's disease). What we know about the mechanisms of cell death does not explain the selective failure of the dopaminergic neurones of *substantia nigra*[4] in Parkinson's.

Classically, the brain pathways of voluntary motricity branch off on leaving the motor cortex in two ways: a pyramidal tract heading towards the spinal cord conveys motor commands to the muscles, and an extrapyramidal closed circuit goes through a series of subcortical nuclei before returning to the cortex. It is assumed that the extrapyramidal circuit controls the pyramidal activity by fostering the crossing of spinal cord inhibition by the motor command once made the selection of movement parameters. The disappearance of the regulatory function of *substantia nigra (pars compacta)* on the activity of the *basal ganglia*[5] brings with it an excessive inhibition of the *thalamus*,[6] reducing its normally reinforcing influence on motor cortex for the initiation of voluntary movements. This inhibition is brought about by an intermediary loop connecting the entry to (*putamen*)[7] and the exit from the basal ganglia (*internal globus pallidus*[8] and *substantia nigra pars reticulata*). There are two paths of projection to accomplish this task, one direct and excitatory and the other inhibitory and indirect (it runs across the *external globus pallidus* and the *subthalamic nucleus*).[9] From which it follows that the initiation of movements is less an originally auto-motor, positive phenomenon and more the result of equilibrium between excitatory and inhibitory pathways

[4] *Substantia nigra*: midbrain structure, its *pars compacta* contains dopaminergic neurons whose death shortens the supply of dopamine neurotransmitter, a cause of motor dysfunction that characterizes Parkinson's disease.

[5] *Basal ganglia*: subcortical nuclei at the basis of forebrain, the release of their continuous inhibitory influence on motor systems of the cortex makes possible the selection and switching of the motor programmes of behaviour.

[6] *Thalamus*: pair of central brain nuclei, a relay of sensory and motor signals between cortex and the body.

[7] *Putamen*: nucleus of basal ganglia with projections to *substantia nigra*, a regulator of movements and learning.

[8] *Globus pallidus*: nucleus of basal ganglia, regulator of voluntary movements through inhibition of thalamus.

[9] *Subthalamic nucleus*: nucleus of basal ganglia, a central pacemaker implied in tremor and control of impulse.

within a cerebral circuit too complex to remain functional for long (Delong 1990). A knowledge of the physiological constraints of motivation and action preparation is all the more welcome to the phenomenologist in that it calls his attention to the horizon of consciousness of acting, where voluntary activity meets passivity and inertia, where contrary tensions counterbalance each other until clearing the way of motor intention to the movement's target.

Focusing now on the cell level, its metabolism is regulated by a large number of interwoven circular causal loops. A disturbance in any one of these loops can be the micro-event triggering a catastrophic process of neurodegeneration leading to cellular death. As a result, the researcher is confronted with a Hitchcockian situation: a crime scenario that could be re-written almost indefinitely starting out with a number of different suspects. The cell's sources of energy are energetic molecules used for the brain functions. Their production in *mitochondria*,[10] intracellular organelles specialized in the process of respiration through oxido-reduction, generates toxic derivatives (calcium, free radical oxygen) that accumulating in the cells expose them to oxidative stress. The mitochondria have to be carried by motor proteins along the cell's skeleton and from the axon right up to presynaptic terminals, locus of the greatest demand for energy. But free radicals damage these cytoskeletal proteins and disturb the transfer of mitochondria, resulting in an energetic crisis threatening the cell's survival (Mattson and Liu 2002)

The gene PARK-1 expresses *α-synuclein*:[11] a phosphorous protein of helicoidal structure abundant in presynaptic terminals, where it performs a regulatory function on the secretion of neurotransmitters by dumping (*exocytosis*) synaptic vesicles into the synapse. The overproduction, or the mutation, of PARK-1 results in an alteration of spatial structure (aggregation or fibrillation) of the said protein in the form of Lewy bodies (Polymeropoulos 1997) and their inclusion as insoluble formations into the fluid contents (*cytoplasm*) of neurones. Moreover, the presence of α-synuclein in presynaptic terminals favours the permeability of vesicles with a leakage of

[10] *Mitochondria*: intracellular organelles, powerplants of the metabolism of neurones.

[11] *α-synuclein*: neuronal protein regulator of dopamine supply and release. Parkinson's is a synucleinopathy.

dopamine in the cytoplasm, which is stressful for the neurone (Dauer and Przedborski 2003).

The infiltration of fibrillar α-synuclein of cytoplasm into the mitochondrial matrix (Hashimoto *et al.* 2003) blocks the chain of transport of electrons into mitochondria's internal membrane by inhibiting the transmembranal proteins, which normally get hydrogen ions to permeate the membrane in such a way as to re-establish the concentration in ions following upon the depolarization due to neuronal discharge. The resulting fall in electrical potential of the membrane reduces the excitability of the neurone (Mattson and Liu 2002; Dauer and Przedborski 2003).

The high excitability of the spontaneously active neurones of *substantia nigra* is maintained, thanks to the conductance (probability of being open) of ion calcium channels, transducers of membrane potential. The mitochondria control the level of intracellular calcium by interacting with organelles responsible for its secretion, the *endoplasmic reticulum*,[12] sequestrating this calcium. This regulation is under the control of other genes, whose mutations also pose a risk of Parkinson's, as they contribute to the accumulation of calcium. This dyshomeostasy of calcium results in the mitochondria losing energy (Reeves *et al.* 2014; Hirsch *et al.* 2013).

Free radicals, oxidative stress, dysfunctioning mitochondria all contribute to the aggregation of α-synuclein. The degradation of abnormal proteins through enzymes is ensured in barrel shaped organelles specialized in the elimination of waste material: the *proteasome*,[13] or in spherical organelles containing dissolving enzymes: the *lysosome*.[14] The dysfunctioning of this waste disposal system results in an accumulation of abnormal proteins in the cytoplasm, a vicious circle bringing with it neurodegeneration and cellular death (Dawson *et al.* 2010). Mimicking human society at large, doomed by its incapacity to eliminate or recycle the disposals

[12] *Endoplasmic reticulum*: network-like intracellular organelle important for the proteins properly folding and transport and the level of calcium regulation in the cell.

[13] *Proteasome*: intracellular complex protein dedicated to the enzymatic degradation of damaged cell proteins.

[14] *Lysosome*: enzyme containing vesicular intracellular organelle, another powerful waste disposal of the cell.

it cannot but generate, the biology of cell metabolism emphasizes the deeply contradictory character rather than the sustainability of the living being. With such a pit of nonsense overflowing inside, it is difficult to see how it could be – what in fact it is – capable of 'making sense of' any natural or cultural environment.

PHENOMENOLOGY VINDICATED

The scientistic policy of ignoring the subject's immanental point of view over his or her own kinaesthetic experience in acting and perceiving in favour of mechanical explanations that make no reference to subjectivity left the researcher with a loss of criteria of *normality*. That this threat is serious becomes clear just as soon as we review the indices of incompatibility between rival claims advanced for the disassociation of the experience of the voluntary agent, whether normal or pathological, each of which proposes underlying mechanisms for motor behaviour. To take simply the current controversy in physiopathology on the function of basal ganglia, their role is differently interpreted depending on the laboratories: sometimes as regulator of the balance of inhibition and reinforcement in the extrapyramidal circuit (Delong 1990); sometimes as selector of a motor programme (Kreitzer and Malenka 2008); sometimes as provider of an internal reward lack which would lead to anhedonism (Wise 1985); sometimes as energizer of action correlate of the 'strength of will' (Salamone *et al.* 2007); sometimes as synchronizer of neuronal oscillations in the motor system preventing a non selective synchronization responsible for trembling (Wichmann *et al.* 2011); sometimes as storekeeper clearing the way for new actions once learned actions have been stored in the repertories of long term memory (Yin and Knowlton 2006); sometimes as a bottleneck of channels of cortical information, a cause of increase in local potential due to their desegregation and linkage under the effect of compression (Brown 2007: 659); sometimes as cost/benefit estimator of the energy required by the movement, eventually resulting in a 'decision' of slowing down in bradykinesia (Mazzoni *et al.* 2007). And so on, and so forth.

This does not mean that it would be enough to simply pay attention to lived experience to be in a position to determine the functions of the basal ganglia. Rather, it means that the discovery of a functional

interpretation able to integrate the different models currently competing is likely to take more years of research. Alternatively our ideas of action and perception are simply condemned to remain without any determinate correlates at the cerebral level, inasmuch as the investigation of substructures of the isolated brain fails to provide any access to the holistic characteristics of the kinaesthetic interaction between an acting subject and the *Lebenswelt*.

From the foregoing, it appears that the functional normality of the totality of subsystems of a living creature definitely is a transcendental idea. There is no *a priori* necessity that the interaction between elements of the organism, reduced to its physico-chemical composition, suffices to keep movement going in the interval between hypokinesia and hyperkinesia, and so across the life-time of the individual. It might well be possible to postulate a law of evolution (still to be discovered (Berthoz and Petit 2014; Berthoz 2009)) according to which a living creature does not get drowned in the complexity of its all too numerous, and all too entangled, anatomical circuits and functions, in spite of the length, the slowness, and the intricate pathways of communication of information in the restricted space of the skull. But a rational reformulation of the teleology implicit in the normal functioning of living beings can best be worked out with reference to the idea of *kinaesthesia* enabling us to understand life in the light of the sense it brings to the world through its own activity.

To recapitulate: normal functionality is that mode of action of the kinaesthetic system of an organism allowing permanent things to appear in its environment, the organism in question being, for itself, an animate body among other such bodies, all these things – along with the un-thinglike things that other subjects are – belonging together within a common world. Such normality is not determined (or not exclusively) at the level of the functional mechanism's underlying behaviour. For, from a strictly mechanistic point of view, such 'normal' functioning has no reason to be preferred to malfunctioning. It is only from the point of view of the living creature itself, as perceiving and acting subject standing in relation to a lifeworld sufficiently stable to support a lastingly congruent experience, that one can talk about and define functional norms for biological mechanisms. Behaviour – as not reducible to its observable and measurable external aspects – has to be apprehended

originally in its lived dimension. Behaviour, which culminates in the field of the conscious experience of the subject in the domain of its practical intervention, remains the sole criterion as soon as one refuses to be satisfied with an apologetic teleology disguised as a law of natural evolution. With regard to the functionality of the mechanisms postulated in the explanation, lived experience, through its phenomenological description, remains the norm. For the latter raises to the level of explicit expression the *a priori* hidden in normal physiology.

So it would be naive, or at least premature, to claim that the knowledge acquired by research into the causes of motor neurodegeneration could not only supplement, but even supplant, the phenomenology of behaviour. Whatever the functional limitations due to the anomalies might be, as long as it is still possible to execute actions, a set of conditions are still available, whose reflective examination will bring to light the transcendental structures of the experience of the free and voluntary agent. No matter how revealing of the nature of human action it might be, the *dexterity* of movements in athletes or craftsmen (Bernstein 1996) simply exemplifies the optimal realization of a more fundamental structure of all living beings, therefore equally applicable to those suffering from deficient modalities. Acting, doing, being able, wanting, deciding, trying, realizing, and so on, all these concepts of action bring to verbal expression a *cogito* of the acting subject, which is not simply the pole of virtual reference of the semantic apparatus of discourse, no matter how under-determined it might appear to be from the physiological standpoint. To the extent that it covers the practical field of intentions, goals, and means of the agent, my voluntary act initiates a causal chain which is, in a certain sense, quite new, without this novelty standing in the way of an explanation in terms of underlying causal circuits. Trusting or not my ability to do, I aim at goals across kinaesthetic pathways made occasionally difficult by obstacles, or handicaps, or as prescribed by the internal articulation of the objects to be constituted. 'Not only is the whole world held within that horizon led by my kinaesthetic activities,' Husserl emphasizes, 'but even though I am actually hindered by paralysis or any physical restraint, that does not prevent a world of surrounding

things appearing, things which are in principle accessible to me' (Husserl: D3, 9b).[15]

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[15] Quoted with permission from the Husserl Archives Leuven. The ability to form motor intentions would be retained in cases of paralysis through spinal lesion: when imagining manual movements (touching its nose or its mouth) a tetraplegic patient can induce in its posterior parietal cortex patterns of neuronal activity coding the goal and the trajectory, thereby enabling it to realize the willed movement through robotic prostheses (see Aflalo *et al.* 2015).

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