ON DEFINING NEURAL PLASTICITY

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The term plasticity is in a precarious position. On the one hand it is a popular term, catchy and modern, and used with gay abandon in neurophysiology and neuropsychology. On the other hand its meaning is becoming so vague and its usage so often obfuscating that the discussant at a conference on plasticity and recovery of function could be " pleased that the word ' plasticity ' has occurred so rarely at the meeting " (10) and in a chapter on the definition of neural plasticity the writer could begin his summary by stating that, " there is a very good reason to avoid the term ' plasticity ' " (21).

Can some order be imposed on the term? I hope to show in this short note that the basis for a restricted definition of neural plasticity has long been available and that by applying the explicit and implicit criteria of the definition one may be able to re-establish the usefulness and scientific status of " neural plasticity ".

The origin of the term is the stem of the Latin word *plasticus* (that can be moulded) and basically it refers to something that is capable of being moulded, of receiving shape, of being brought to a definite form. An obvious scientific use of the term is in physics and engineering and it also has a natural application in biology in describing the same kinds of mechanical properties except with reference to organic rather than inorganic materials. For instance, before the fine structure of muscle contractile elements was understood, it was popular to refer to the plasticity of " actomyosin " filaments because, unlike the sarcolemma, they appeared to lengthen without exerting appreciable tension when the muscle was stretched

(see *e.g.* 8, p. 122; 20, p. 93). When found in this context, plasticity is often used in conjunction with the terms elasticity, distensibility and viscosity, with the following standard definitions (20): 1) *elasticity* is the tendency of a stressed material to return to the unstressed condition; 2) *distensibility* is the property of a material which permits extension, the greater the distensibility the less the elasticity; 3) *plasticity* is the lack of a tendency to return to the unstressed condition; that is, if a plastic material is deformed, it remains in this condition when the deforming stress is removed; 4) *viscosity* is the resistance to change of shape, an inherent property of some materials.

The figurative use of the term plasticity also has a long and respectable history in biology as in the following passage from the last of Sherrington's Silliman Lectures: "Mere experience can, however, apart from reason, mould nervous reactions in so far as they are plastic. The '*bahnung*' [facilitation] of a reflex exhibits this faculty in germ. In the humble spheres of nervous activity, such as alone fall within the scope of these lectures, simple sensori-motor experience seems to count for more than reason in the actual process of acquiring new motor co-ordinations" (19, p. 392). The use of the word plastic here is already somewhat removed from its application in the physical sciences since it is no longer a substance but a "reaction" which is being moulded. It is also clear that Sherrington had in mind something more than simply "capable of being moulded" in his use of the word since otherwise the sentence would reduce to the unilluminating tautology, "nervous reactions can be moulded in so far as they can be moulded", but he leaves the reader in ignorance as to whether he is using the term to refer to an abstract capacity or to a mechanism, to an explanation for an observed change or to an underlying physical process. In any case, the next sentences in the passage show that by plastic he was referring to a property connected intimately with, if not actually underlying, the capacity to learn.

If Sherrington was unclear as to the implications of the term plastic in reference to nervous organization, many modern writers have compounded the problem by being so unwilling to define or limit the use of the term that it may now be used to refer to virtually *any* long-lasting change in the nervous system which can somehow be attributed to the internal or external environment (*e.g.* see 13; for an attempt to categorize the different kinds of plasticity, see 21).

An exception to this tendency to enlarge the set of plastic phenomena is Konorski (16) who coined the expression neural plasticity but whose formulation of the term has largely been ignored. Konorski considered neural plasticity to be one of the two fundamental properties of the central nervous system in higher animals (the other important property being "reactivity"), and it was defined as the capacity of the nervous system to change its reactivity as the result of successive activations. In other words, neural plasticity consists of a process responsible for and identified with learning and memory, and by implication is an adaptive response to the constraints of the environment. The connection with the term in the physical sciences is not difficult to trace: A stress (neural activation) is repeatedly applied to a material (the nervous system) and as a consequence the material undergoes a lasting or permanent modification (presumably at the synapse). The only significant departure from the common usage in physics is that the stress has to be applied more than once to have an effect, suggesting an aspect of "malleability".

The definition will have to be updated to take into account certain developments which Konorski may not have anticipated in the late 1940's. For example, given the fact of almost continuous activity throughout much of the central nervous system most of the time, the "activation" which produces a change in reactivity will almost certainly turn out to consist of an interaction of particular temporal and spatial factors rather than simply the transmission of impulses from one neuron to the next. Second, along with successive activations, one would have to acknowledge that short-lasting decreases in the input to a cell, which one might call successive absences of activation and which would be due to inhibition or the lack of facilitation at an earlier stage of the neural circuit, can themselves carry information and may therefore play a role in changing the reactivity of the nervous system (*e.g.* see 15). Finally, in using the expression successive activations, Konorski undoubtedly had in mind Pavlovian conditioning, but the same neural modifications which occur with several pairings of stimuli and responses could also be responsible for the learning which is at or near asymptotic level after a single trial, a situation which has been useful in the study of consolidation of learning.

When the term neural plasticity is used in this limited way (which was probably also Sherrington's intention), some of the ambiguity in present-day usage can be resolved. Let us see some of

the consequences of adopting Konorski's definition in judging whether an observed change should be attributed to neural plasticity or not. There are two main classes of experimental conditions in which plasticity is supposed to play a role: 1) studies during development and maturation (adjustment to unusual rearing conditions or early injury to the central nervous system); and 2) studies during adulthood (adjustment to a change in the environment or to central nervous system damage late in life).

The first class of experimental conditions must be further divided since it is unlikely that the underlying neural mechanisms are the same in the cases of adjustment to the environment and in recovery from damage to the central nervous system. Concerning adjustments to the environment, one of the most interesting and celebrated examples of an apparent change in reactivity implying neural plasticity has been the biasing of receptive field characteristics of cells in the visual cortex of kittens which have been exposed to an unusual environment during early life (4, 12). Considered in the light of Konorski's use of the term, these effects would not be attributed to neural plasticity unless it could be demonstrated that they were primarily due to an active redefinition of the adequate stimulus for the cell and not just the selective maintenance of responses of cells which would in any case have been sensitive to the stimuli present during the restricted visual experience (the selectional hypothesis, see 12, p. 525). Konorski's limited use of the term neural plasticity would not encompass a change in reactivity by virtue of synaptic or neuronal degeneration due to the lack of appropriate stimulation any more than one would speak of an adaptive response in the case of a plant dying for lack of water. Of course, if it can be shown that the receptive field characteristics of cells which would normally *not* have responded to the stimulation present during the rearing period were able to modify their orientation sensitivity by up to 90° and thus acquire a new adequate stimulus, then one could argue that neural plasticity helped to bring about the final neural organization (see 18).

The effects of central nervous system damage in infancy are difficult to classify because it is still not known to what extent the infant nervous system has undifferentiated ("uncommitted") cortical and sub-cortical areas, and it is correspondingly difficult to devise a means for distinguishing between dynamic adjustments to damage and a sparing of function due to the presence of functional

alternatives which disappear or become suppressed in adulthood. A straightforward judgement can be made in some cases. For instance, sprouting of nervous elements into a deafferented area of nervous tissue appears to follow rules which are independent of neural activation and thus would fall outside the scope of plastic phenomena (11).

Successful adaptations to an unusual environment in the adult animal are mostly found in the sphere of adjustment to distorted sensory input or long-term perceptual negative aftereffects (see 7 for a possible electrophysiological analogue in the cat). Adjustments to a distorted sensori-motor loop would fit without difficulty within Konorski's concept of neural plasticity, both because they suggest an instance of adaptive learning in which errors (or perceptual mismatches, 14) are reduced through experience, and because they lend themselves to an interpretation in terms of a functional remapping of neural connections. Some neural changes, perhaps plastic in origin, have been seen in the organization of receptive fields of visual cortex cells in adult cats (*e.g.* 7 mentioned above), and I mention here a special case involving the effects of monocular paralytic strabismus because the initial results may have to be reinterpreted in the light of some new studies. Paralytic strabismus in the adult cat has been found to reduce the number of binocularly excitable cells in the visual cortex (5) and this effect was interpreted as a plastic change serving to reduce or eliminate double vision (6). A second change, seen in different animals, was a apparent shift in the position of receptive fields of binocularly driven cells such that single vision might be expected at a certain distance from the animal's head despite the divergent axis of the paralized eye (2, 6). The first effect, an exaggeration of monocular representation, has now been seen to occur very quickly following the eye immobilization (9) and to occur even if the animal does not experience double vision (17). Therefore the role of neural plasticity in bringing about the neural organization is called into question. The second effect, a shift in the position of receptive fields, appears to take much longer to develop and it is still possible that it depends on a plastic process.

Cases of recovery of function following central nervous system damage in adult subjects must be judged on their individual merits. There has been a tendency to use plasticity as a blanket term to refer to reorganization of the nervous system after injury regardless of the mechanism responsible (1, 3; but see also 22). It seems probable that once the temporary effects of such factors as diaschisis and

edema have been eliminated, the behavioral improvement in most cases will be seen as the substitution and refinement of different but adequate response strategies rather than the establishment of a new sensori-motor connectivity, and that in such cases neural plasticity will be seen to play a complementary rather than leading role in the recovery.

In this short note there has not been space to make more than a cursory examination of a few behavioral and neural modifications which are supposed to involve neural plasticity. It should be possible, however, by applying the criteria of Konorski's definition, to judge whether an observed change may or may not be attributed to neural plasticity. Certainly, one should refrain from using the term indiscriminately to refer to virtually any change in neural organization, and the definition provided by Konorski some 30 years ago can be used to create a distinct and useful category of phenomena and will, perhaps, restore the term to a position of scientific respectability in neurophysiology and neuropsychology.

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