

ROOTS: 5 NOTES ON THE HISTORY OF NEUROLINGUISTICS

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Prolegomena 1: using historical studies

Norman Geschwind's work in the 1960's [1,2] was influential in kindling an interest among contemporary neuropsychologists in the historical contributions of late 19th century neuroscientists, notably that of Wernicke, Liepmann and Dejerine whose models of brain function had, over the intervening half-century, slipped from their former prominence. Geschwind's interest in history was straightforward: the connectionist model of brain function which he had come to believe in, had clear origins in the work of these earlier scientists; it was good scholarship to recognize that indebtedness as well as interesting and entertaining, all of which we should consider a first 'use' of history: finding the roots of scientific concepts, the background of contemporary ideas.

Concomitant with searching out roots is the more difficult task of placing historical contributions in their proper context, to understand what might have been dictated by necessity, what might have been a limiting factor because of the then-dominant scientific paradigms, where there were true breaks with tradition and where not, what knowledge was built up incrementally through one or more trends and what accidents of popularity, influence, social pressure and the like might have led to one event rather than another. Consider, for example, why

Broca focused on disorders of speech production (*langage articulé*): at least part of the reason is that before the 1860's, one did not talk about the <comprehension> of language. Language was *constructed* as speech output and the rest, what would have been or could have been discussed under the notion of comprehension, fell under the notion of <mind> which at the time was in the province of philosophy, religion and nascent psychiatry (the alienists). Since the 1820's the medical, and as well the phrenological, journals had been filled with case reports of expressive language impairments arising from stroke and trauma, with and without autopsy evidence of the involvement or lack of involvement of the frontal lobes [3]. The work of Bouillaud, Lallemand, Broussais, Dax and Lordat are the more familiar names, however there were dozens of obscure medical practitioners publishing these reports; Alexander Hood was one such lesser known researcher [4], about whom more will be said below. Beginning at least as early as 1866 with the publication by Theodor Meynert of a case of receptive aphasia with jargon [5], followed by Bastian's symmetric model of language input and output in 1869, and Schmidt's case of receptive aphasia in 1871, the stage was set for the *re-construction* of language --by the neuroscience community-- to include comprehension, seen in the work of Hughlings-Jackson and Wernicke starting

in 1874. Two exemplary models of historical analysis of the trends in the neurosciences as well as insightful expositions of the varying milieu are to be found in Harrington [6] and Clarke & Jacyna [7]; a third and monumental compendium of the origins of neuroscience from the earliest written records --nearly a third of the chapters bear directly upon neurolinguistic issues-- is Finger's recent text [8].

Prolegomena 2: pitfalls in historical research

Establishing priority is, in a word, fun. It is intellectually entertaining to learn that Roberts Bartholow, in 1874, was the first person to electrically stimulate the human brain with an electrode directly inserted into the cortex (when the electrode was pushed deeper into the cerebrum, possibly in the basal ganglia, possibly in the thalamus, most certainly sub-cortical, the subject of this experiment did cry out; otherwise, he did not elicit speech and did not test to see if his electrical stimulation interrupted speech), regardless of what one may think of the ethics of this experiment (he was publicly berated in a British medical journal) [9]. However, establishing priority is typically a very tricky enterprise. Consider the following quote from David Caplan [10, p. 46]: "The 1861 paper by Broca is the first truly scientific paper on language-brain relationships." Caplan supports this conclusion (here, as elsewhere, Caplan successfully integrates historical with contemporary research) with three claims, viz. that Broca presents a detailed case history with "excellent gross anatomical findings at autopsy", that Broca has the insight that the gross brain convolutions

are constant anatomical features that may be related to particular psychological functions, and that Broca's primary conclusion that expressive speech depends upon a small part of the inferior frontal gyrus is a good first approximation which we generally accept today. Clearly, <priority> in this example is a matter of scholarly judgment. What then to make of the fact that Alexander Hood, in 1824, did a better job of analyzing expressive language functions and correlating them to frontal lobe anatomy? Hood had postulated a lexical-phonological level, a phonological-articulatory level and a motoric level for expressive speech, based upon the speech and language impairments which he observed in stroke patients. The oddity is, he used the phrenological model of Gall & Spurzheim [4, 11, 12]. What then to make of the fact that excellent clinical-pathological --autopsy-- studies of aphasic cases may be found in the 17th century studies of Wepfer [13], studies that are so good one may verify the left hemisphere localization of language from them, or, the fact that Lallemand and Bouillaud in 1824 and 1825 published dozens of autopsy reports of patients with aphasia? What then to make of the fact that the classical neuroanatomists of the late 19th and early 20th century virtually abandoned the possibility of systematically describing gyral geography because of its evident variability, a variability that currently plagues PET researchers who need to co-register sites of PET activation with MRI images in order to cross-subject compare results? And, finally, what to make of the fact that the autopsy of Broca's 1861 patient actually demonstrated a very large left hemisphere lesion encompassing frontal, parietal and temporal cortex?

Broca “inferred” that the 3rd inferior frontal part of this large lesion was the one responsible for the patient’s aphemia, by estimating the degree of necrosis and trying to back-correlate that with the patient’s medical history. What is important to appreciate here is that it is not a question of disputing the facts but a question of how one chooses to interpret the historical record. A view which I prefer is (a) that Broca inherited a tradition of clinical-pathological correlation that already presupposed that different brain regions had different functions, (b) that Broca was theoretically constrained by a construct of language that placed psychological pre-eminence on speech production, (c) that Broca was immediately challenged and certainly intrigued by the debates (involving many famous members of the French scientific community, e.g. Gratiolet, Bouillaud, Auburtin, Flourens, et alia) concerning the role of the frontal lobe in speech and therefore was predisposed to see the age of that lesion as having a significant frontal component, and, most important of all, (d) that Broca had the position, power and prestige to take advantage of a serendipitous clinical observation.

Prolegomena 3: what not to do

Although some historical “facts” are subject to interpretation as we’ve just seen, some are just plain right or wrong, and, it behooves us to get it right. It is quite another matter, however, to commit the unpardonable historical sin of *presentism*. Consider the following quotation from John Morton [14, p. 40]: “We have a number of lessons to learn from history. If we are lucky we can avoid making the same mistakes as

thinkers in the past.” The mistake made by the “diagram-makers”, according to Morton, was to confuse the goal of representing the elements of language processing in the brain with the goal of determining the localization in the brain of these elements. Needless to say, Morton assures us that “the same mistake will not be made again...” [14, p. 61], leaving this reader with the clear impression that his logogen model has, at last, revealed the truth about language (*veritatem patefacere* - Cicero). There is a fine line between science and religion and Morton’s rhetorical style makes it hard to tell if the line has been crossed. That, however, is of less concern at present than the notion of an historical “mistake”. Following the scientific paradigm of the day is simply not a mistake; to evaluate an earlier paradigm using the principles of one’s own paradigm is *presentism* --to judge the past by today’s standard. Most historians do not regard this as very productive. On the other hand, scientists do make mistakes, past and present company included, and some of the historical errors in brain-language relationships are quite interesting. Consider Franz Joseph Gall’s localization of language functions (*sprachsinn und wortsinn*) in the anterior, inferior frontal lobe. The craniological method of relating skull protuberance (the “bumps”) to hypertrophy of the underlying brain region, in turn due to above-normal development of the faculty which is expressed by that same brain region, is an unexceptional scientific method. We may find it humorous but it is a clear and falsifiable hypothesis. And in fact, one could argue that Flourens’ experiments which demonstrated that animals whose cerebellums he had lesioned still exhibited

copulatory behavior, which thus provided evidence against Gall’s localization of the reproductive faculty in the cerebellum, was one of the principle reasons why many scientists rejected craniology, later phrenology; Gall refused to accept Flourens’ evidence -- the scientific community, particularly Bouillaud, accepted it. On the other hand, Gall’s argument that a well-developed language faculty, particularly verbal memory, would cause a protuberance of the inferior, anterior frontal lobe, which in turn would make the eye sockets shallow --thus, folks with high verbal skills were said to have “cow’s eyes”-- was not successfully challenged by the scientific community. Rather, Bouillaud not only accepted *this* localization but championed it unceasingly right up to 1861 when Broca’s publication seemed to vindicate Gall’s model. What is curious is that it was well known at the time that the backside of the eye sockets do not abut the frontal lobe --a great deal of sinus cavity lies between the two. It is virtually impossible that a frontal brain bump could impinge upon the eye sockets. Evidently it was the accumulating evidence that frontal lesions typically led to speech disturbances, documented by Lallemand, Bouillaud and others from the 1820’s on, that kept the phrenological language model alive until the great paradigm shift of the 1870’s. Another error, not fully appreciated until recently, was committed by Lichtheim, one of the diagram-makers discussed in Morton’s chapter. Laubstein [15] has elegantly shown that this “paradigmatic diagram-maker” had produced a neurolinguistic model that is ambiguous with respect to some predictions of language disorders, that fails to predict some language disorders

that had already been described, that is internally inconsistent and, finally, that cannot be falsified, all in terms of the 19th century paradigm within which Lichtheim operated. This is the kind of analysis of the diagram makers diagrams that goes to the heart of the basic model-making assumption of that period and of our own: the correlation between aphasic language data and the components of the processing model of language used to account for such data.

Prolegomena 4: psychological vs neurological modeling

Having argued that what Morton says is the diagram-makers “mistake” should not be considered a mistake, let us examine the actual claim that Morton makes: did the diagram-makers confuse the psychological (processing elements) with the neurological (localization of elements)goals of their neurolinguistic enterprise, as Morton asserts? Baginsky (1871), the first diagram-maker discussed by Morton, believed he was basing his model on the “physiology of speech formation”; he did not stipulate specific anatomic sites for each of his language “centers”, maintaining that “we do not yet have a precise conceptualization” of this relationship [16]. Kussmaul, the sixth diagram-maker discussed by Morton, claimed that his colleagues, particularly Wernicke, were mistaken in trying to localize the various speech centers to specific regions of the brain. Kussmaul was “acutely aware of the limitations of the localizationist approach to linguistic processes” [16, p. 509]; “extraordinarily removed from strictly anatomical and physiological considerations, Kussmaul the physician achieves an understanding

of the psychology of language in terms of the concepts which constitute the core of present day models, e.g. the distinction between various levels of representation ...their respective autonomy yet interconnection, and the notion of linguistic processes" [16, p. 497]. The same may be said of Elder [17] and Grasset [18] both mentioned by Morton, as well as of Bastian [19] who, though not discussed by Morton, was one of the best-known of the British diagram-makers of the period. As Paul Eling [19] remarked: "In general, characterizing the work of these classical aphasiologists with a few short statements and adjectives does not do justice to the careful analytic description and argumentation of these scientists." In fairness it ought to be noted that Morton's questionable analysis of the model-theoretic assumptions of Baginsky, Kussmaul, Elder, and Grasset, may not be entirely his fault; he relied on Moutier's 1908 dissertation as his secondary source material. Moutier was the student of Pierre Marie, notorious for his antipathy toward anyone who fractionated language into its component elements and thus anyone who believed that there were several different types of aphasia, obviously the main tenet of the diagram-makers. Ironically, and history sometimes has a penchant for the ironic, it was Pierre Marie who proposed that the insula (Island of Reil) was a functional component of expressive language (Marie's quadrilateral). George Ojemann and myself, about two decades ago, established that the insula can be language cortex (electrical stimulation of the insula elicited naming errors) and recent work by Nina Dronkers, using the lesion-overlap technique, suggests that insular lesions lead to apraxia of speech, a view

quite consistent with Marie's view as Dronkers has noted. To return to the question of psychological Vs neurological modeling, Marie's fundamental objection to the localizationists/diagram-makers was a psychological one, viz. the dictum *l'aphasie est une*. In Marie's view the reconstruction of language in the 1870's to include comprehension now became reconstructed again so that comprehension (understanding, the lexicon, etc.) now was language and speech production was relegated to the status of motoric output. To this day neurolinguistics has wrestled with the motor component of the expressive aphasias. In the 1960's and 1970's this was one of the major theoretical disputes between the Mayo School (Darley, Aronson, Brown et alia) and the Boston School (Geschwind, Goodglass, Benson et alia), a dispute which, with the benefit of two decades of hindsight, squarely addressed and never resolved the different demands of a psychological Vs a neurological model of language.

Prolegomena 5: gaps in the story (1600-1900)

As entertaining as it may be to learn that Pharonic medicine circa 3000 B.C. recognized temporal lobe injuries as leading to aphasia [8], the knowledge was not passed on to later cultures. Comments in the Hippocratic texts --which do have historical continuity with the present through the reintroduction of Greek texts via Arabic in the early Renaissance-- refer to what we would likely label dysarthria or aphasia and additionally to right-sided paralysees sometimes associated with speech disorders; it is debatable that these neurolinguistic observations were ever

systematically understood [20], although several recent historians have argued that the epilepsy commentaries indicate that the Hippocratic physicians did understand the connection between unilateral brain lesions and symptoms to the contralateral side of the body. From the period of Plato and Aristotle through the time of Galen and up to the Renaissance, many observations on the loss of speech and language associated with either intrinsic brain disease or traumatic injury, were written. However, based as they were for the most part upon theories of meningeal or ventricular function, these accounts differ substantially from our concepts of brain function, as is very well documented in O'Neill's scholarly text [20]. Benton & Joynt [21] pointed out that most of the "classical" aphasias had been described ("observed" would be more apt terminology) by 1800. O'Neill demonstrated that at least through the Renaissance (beginning of the 17th century) these observations were hardly part and parcel of any general, coherent theoretical model of brain-language relationships [20].

The dominant brain function model before the Renaissance was "ventricular theory", derived from Galen and elegantly modified by Descartes among others; basically, this was a model based on fluids and fluid flow for the obvious reason that thoughtful early scientists realized that something in the brain must move in order to be responsible for functions -- something passes from sense organs to effectors and the animal spirits were as good a candidate as any available. One marvels at medieval and early-Renaissance discussions of memory disorders following damage to the 4th

ventricle which are models of clinical-pathological correlation despite the casual disregard of the actual neuroanatomy. However, by the end of the Renaissance period, ventricular theory had been disproven by, for example, the cases reported by Johannes Schenck (1530-1598) in 1584, cases with 4th ventricle damage in which memory was spared and cases with damage to the cortical substance in which the 4th ventricle was intact but memory was impaired [13]. Ynez O'Neill's summary of early neurolinguistics ends at the 17th century, leaving us with a number of gaps in the story from the Renaissance to the 20th century, gaps that are only partly filled in by current research on persons who have actually made substantial contributions to the development of neuropsychology and neurolinguistics.

Little has been written [13] about the 17th century brain scientist Johannes Jakob Wepfer (1620-1695); for our interests here, his posthumously published book, *Medical-practical observations of affections inside and outside the head* (1727) is most relevant. In it Wepfer discusses 13 well-described cases of aphasia, often noting *paralysis in dextri lateris, cum loquelae impedimentum* and yet never drawing the self-evident conclusion that left hemisphere lesions and right sided paralysis were associated. Perhaps the fever from which he died, overtook him before he completed his work; perhaps the reason for his silence on the matter of laterality was that his contemporaries, particularly those in the church, might have viewed such localization as too materialistic. Galileo was "processed" less than 30 years before

and the squares of Europe still smelled of the stakes of the Inquisition [13].

Although David Hartley (1705-1757), an early 18th century village doctor practicing without benefit of a medical degree, did not write on language nor did he study patients with brain damage, he was one of the first to explicitly propose a brain-based model of psychological functions [22] (Thomas Willis, a contemporary of Wepfer, had proposed the rudiments of such a model in the century before). His psychological theory was the associationism of Locke and was later destined to be the dominant neuropsychological model of the 19th century. His physiological theory was based on elements he called vibratuncles (analogous to Willis' corpuscles which were in gentle vibration and directly borrowed from Isaac Newton [8]) which allowed him to account for the transmission of sensory images into the brain, motor operations out of the brain, attentional and memory mechanisms in the brain and, presaging neuroscientists of the 19th century, his vibration theory led him to a concept of domain specific localization of function.

Much has been written about Franz Joseph Gall's (1758-1828) contribution to neuroscience [3, 4, 6, 7, 8, 11, 12, 19 and references in these studies] but not a lot is known about the roots of his ideas. Christine Grou, in her unpublished doctoral dissertation, demonstrated a close parallel between the faculty psychology of Thomas Reid (1710-1796) and the faculties of Gall & Spurzheim and also a commonality between the many (hundreds of) physiognomic characteristics proposed by Johann

Kaspar Lavater (1741-1801) and the phrenological faculties. Gall's idea that growth patterns of the cortex, i.e. hypertrophy or atrophy, would impress themselves on the inner table of the skull and thus be "readable" as bumps on the skull, was directly borrowed from Lavater. The great 18th century naturalist Charles Bonnet (1720-1792) proposed a vibration-based theory of memory reminiscent of Hartley; Bonnet also proposed a doctrine of localization of function in the brain that clearly had influenced Gall as the latter cites the former in several of his books. However, the details of Gall's indebtedness to these 18th century scientists remain to be elucidated. On the other end, we have worked out a few of the connections between craniology-phrenology and the development of neuropsychology in the period from 1820-1860 [3, 11] and we have also begun an analysis of how the early phrenologists helped to found the doctrine of clinico-pathological correlation of language impairments [4]; little is known about phrenology's contribution to other aspects of neuropsychology and psychiatry. Craniology-phrenology was quite clearly an early personality theory, cf. its roots in physiognomy; whether and in what respects it may have influenced the development of personality theory in modern psychology as well as psychiatry are not well worked out.

Historical analyses can help us realize that our neurolinguistic models (a) have precursors, (b) are contextually influenced by the scientific milieu and (c) are relative to the assumptions and constraints of the paradigms we happen to currently accept. And they can amuse.

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