Peer Commentary on "Neurobiology Relevant to Some Central Auditory Processing Disorders" By D. P. Phillips

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In this article, Phillips provides a comprehensive review of certain aspects of auditory neurobiology and their relationship to deficits of auditory processing. Toward the end of his article, Phillips discusses auditory processing deficits subsequent to cortical damage, concentrating in large part on word deafness. In my comments, I would like to focus on two issues relating to auditory processing in brain-damaged individuals.

The first concern Phillips' discussion of several categorical perception studies carried out with subjects who exhibited word deafness. Phillips states that patients with word deafness demonstrate deficits in temporal processing based on their inability, "in contrast to normal listeners...to discriminate consonant-vowel syllables on the same side of the phonetic boundary, but which differed in VOT by 20 milliseconds." It is the phrase "in contrast to normal listeners" which, although not quite inaccurate, is somewhat misleading. It is true that some studies have shown that under conditions of reduced uncertainty, *highly-practised* subjects are able to discriminate within category contrasts (e.g., Carney et al., 1977; Pisoni & Lazarus, 1974). Typically, normal subjects are not able to discriminate such contrasts (e.g., Liberman et al., 1961). In fact, the phenomenon of categorical perception is defined in part by the inability to discriminate stimuli which cannot be differentially identified (Studdert-Kennedy et al., 1970). Although word-deaf subjects may (and likely do) demonstrate deficits in temporal processing, the cited contrast with normal subjects may be less than compelling evidence for such an impairment.

The second issue I would like to discuss concerns some recent findings which may shed additional light on the nature of auditory processing in aphasia, a subject very briefly touched upon in Phillips' article. Citing research by Blumstein and colleagues (1977), Phillips contends that for some aphasic patients, "the strictly acoustic analysis of speech (and other) sounds may be relatively unimpaired, but their linguistic elaboration is not." Indeed, based on the categorical perception studies by Blumstein and others, this conclusion certainly seems valid. However, recent work by Divenyi and Robinson (1989) calls into question the assumption of intact auditory "non-linguistic" processing in aphasic patients. It is essential to determine the extent to which non-linguistic auditory processing impairments underlie or interact with phonetic and higher-level processing deficits.

Divenyi and Robinson (1989) assessed frequency discrimination, gap detection and discrimination, and frequency sweep discrimination (among other capabilities) in left hemisphere-damaged aphasic subjects, right hemisphere-damaged nonaphasic subjects, and normal control subjects. Their findings indicate that the discrimination of spectral changes over time is particularly vulnerable in left hemisphere brain damage. It should be pointed out that the auditory processing deficits found were by no means specific to left hemisphere damage; that is, patients with right hemisphere damage also exhibited impairments in auditory non-linguistic functions. The notably interesting result, however, is the (unexpected) low-level auditory processing deficit in left hemisphere-damaged patients. Divenyi and Robinson attempted to correlate the results of their psychophysical tests with results of auditory comprehension tests such as the Token Test (DeRenzi & Vignolo, 1962) to determine whether non-linguistic auditory processing abilities could predict linguistic auditory comprehension. Results indicated a moderate relationship, suggesting that non-linguistic auditory processing skills may play more of a role in language comprehension than previously thought. These results are clearly preliminary and require further substantiation; nevertheless, one is left with the suggestion that future research should be directed toward pinpointing the degree to which deficits in non-linguistic auditory processing contribute to or account for phonological and higher-level auditory comprehension impairments in brain-damaged individuals.

S.R.B.

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A reduced emphasis on temporal coding and an increased emphasis on rate coding seems to be one of the main hierarchical principles in central auditory system processing. The temporal code, in which the fine temporal structure of sound is coded by synchronization of neural activity to the phase of the sound and to that of its envelope, is dominating at the level of the auditory nerve and in the cochlear nucleus complex. In the pons and auditory midbrain, the temporal code becomes less dominant and a large diversity of average firing rate characteristics emerges on this level. This points to a more important role of the rate code, which carries information of fine temporal structure of sound by the average firing rate without a need for synchronization (Epping & Eggermont, 1986). At the level of the cortex, most of the temporal information processing seems to be replaced by rate coding. This has found its ultimate adaption in so called connectionist models of higher brain processing. In such models, used with surprisingly good results in the domain of language learning and processing (Rummelhart & McClelland, 1986), the result of the activity of a "neuron," a value related to its instantaneous firing rate, is passed on to other neurons and further elaborated upon. Such models have computational advantage above traditional (Artificial Intelligence) computer models because they are supposed to mimic the structure and action of the brain. Hence their computational units operate in parallel and have distributed memory properties.

In the excellent review on neurobiology relevant to central auditory processing disorders, Dr. Phillips refines the concept that information in the auditory cortex is largely handled as firing rate. We learn for instance that the temporal coding of interaural time differences used for sound localization is transformed (in brainstem and/or midbrain) into a firing rate code in a population of cortical neurons. Thus, interaural time differences less than about 0.5 ms are transformed into differences in firing rate. The acoustic structure of complex sounds, however, is still encoded in a temporal pattern of spike activity in a population of cortical neurons. Thus, firing rates modulated at intervals in the tenths to tens of ms range appears to be necessary to code speech-like sounds. Typically unmodulated sounds cease to elicit firing activity quite rapidly: there appears to be no place in the cortical representation for continuous and constant sounds, at least not in anaesthetized animals which provided most of these results. As a consequence, cortical neurons are not responsive to continuous masking noise but are able to code transients in noise with increase in firing rate. These observations then suggest that the auditory cortex acts like a bandpass filter for temporal modulation frequencies, much the same as the visual cortex does for spatial frequencies.

All this emphasizes that coding for sound localization is carried out completely differently from coding of other sound properties. Because, most likely, the same neurons are involved in the coding of localization (where) as well as type and intensity of sound (what), one aspect (where) can be coded in overall rate and the other (what) in a modulation of that rate. The review emphasizes the single neuron view of the brain: firing rates in individual elements code different aspects of sound. The overall firing rate distribution in a population of neurons may code the location of the sound source. In short, this is the view of the brain that the connectionists have adopted.

One of the major tasks of the auditory nervous system is to decide whether a particular spike train in an afferent neuron is caused by an external stimulus or must be attributed to spontaneous activity. In addition, intensity or envelope discrimination requires that the difference in afferent neural activity for the two intensities be evaluated as "real," or merely due to spontaneous fluctuations. There are several strategies that can be employed. The first is the evaluation of the number of spikes in a single neuron over a certain time period, followed by a comparison of this estimate with a stored "norm" of spontaneous activity for that particular neuron, and then, making a decision based on the probability that the spike number is above the norm. Because of the stochastic nature of the firings in neurons, this can only be a statistical evaluation and may require lengthy evaluation periods for increased accuracy. Some time can be gained when this evaluation is carried out for an ensemble of neurons: the population spontaneous rate will be more constant than that for single neurons, and the comparison can be made in shorter time and with greater precision. The problem of the storage of the "norm" remains,

however. This approach is implicitly assumed in optimal processor theories of auditory processing (e.g., Viemeister, 1983).

An alternative is to compare the detailed firing patterns of at least two neurons with the same CF: when both neurons fire at about the same time an external (e.g., a stimulus) or internal synchronizing mechanism is very likely to be at work. The more neurons are simultaneously compared, the more reliable the decision making process will be. Comparisons within a small ensemble of otherwise independently firing neurons may thus result in a fast and reliable indication of the presence of an external stimulus. Such a coincidence mechanism can detect the presence of an external signal, and can also enhance stimulus detection in noisy backgrounds as we have demonstrated previously (Eggermont & Epping, 1988). The number of synchronized spikes per second, then, may indicate the relative strength of the stimulus. Again, the cooperative effort of a number of nerve cells will enhance the speed and accuracy of this process and will extend the dynamic range of an output neuron if a certain range of threshold values is present for the input neurons (Eggermont, 1989).

Thus, certain synchronous activity patterns would be codes for various stimulus conditions because different combinations of neurons would synchronize depending on, for instance, characteristic frequency, localization, duration, signal-to-noise ratio, and best modulation frequency. This, incidentally, is also feasible as a mechanism for dreaming; due to some internal process, groups of auditory neurons will become synchronized, and this is experienced as (external) sound. This synchronization is also the likely mechanism for certain pathological phenomena such as tinnitus (Eggermont, 1984). In addition, one may propose that higher brain modules influence this synchronization under certain context dependent conditions as needed in phonemic and linguistic coding. These higher order modules may have an influence on the firing patterns of the "synchrony groups" in auditory cortex. Such influences are not unlike those of selective attention and cannot readily be distinguished from them. They allow a certain hierarchical structure in speech perception and provide a down stream influence in case certain higher processes become corrupted.

This, admittedly speculative, commentary suggests additional mechanisms that may be of importance in the understanding of central auditory processing disorders. Whether these mechanisms are to replace the firing-rate view of the brain or are merely a refinement or particular adaption of it remains to be seen. At least they might stimulate speculation and theory formation required for a better understanding of the biological basis of hearing disorders in general.

J.J.E.

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This lengthy and detailed paper represents a considerable amount of work on the part of the author. One of the positive aspects of this paper is that a highly respected auditory neurophysiologist has attempted to tie in and relate basic mechanisms to certain clinical manifestations of the central auditory nervous system. The attempt to do this is the type of writing that is sorely needed in our field and certainly is most difficult to do. It is also, I feel, the area where the most progress can probably be made; that is, in terms of combining clinical and basic information about the central auditory nervous system. Hence, Dr. Phillips is to be complimented on this noteworthy endeavour. This paper is also very well written and covers many of the neurophysiological aspects of the central auditory nervous system. The author's description of the endbulbs of Held and other types of neural connections was well featured. In addition, I would like to point out that the entire section on cortical neurophysiology is highly relevant and has many clinical correlates. The concept that sound pressure level of the signal is not what defines threshold of a cortical response, but rather it is a signal relevant to its background or the contrast of the signal to its background, is an important concept. This may very well relate to many of the problems clinicians see in auditory processing disorders. Even if it is not, it is thinking along the lines presented by this concept that I believe is most worthwhile for clinicians to read.

I am a little surprised that the author did not try to correlate more ABR information in terms of generator sites and possible clinical correlates to his discussion on brainstem physiology. The ABR is an extremely popular and relevant clinical assessment technique, and there are multiple places where correlations could be made to this technique. I would like to mention

a rather thought-provoking comment that is relevant to basic auditory neurophysiology as well as clinical auditory brainstem response assessment. That is, why is it that in lesions of one side of the brainstem that are in the mid to rostral pons and in some cases even higher, that the deficit on ABR is ipsilateral or bilateral and hardly ever contralateral. This is well-documented in the literature. Yet we know from the author's article as well as many others that most of the auditory fibers cross at the level of the superior olivary complex and certainly rostral to this structure, primarily is a contralateral system. Why then, do we not see ABRs for lesions of the upper pons that reflect deficits in the ear contralateral to the side of the lesion? This question is a common and very relevant one that ties in clinical as well as basic sciences approaches to understanding the central auditory nervous system.

The title of the article "Neurobiology Relevant to Some Central Auditory Processing Disorders" might lead one to believe that some of the neuropharmacology or neurochemistry of the auditory system would be included, which it really has not, at least to any great degree. The neurochemistry of the auditory system is one of the new frontiers in terms of understanding function. Specifically, the effects of the olivocohlear bundle on cochlear activity has gained considerable interest over the past five years. Since the olivocohlear bundle and certainly most of the efferent system is part of the central auditory nervous system, I am surprised that this was not included in this paper. Some early studies (Pickles & Comis, 1973) and more recent studies (Caspary, 1986) show how neurotransmitters and biochemical influences on the central auditory nervous system may affect auditory perception in noise as well as change the sensitivity of various brainstem neurons to acoustic stimuli. From the theoretical standpoint at least, these are extremely important findings which have strong clinical correlates, even though they are very preliminary. Discussion of this sort and also inclusions of more information about the neurochemistry, especially of the olivocochlear bundle, would seem to be a relevant and important part of a paper on central auditory processing and its related disorders.

F.M.

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Dr. Phillips has given us a very well written, comprehensive, and intriguing article on some of the current understanding of neurophysiological processing in the central auditory nervous system. I can only agree with the several premises and speculations made regarding the probable or possible processing mechanisms of the central auditory pathways. Moreover, Dr. Phillips should be commended for attempting to bridge the gap (sometimes all too vast) between the experiments of the basic sciences and the clinical domain.

Although a very technical paper in many respects, there are some general and specific clinical implications for speech-language pathology and audiology that can be made. We have known for some time, from the speech science literature, of the importance of timing factors in speech recognition and speech production. The early work in speech intelligibility pointed out that, while speech was highly resistant to many other forms of distortion, interference with timing typically was very detrimental to intelligibility. It is very interesting to read in Phillip's paper how precisely the auditory system encodes temporal patterns, and that there is reason to believe that the primary auditory cortex is, in fact, designed to respond to rapid temporal shifts of the kind characteristic of the acoustic patterns of speech. Further, this precision in transmitting the temporal aspects of complex patterns like speech can be seen at successive levels of the auditory system. I cannot but think that there is much work to do to develop diagnostic measures in speech-language pathology and audiology to better evaluate the integrity of this important aspect of auditory processing. In the area of hearing impairment in children, for example, we have only just begun to consider factors beyond the loss of acuity. Clinically we have known for some time that two hearing impaired children with the same audiogram will not necessarily have the same potential for developing speech and language (all other known factors being equal) or for benefitting from amplification. We are at a loss to explain the child whose audiogram says that they have good potential for speech and language development (again all other factors being normal) but who does not respond as expected to our aural habilitation. Are we missing some other critical aspect of hearing that may be disordered in these children? Phillips certainly gives us several good candidates for consideration in these cases. The important point is, of course, that if we could identify children with specific processing deficits beyond a loss of acuity alone, we could begin to try new strategies for management and do so at the earliest possible time.

This article also has implications for the area of central auditory disorders in children. I am referring to those children who do not have known or identifiable lesions in the central auditory pathways, but who exhibit certain behaviors and patterns of response on testing that have come to be associated with central auditory processing deficits or with a lack of maturation in the central auditory system. There has been

considerable controversy over this particular diagnostic group in both speech-language pathology and audiology. Even though research and theory in the neurosciences (like what is presented here in Phillips' article) and in speech perception has suggested that there is an acoustic level of analysis of speech (what Phillips calls a "sensory-analytic one") that takes place perhaps prior to or in parallel with other linguistic analyses, researchers and theorists in our disciplines have been reluctant to attribute deficits in speech and language processing to a central auditory disorder or dysfunction. In speech-language pathology, for example, it is more acceptable to attribute deficits, for example, in comprehension, to problems at a linguistic level of analysis. Usually this is done in the absence of any assessment of auditory processing or speech perception abilities.

One very real problem is the lack of reliable and valid diagnostic testing procedures both in the auditory perceptual and the linguistic domains. Nevertheless, if one adopts a framework that assumes that there are levels or specific aspects of processing (acoustic and linguistic) and proceeds to utilize whatever information can be obtained reliably (either from history, behavioral observation, or tests), it is possible to make a hypothesis regarding the nature of the deficits, to test that hypothesis against all available information, to formulate a program of management based of this hypothesis, and to measure the effectiveness of your approach (see Jerger, Martin, & Jerger, 1987; Jerger, Johnson, & Loiselle, 1988; Breedin, Martin, & Jerger, in press; Sloan, 1985,1986). In this way, we can test the validity of our diagnostic hypothesis against its clinical usefulness.

It is important for clinicians to consider these new theories of auditory processing and to consider how these ideas can apply to clinical populations, both patients with known lesions and those with suspected central auditory disorders. These new ideas about auditory processing give us a new window through which to view the client's behavior and another possible explanation for that behavior. With that, we can generate more ideas for possible management strategies.

In conclusion I would like to offer a few additional observations regarding children with suspected central auditory disorders. In reviewing the types of presenting complaints that have been presented to me over the years by parents, several are very interesting in light of Phillips' discussion of "auditory space" and the discussion of the sensitivity of cortical cells to short-term stimuli presented in a background of noise, the threshold of a cortical response defined as the "signal *re* background." I have frequently heard parents report that their child does not seem to hear at times or does not know when they are being called. In light of Phillips' discussion of how we might perceive auditory space, it is clear that we, as clinicians and diagnosticians, rarely consider this phenomenon. We certainly have a visual space in which objects have their location and their inter-relationships; all this by virtue of a complex visual perceptual system. We are familiar with cases of visual neglect and hemianopsia in adults with cerebral lesions. We seldom consider our auditory space in the same way, but in the case of some of these kinds of observations made by parents, it may be worthwhile to investigate this further. We might ask, do these children have an auditory perceptual space? Have they sorted their world into "auditory objects" distinct from one another? Are they neglecting part of their "auditory field"?

Finally, it is interesting to read in Phillips' discussion of the response characteristics of primary auditory neurons that there is evidence linking acoustic feature selectivity (necessary to speech discrimination) to signal saliency (ability to perceive signal in noise). Heretofore there has been no unifying principle or body of evidence to explain two of the most predominant clinical features of central auditory disorders, difficulties identifying/discriminating speech sounds and difficulties perceiving speech in noise. Robert Keith and others have simply referred to these two characteristics as two different abilities or processes. Such explanations have not been helpful to attempts to understand the basic nature of the central processing disorder.

Dr. Phillips has presented us with some very interesting hypotheses for our further study and consideration. There is much to look forward to as research expands in the area of central auditory processing.

C.S.

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Reply To Musiek, Eggermont, Baum, and Sloan

Most of us take hearing for granted. A sound occurs and we, quite simply, *hear* it. One of the implicit goals of my paper was to remind us that what we take as an automatic and practically instantaneous perceptual response is in fact mediated by countless actions among neurons numbering in the millions. My approach was to examine the physiological activities of central neurons at different levels of the ascending auditory system. It was further to point at how the nature of the neural representations of a sound built at those levels predispose them to various pathological processes and, by so doing, to provide a framework for understanding the nature of the perceptual sequelae that follow from pathology. In short, I tried to link the structure of sounds, the physiological mechanisms that shape the neural representation of those sounds, the perceptual dimensions that result from the representations, and the auditory-linguistic consequences attendant to pathology of the representations.

If this seems an ambitiously plenary goal, then Dr. Musiek reminds us of its incompleteness. First, my paper deals only with physiological processes in the brain, and Musiek reminds us that the physiological activities of neurons are themselves mediated by biochemical processes. The fact is that neurons differ in their neurochemistries, and this renders them differentially susceptible to metabolic disorders. The harsh reality of this principle can be seen in studies of hyperbilirubinemia in infants. This is an inherited metabolic disorder which results, *inter alia*, in a relatively selective accumulation of neurotoxic metabolic byproducts of haemoglobin in structures of the central auditory system. Only recently has a systematic investigation of the further neurobiology of this disorder been initiated (Brugge et al., 1987; Shapiro, 1988; Shapiro & Hecox, 1988; Perlman et al., 1983; Nakamura et al., 1985).

Second, while I emphasized the *ascending* auditory system, Dr. Musiek reminds us that there is also a *descending* auditory system. The study of this series of systematic connections between higher and lower centers has been ongoing for some time (Harrison & Howe, 1974). This area of research has taken on a special significance in recent years because it has become apparent that the cochlea's outer hair cells are able to influence basilar membrane motion and that the descending connections may, presumably through their actions on the outer hair cells, modify afferent auditory nerve fiber activity (Warren & Liberman, 1989; Dolan & Nutall, 1988; Rajan, 1988; Winslow & Sachs, 1987).

Finally, Musiek asks for a more detailed account of the neural basis of ABRs. Such an account would be a worthy endeavour, but, like the two issues above, was simply beyond the scope of a single article.

Dr. Eggermont raises a number of issues, and I have few disagreements with them. Because of this, I would like briefly to pursue a principle of brain function which we both directly or indirectly allude to: "grandmother" cells. There are many descriptions in the literature of sensory neurons responding (apparently) selectively to highly specialized stimulus configurations. While these descriptions can be both intriguing and instructive, they engender a view of brain function in which particular stimuli are encoded by equally particular neurons. This view should be taken with great caution. It is true that the central auditory pathway is tonotopically organized and, therefore, that the presence in a sound of a given spectral element is indicated by *which* neurons are excited (and not excited). Nevertheless, it is also true that the spike output of any given neuron is highly ambiguous. This is because that output is shaped by many factors: the frequency, amplitude, spatial location, and temporal position in a sequence of the relevant sound element (see also Phillips & Brugge, 1985). Eggermont and I are in agreement that a useful recourse from this dilemma is to appeal to the population response, which is much less ambiguous. The auditory system is built on a multiplicity of tonotopic representations: if there are neurons tuned to the same frequency, but to different sound amplitudes or locations, then any ambiguity inherent in any single neuron's response rate is resolved by the pattern of activity across these populations.

Dr. Baum raises two main points. One concerns the discrimination of voice onset times (VOTs) by both normal and word-deaf listeners. Baum is correct in that it probably takes a well-trained listener to discriminate CV syllables with VOTs on the same side of a phonetic boundary. It follows from this that any evaluation of word-deaf listeners in this respect should be qualified. The discrimination of CV syllables with VOTs on opposite sides of a phonetic boundary is, however, easier, and word-deaf patients are impaired in even this task if the VOTs distinguishing the syllables differ by only 20 milliseconds (Auerbach et al., 1982; see also Miceli, 1982). Accordingly, the point about temporal resolution in word-deaf patients as revealed in the VOT discrimination studies probably should stand, though we should heed Dr. Baum's remark that the perceptual tasks in these studies are not trivial.

Baum's second point concerns the recent observation that patients diagnosed as aphasic (but not word-deaf) might, if carefully tested, betray evidence of a nonlinguistic auditory deficit. This is a potentially important finding, because it may bear on the extent to which the auditory and linguistic levels of speech processing are actually separable. It will take many years to sort this out for at least two reasons. One is that our best imaging techniques (e.g., magnetic resonance imaging, PET scanning) do not identify cortical territories with the

spatial or functional accuracy we need. Secondly, in both cats and primates, the spatial loci of functionally defined cerebral territories vary considerably between individuals (Merzenich et al., 1975; Knight, 1977; Imig et al., 1977). If this phenomenon extends to human beings, which is very likely given the striking individuality of cortical fissural patterns in man, then we have both factors working against us. This is all in addition to the problem of defining the boundaries of the cortical insult, which may be very diffuse.

Dr. Sloan's commentary makes the important point that audiometric sufficiency is an ambiguous indicator of the integrity of the other auditory processing capabilities of the individual. Our point here is that the process of "hearing" has many dimensions and, therefore, that no single test is likely to reveal the functional status of all of them.

Sloan, like Musiek, refers to my notion that the threshold stimulus level for a cortical neuron might usefully be defined in terms of a signal-to-noise ratio. This concept evolved from our studies of the effects of noise masking on tone-evoked responses in the cortex. While we have pointed to the salience of these tone responses as being in marked contrast to that seen in lower structures (especially the auditory nerve: Costalupes et al., 1984), we must be cognizant of the fact that the mechanisms which give rise to that salience are not wholly cortical in locus. The processes that shape this behavior begin at least as far caudally as the auditory nerve, where there is also some dynamic adjustment of tone sensitivity in the presence of masking noise, although it is manifested in less dramatic fashion than in the cortex. What we see in the cortex represents the cumulative effect of many synaptic processes exerted across the length of the afferent pathway.

This issue may be relevant to the "cocktail party" problems experienced by listeners with cortical lesions. My paper addresses this issue only in terms of spectral coding. There is, however, a *temporal* aspect to this issue. The general problem of discriminating one temporally-varying signal (e.g., speech) in the presence of other sounds is not usually as simple as discriminating a structured signal against a background of continuous, invariant noise. The background mask is typically in the form of other speech. Normal listeners can use a number of cues to separate these streams: spatial location, pitch, and so on. Now, if we accept that the primary auditory cortex has a role in the perceptual elaboration of signals with a particular time frame (milliseconds to tens- of-milliseconds), then in the presence of multiple, simultaneous conversations, the temporal processing power of the word-deaf listener is very rapidly exceeded, and the conglomerate sound might be perceived as "jabber" or "noise." They may be more susceptible to this "temporal" noise than the normal listener.

Finally, Sloan implicitly makes another point: that we must be careful and thoughtful observers. It does not matter whether we are neurobiologists or practitioners. The onus is on us as professionals to be alert to every relevant nuance of our subject's behavior, whether our subject is an individual neuron or a person. Perhaps the challenge lies in knowing which of the nuances are the relevant ones. D.P.P.

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