Review paper

Is cerebral plasticity always a blessing?

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Abstract

Neuroplasticity is commonly acknowledged as a beneficial phenomenon. However, it should no longer be interpreted dogmatically, as the literature timidly discusses the phenomenon of negative plasticity. Taking up the interdisciplinary dialogue between the humanities and medicine, we recall the thought of French philosopher Catherine Malabou. She accused medicine of focusing only on the positive aspects of neuroplasticity. We therefore look at clinical examples of references to its negative dimension. The substantial advancements in neurosurgical techniques afford the removal of larger tumour volumes, resulting in prolonged patient survival rates, but the quality of life may not consistently result in satisfactory outcomes. Patients may undergo discernible alterations of their identities in a way apparent to both their loved ones and themselves. The neurosurgeon relies on neuroplasticity as a rehabilitative strategy, nevertheless its impact is not entirely predictable. We therefore encourage future discussion on the ethical implications of this phenomenon.

Introduction

Brain plasticity (neuroplasticity, cerebral plasticity) seems to be an intriguing notion at the intersection of philosophy and medicine. It is a "capacity of neurons and neural networks in the brain to change their connections and behaviour in response to new information, sensory stimulation, development, damage, or dysfunction" [1]. In the humanities of recent decades, it has come to life thanks to the philosophy of the French thinker, Catherine Malabou. She accused philosophy of lacking interest in the brain as an organ conditioning thinking and consistently replacing it with reflections on the mind and spirit: "Granted, it plays an important role for Descartes (in *The Passions of the Soul*) and Bergson (in *Matter and Memory*), but it remains a secondary organ that receives and transmits information without enjoying the slightest symbolic autonomy" [2].

Although the French philosopher emphasised the urgent need for a dialogue between brain sciences and philosophy, it seems that this dialogue has not yet been fully undertaken. It should be noted that attempts have already been made to assess both the limitations and possibilities of neuroplasticity as an intriguing concept – not only from a medical perspective, but also from a social one [3]. Steen N. Larsen's assessments included authors who referred to the possibilities hidden in cerebral plasticity [4, 5]. This sheds a slightly different light on the concept of cerebral plasticity, which Malabou seemed to monopolise in some of her statements.

In the present article, we are particularly interested in the scope of the concept of neuroplasticity and the way in which it is used in the humanities and medicine, both in a positive and negative sense.

Plasticity – sources of the philosophical concept

"I entered the neural world through the concept of plasticity – a philosophical concept that I discovered through Hegel, who talks about the role of plasticity in designating a system that transforms itself from the inside" – states Malabou indicating one of the main sources of rethinking her concept of plasticity. "Initially it wasn't scientific; it designated this ability to transform oneself from within, under the influence of education or experience. In the way you can integrate modifications that can come from outside, but that modify the inside – keeping the same structure but, at the same time, transforming it" [6].

As a counterpoint, a sociological thread can be cited here. Larsen, for example, showcases Adam Fergusson's (1723–1816) thought, as the Scottish philosopher "stressed the peculiarity and specificity of man: his open-mindedness, learnable and plastic characteristics, enabling the design and development of the most diverse societies" [3]. It is surprising, however, that Larsen places Ferguson and Malabou in the same register, calling their statements laudative hymns to plasticity [3]. What is undoubtedly a pure affirmation in Ferguson's case sounds a bit disturbing in Malabou's books – because she affirms what is destructive,

fatal, negative in plasticity. It would not be without reason to conclude that, in Malabou's essays, there is a perverse fascination with a phenomenon that could be included in the framework of the negative sublime.

Another author mentioned by Larsen, Martin Hollis (1938–1998), cannot be regarded as her ally. While Malabou changes the meaning of a concept commonly treated as positive, Hollis simply devalues the plastic man as someone who is determined by social structures or biological processes. He even notes the superiority of the self-conscious human being, who creates the social life, over the plastic man. Hollis therefore speaks, abstracted from medical cases, about plastic nature as man's questionable capacity for conformity [3].

It is worth emphasising that in Malabou's works, it is the subject that is the victim of plasticity – as if it were an alien force, even though it potentially sleeps in every human brain. The power of her rhetoric lies in the fact that the plastic brain becomes a threat to humans, which paradoxically may be an intriguing weapon in the fight against neuroreductionism. Walter Glannon is one of those who claim that our brains are not us [7] and interpreting Malabou's thought in this spirit, we could add: our brains' explosive power may turn against us. Human subjectivity is therefore threatened by the brain as the carrier of this unpredictable potentiality.

It is Malabou's reliance on empirical embodiments of negative plasticity in a destructive context that strikes the most, as she constructs her concept of plastic explosion and ontology of the accident. This is where Malabou's thought becomes disturbing for neuroscience. The beginning of her book *Ontology of the Accident. An Essay on Destructive Plasticity* is, among others, a literary journey to the Greek sources of metamorphosis depicted by Ovid. Here is Daphne on the run from Phoebus' unwanted advances, turned into a tree, thanks to which she saves her femininity from defilement. Although the reader of *Metamorphoses* laments Daphne's fate, the loss of her bodily self, Malabou argues, the heroine does not fall victim to the irreversibility that characterises the most harrowing cases of destructive plasticity. Daphne's metamorphosis is a form of salvation, whereas "the flight identity forged by destructive plasticity flees itself first and foremost" [2].

Thus, the story of the mythological Daphne plays a double role. Firstly, it clearly makes the reader aware of the drama of closing the awake brain in a body devoid of contact with the outside world. Secondly, it creates an imaginary space for a reverse drama: being void of subjectivity. This is how the philosopher herself comments on this state: "These phenomena of coldness and indifference are characteristic of destructive plasticity, of this power of change without redemption, without teleology, without any meaning other than strangeness. The new identities of neurological patients have one point in common: suffering to various degrees from attacks to the inductive sites of emotion, they show this often-unfathomable absence" [2].

Already at this point, we could ask if the phenomenon of indifference is really a product of cerebral mechanisms of negative plasticity or simply a consequence of injury or illness? Isn't cause and effect confused here? The second question, or rather an accusation, is the lack of respect for intermediate states, which Malabou seems to cross out by putting the Daphne's case on one side and patients whose negative transformation is associated with anosognosia on the other.

Following the lead suggested by Malabou, let us now ask about a situation when a disease forces a physician to undertake therapeutic activities, the unintended therapeutic effect of which may be a change in the patient's personality. Whether the "other identity" that appears then is the price to be paid for prolonging life is just one of the questions that a clinician and ethicist can ask themselves here. Another fundamental question is whether these mechanisms of brain plasticity, as the previous arguments suggest, have more than just a bright side.

Plasticity seen by neurology and neurosurgery

Vertosick in his book *Tales of Neurosurgery* uses the phrase "you ain't never the same when the air hits your brain" to describe the profound impact of brain operations, which can radically change a person's cognitive and emotional functioning. Brain surgery, like any surgical procedure, carries risks, although in the case of the brain, the fear of the unexpected is particularly strong. Malabou's use of the term *accident* is not necessarily an exaggeration for many neurosurgical procedures. Firstly, we should assume that the effects of treatment or disease must be distinguished from the effects of negative plasticity processes, since it is not always so obvious. The effects of an injury or surgery complication will appear in a short period of time, while the effects of neuroplasticity are observed in the long term. Therefore, the risky question should be considered whether negative plasticity is underestimated in neurosurgery.

Negative neuroplasticity we define here as a process of central nervous system regeneration, formation of new cells or new connections between them as well as cell internal reorganisation leading to unwanted function of the nervous system. Negative neuroplasticity may take course in the brain and spinal cord as well as in the peripheral nervous system, whilst the latter has the most potential to regenerate or regain motoric functions (but equally, they may form loops generating neuropathic pain). The spinal cord may also heal pathologically, leaving the patient with this debilitating suffering, which is a clear example of negative neuroplasticity. It can also be recognized as the cause of

spasticity, which manifests as overactive reflex arcs. In the brain the negative plasticity is typically connected to epilepsy, extrapyramidal motor dysfunctions, synaesthesia, or changes in higher brain functions, for example anxiety or even identity change.

The biological basis of identity is linked to the network of brain connections, called the connectome, which is a total brain network. We may also define subnetworks addressing particular brain functions, like for instance speech connectome. In *Connectome: How the Brain's Wiring Makes Us How We Are*, Sebastian Seung maintains that what is usually considered identity, is linked to the total network of the neurons in a nervous system [8]. If we assume that plasticity is more apt to occur in functional network reorganisation, than in structural changes in the brain like new cell growth, then we might conclude that connectomes can change both in positive and negative ways over time.

One of the few publications in which the term negative neuroplasticity appears concerns spinal cord injury (SCI). As Brown and Weaver emphasise, "the same neuroplasticity that allows locomotor function to recover also produces negative consequences such as pain and dysfunction of organs controlled by the autonomic nervous system […]. The literature supports the view that the negative consequences of neuroplasticity develop more commonly with therapies that directly stimulate nerve growth than they develop in the untreated injured cord. Compared to these conditions, neuroplasticity with negative outcomes is less prevalent after treatments that neutralise axon growth inhibitors, and least apparent after strategies that promote neuroprotection" [9]. The authors explicitly state that neuroplasticity underlies both neurological recovery and the neuropathology that develops after SCI. They call the latter *the dark side of neuroplasticity*.

This trail is also taken up by Johnston in relation to the unanimously praised plasticity of children's brain, which is divided by the author into the following types: "adaptive plasticity that enhances skill development or recovery from brain injury; impaired plasticity associated with cognitive impairment; excessive plasticity leading to maladaptive brain circuits; and plasticity that becomes the brain's Achilles' heel" [10]. Right from the outset, we must emphasise that some of the following examples from Johnston may invite objection, as they concern mostly the molecular and genetic processes of human development rather than plasticity in the adult brain. However, we are more interested in the presence and scope of negative plasticity in medical discourse than in its in-depth medical analysis. As defined by Johnston, it turns out that a broad group of paediatric neurological disorders could be understood in terms of their impact on fundamental mechanisms for brain plasticity. These include neurofibromatosis, tuberous sclerosis, Fragile X syndrome, other inherited forms of mental retardation, cretinism, Coffin-Lowry syndrome, lead poisoning, Rett syndrome, epilepsy, hypoxic-ischemic encephalopathy, and cerebral palsy" [10]. Therefore, Johnston introduces new terms into the conceptual map within the framework of maladaptive plasticity: after all, he talks about the Achilles' heel of the brain, which thus becomes somehow dramatised because it is vulnerable to injury [10].

Leaving ontogeny and the prenatal period, the other extreme needs to be discussed: neurodegenerative diseases. It is worth mentioning that Malabou interprets Alzheimer's and Parkinson's diseases as examples of destructive plasticity in medicine, and there are two other researchers who follow her trail in the context of these patients. According to Mogensen, their brains are plastic to the extreme: they are losing and reorganizing neurons but not in an organised way [11]. Therefore, Larsen ultimately accepts the term negative plasticity proposed by Malabou and sees its usefulness in medicine. Another example of the same phenomenon would be epilepsy, which can be understood as overactivation of the brain [3].

It seems that maladaptive plasticity sometimes also includes dystonia [12] and phantom pain after strokes. We can quote Takeuchi and Izumi's words here: "injury and excessive training drive neural plasticity in a maladaptive direction. This neural plasticity is called *maladaptive plasticity*, which contributes to the pathogenesis of phantom pain and dystonia. Moreover, several studies have reported maladaptive plasticity weakens motor function and limits motor recovery after stroke" [13].

Interestingly, the publications also distinguish between injury-induced and natural, experience-dependent plasticity. The interactions between them are complex, depending on the patient's age and other factors, but we still do not understand these interactions well enough [14]. In addition to the term injury-induced plasticity, surgery-induced plasticity for diffused low-grade glioma (DLGG) has recently been used, but the latter is only presented in bright colours [15, 16]. On our conceptual map we should still mark the term post-lesional plasticity and with it, an indication of a crosstalk between the brain and the tumour which also generates various modulations of plasticity [17]. Importantly, the publications also emphasise the dynamic interaction between brain metaplasticity and the disease itself [18, 19]. This issue seems to be of primary importance in view of the paradigm shift in neurosurgery observed for several years. One of the contemporary leaders of this field, Hugues Duffau, heralding the end of localizationism, refers to the extraordinary possibilities of neuroplasticity, which in the case of CNS tumours reorganises the patient's brain in a highly individualised way. Duffau titles one of the chapters of his book, intended for a wide audience, as "The Infinite Possibilities of the Brain" [20]. Already at this stage, it is worth noting that the message for the average reader is clear: the brain has

qualities such as 1. infinite possibilities consisting essentially in regeneration as part of neuroplasticity – and 2. high individuality, especially in terms of reorganisation of networks and connections.

Regardless of the state of our knowledge of the connectome, individual variability needs to be carefully respected, i.e. a complex and highly dynamic neural network. Due to this variation and intraoperative brain shifts, neurophysiological monitoring of the patient awakened during tumour removal surgery is a common practice [21, 22]. Moreover, radiotherapy and chemotherapy, either separately or in combination, have been identified as significant therapeutic alternatives for the treatment of brain tumours. Obviously, we should be aware of the risk of neurotoxicity, with radiation necrosis, chemotherapy-associated leukoencephalopathy, and cognitive deficits being the most frequent long-term side effects [15].

The changing face of modern neurosurgery is well exemplified by the DLGG treatment, described in an extensive multi-authored publication *Diffuse Low-Grade Gliomas in Adults*. This work "proposes new individualised strategies dealing with the interactions between the natural course of DLGG, reaction neuroplasticity, and onco-functional modulation induced by serial treatments […]. Although clinicians have to remember the classical *primum non nocere* principle, they also have to be aware about the fact that they can be harmful by inaction, i.e. by observing the tumour evolution while doing nothing: their role is first of all to be useful by treating the disease. For instance, although a complete surgical resection was not achievable during a first surgery due to the invasion of structures still critical for brain functions, such a total resection can become possible later owing to mechanisms of cerebral reshaping and/or following a shrinkage induced by adjuvant chemotherapy" [23].

Here we touch on the controversy which is the wait and see approach that was used in neurosurgery until recently and based on the assumption that it is better not to surgically mutilate the patient's brain. DLGG sooner or later turns into high-grade gliomas. Leaving the patient as long as possible without the intervention of a scalpel could be a strategy taken into account, if another alternative was, for example, a personality change. Here, the ethical aspect linked to the clinical decision needs to be emphasised, especially when the side effects of the therapy significantly reduce the patient's quality of life. A rather drastic question arises as to whether it is better to live a few years longer without feeling the joy of one's former passions in life but providing medicine with the satisfaction of a relatively longer period of survival. In the case of brain tumours such as glioma, this question should be formulated with the utmost ethical sensitivity and respect for the subjectivity of the patient – not only by the neurosurgeons but by the entire team of specialists involved in the therapy.

It is a *cliché* to say that medicine cannot thoughtlessly approach the issue of prolonging life, as the price for it may be a change in the patient's personality. In this case, the thinking of the French neurosurgeon (Duffau) and the philosophical concept (Malabou) intriguingly complement each other. According to the latter, it is the potential of neuroplasticity that testifies to individuality and subjectivity of a person. Value-based neurosurgery cares for this unique subjectivity no less than philosophy [24].

As we have already signalled, the current paradigm of some brain tumours like low-grade gliomas assumes that more tumour volume is removed surgically the longer the suspected life of the patient is achieved, on the condition the quality of life is not diminished [25] and instead of this the onco-functional balance is achieved [26]. Technical progress of neurosurgery has led us to the point of capability of removal of nearly all the tumour volume, but on its margins, there has been a high risk of motor system or speech network disruption. Nowadays the surgeon can remove the tumour to the safe margins avoiding for example motor dysfunction [27]. There are trends to even cross that border into more aggressive tumour resection resulting in slight patient's motor dysfunction that can be immediately rehabilitated in the first days after the surgery with means of physiotherapy and transcranial magnetic stimulation, relying on brain plasticity [23, 28].

Contrary to Malabou's negative rhetoric, medicine has ample evidence for the conscious control of neuroplasticity through methods such as physical activity (stimulating the production of brain-derived neurotrophic factor, which promotes the growth of new neurons and synapses), learning new skills, memory exercises, social interaction, creative engagement or pharmacological interventions (dopamine enhancers). Neuroplasticity can also be positively directed by virtual reality (VR) and brain–computer interfaces (BCIs) [29].

The healing ability of the brain to self-reorganise appears to be more evident in more basic brain functions, such as the motor system and speech networks. Less is known about the plasticity of higher brain functions, still underestimated in modern medicine. More emphasis is still put on the length of survival than on higher brain functions.

A two-fold tendency appears in the publications. On the one hand, the praise of brain plasticity, which neurosurgery seems to count on by removing as much tumour as possible even in eloquent areas, on the other hand, the introduction of the term surgery-induced plasticity. Neurosurgeons not only rely on the brain and its properties, but also become an external stimulus that forces the brain to adapt. From an ethical point of view, this is worthy of in-depth reflection in future publications. An important question is whether neuroplasticity can turn against a person changing someone's unique features, which are difficult to monitor

during surgery. Here we are addressing the underpublicised problem of the manipulation of human brain plasticity, whose greatest enemy is not so much ignorance itself as overconfidence in the certainty of currently available knowledge. Although we assume the good intentions of the surgeons, we do not know the exact consequences of their actions for patients. Brain surgeons should not remain silent about this.

Conclusions

The answer to the question posed in the title would be positive, provided that the unfortunate quantifier "always" is removed. Neuroplasticity is above all potential that allows us to overcome illnesses and disabilities, but its unpredictability presupposes the interference of chaos. Surely, it gives direction to Catherine Malabou's deliberations, but to summarise the selected neurological threads, one obvious thing should be noted. It appears that, at least sometimes – as in the case of SCI, omitting certain therapies results in fewer unforeseen changes than targeted clinical interventions. Therefore, the principle of *primum non nocere* would need to be rethought in such situations. Maladaptive plasticity is not sufficiently described in neurosurgery, which deals with the fragile home of the soul in a particularly invasive way.

The controversy surrounding Malabou's radical statements may also be related to the fact that she avoids shades of grey. Her examples are extremely dramatic which distances these considerations from the experiences of many doctors and patients. If we repeat Larsen's words that learning may be synonymous with neuroplasticity, we should ask whether learning is not sometimes an adaptation to limitations or acceptance of losses. Perhaps that is why we should abandon the idea of whether neuroplasticity can only be positive or negative. Change cannot always be assessed in this way.

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