EVOLUTIONARY PERSPECTIVES
ON LANGUAGE AND BRAIN
PLASTICITY

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Our understanding of speech and language disorders may be aided by information about the constraints and predispositions contributed by neural developmental processes. As soon as we begin to look at human neuroanatomy and development from a comparative perspective, it is possible to recognize a number of ways that human brains diverge from the general pattern of other ape and monkey brains. These divergences may offer clues to language evolution. Large-scale quantitative changes in the relative proportions of brain regions (as opposed to just overall expansion) offer some of the most obvious clues. Additional information about how axons are guided in their extensions to distant developmental targets and how competitive trophic processes sculpt these connections also provides a way to understand how gross quantitative changes in cell numbers could affect circuit organization and ultimately behavior. © 2000 by Elsevier Science Inc.

Educational Objectives: The reader will learn how general principles of brain development have contributed to both human brain plasticity and the acquisition of the human capacity for speech.

KEY WORDS: Evolution; Speech; Language; Neuroanatomy

INTRODUCTION

Neural plasticity is currently a hot topic for discussion and research in the study of recovery from brain damage. There is now growing evidence that adult brains are capable of plastic adaptation to injury including some degree of structural reorganization, contrary to classic theories. However, it is also becoming clear that some earlier ideas about the ubiquity of plasticity of children’s brains has been overexaggerated and that quite specific effects of early brain insults may persist and produce complex patterns of impairment that are different than in more mature brains. These somewhat confusing aspects of neural plasticity may make more sense if viewed in the context of the origins of the mechanisms involved. This is because what appears to be the operation of a reparative mechanism in the brain is actually the side effect of a set of mechanisms evolved for very different functions.
The neural plasticity widely evident during brain development and the more modest flexibility exhibited by adult brains in the face of damage turns out to be mostly the modified expression of normal ontogenetic mechanisms, not reparative mechanisms. Their openness to extrinsic information makes these brain development mechanisms incidentally available to respond to damage where responsiveness to extrinsic change can make a difference. This means, however, that the concept of neural plasticity, conceived as a mechanism for repair or adaptation to abnormal conditions, is mostly a misnomer. Reframing these plastic effects in terms of normal developmental processes and how they may be affected by disturbances of normal brain development or by damage at other stages of maturity provides a context for explaining some of the complex functional reorganizations that may otherwise seem odd in the context of repair and recovery of function. It may also help to guide therapeutic approaches to know to what extent responses to brain injury are not merely neural analogues to tissue replacement, scarring, or bone repair.

To understand these mechanisms, we must look beyond their roles in development, to evolution. Evolution is accomplished by changes in developmental mechanisms. The openness and responsiveness of these mechanisms is a reflection of the advantages they provided for the evolvability of brains. Thus one purpose of this review is to exemplify the role played by plastic developmental processes in the evolution of specific neural architectures. To make it more relevant to human-specific disorders, their likely role in the evolution of a uniquely human neural adaptation is examined: the evolution and development of articulate control over vocalization in speech. Human speech is particularly useful as an exemplary case to study because it demonstrates how even a highly unusual human adaptation is totally dependent on the operation of relatively generic but context-sensitive mechanisms. Indeed, it turns out that plastic developmental mechanisms have played a particularly important role in the evolution of language and speech. As a result, functional plasticity can be a major factor in the ways that language abilities can be disturbed and reorganized in processes of recovery.

THE EVOLUTIONARY-DEVELOPMENTAL CONTEXT

If brains were designed the way we design watches or computers, the flexibility from generation to generation and evolutionary adaptation in the long run would be a near impossibility. The brains of large mammals, like ourselves, are some of the most complex objects that have ever existed. With so many billions of interconnected interdependent parts, significant modifications of one component would need to be correlated with complementary modifications of innumerable other interdependent components to avoid catastrophic disruption of system-wide functions. This is one very good reason why no one recommends redesigning computers or television sets by randomly modifying connections or parts in millions of devices and then testing to see which “mu-
tations” work best. Aside from the obvious waste involved, the chances that any of the millions of possible changes would produce enhanced function are essentially zero. Technological progress is the result of redesign where the engineers involved must pay very close attention to all the detailed ways that their innovations interact with one another and the other parts of the device. Redesigning and upgrading complex systems, such as microprocessors and jet airliners, requires tens of thousands of engineers contributing thousands of hours of checking and rechecking both how their own parts work and how these parts interact with those designed by others. Even then, surprises tend to emerge when the whole system is first assembled. Further, these designs are nowhere near as complex as even simple organisms and their brains. Living things are not like designed devices because that approach is far too cumbersome ever to have evolved. So what is the alternative?

For generations, biologists conceived of the evolution of brains as a kind of incremental trial and error engineering design process. New adaptations, new structures, and new functions were presumed to be added to previous ones, producing more and more complicated brains, which were consequently larger as well. This model of brain evolution and function shares many features with models of technological progress and with modular design strategies for building complicated human devices. Development during a lifetime was conceived as adding new parts to an earlier brain plan. Thinking about evolution in this way suggested, for example, that ape mental abilities may be comparable with those of a young human child of a few years of age, but that human children “progress further” in brain development than apes by adding new stages and the maturation of new parts.

However, this view of evolution and development is an anachronism (Deacon, 1990b). Evolution produces organisms according to a very different modus operandi than followed by the purposive design of useful tools and other devices. This inevitably produces very different kinds of solutions than would be expected by engineering design. One reason is the shortfall of genetic information that is available for specifying the neural architecture of a developing brain. This shortfall is exemplified by the fact that although human brains are just under three orders of magnitude larger than mouse brains; with vastly more subdivisions and connections to be specified, they appear to be constructed using little if any additional genetic information. The information must come from somewhere. But where? And what role could processes we tend to associate with neural plasticity play? It appears that this extra information is recruited from extragenomic sources by virtue of ontogenetic mechanisms evolved to be open to contextual information about linked systems and even environmental input. This openness to extrinsic information is enabled via the signal-carrying capacity of neurons. In this way, neural circuits can participate directly in their own construction.

It is not too much of an oversimplification to say that the information that was used to build my foot was contained in the cells of my foot, but it is quite
mistaken to say that the information used to build my visual cortex was contained in the cells of my visual cortex. In fact, only a minor fraction of the information that determined this region’s complex structural features came from visual cortex cells. Some of the most crucial information came from other parts of the brain, and only a fraction of this is directly reflected genetic information. Some of the information even came from the patterning of light that struck the retina during development.

The detailed functional architecture of the visual cortex is in part determined systemically, by a process that crudely parallels Darwinian selection. Parts of the brain and peripheral nervous system can influence structural and functional differentiation some distance away as regularities of signaling are carried throughout the developing neural networks and influence how connections are made and persist. This signaling can indirectly convey information about population attributes of other brain regions, such as the numbers of cells or connections, their topographic organization, and the patterned structure of the perceptual environment.

Not all attributes of brain structure are determined this way. Large scale structures exhibiting continuities of tissue organization and cell type are clearly determined by ontogenetic events orchestrated by gene systems like those that determine other major organs and their major parts. These genetically constrained “segmentation” processes predominate in the early stages of embryogenesis and, by determining major spatial and temporal relationships in the developing brain, play a major, although indirect, role in shaping the later dynamic processes that convey systemic effects. Local intrinsic cell fate determination is mediated by diffusible signaling molecules, by clock-like differentiation events in contiguous regions, and by systemic effects on cell–cell interrelationships that link separated structures. The relevance of these global and relatively early acting genetic influences will be considered in some detail later. But over the course of ontogenesis, there is a progression from an early predominance of differential gene expression to a later predominance of systemic activity effects on morphologic features. This produces a global-to-local trend as well. Large-scale distinctions between tissues that contain distinct cell types are generally the products of early molecular expression domains, whereas local distinctions of cytoarchitecture are more often the products of later activity-mediated processes. The final brain organization reflects a complicated mix of these influences.

**DARWINIAN-LIKE PROCESSES IN NERVOUS SYSTEM DEVELOPMENT**

Starting with the process of cell production, we can see clear evidence of Darwinian-like selection processes at many levels of the nervous system. Some of the first examples discovered involved the production of neurons di-
rectly controlling motor neurons. During development of the spinal cord these output neurons are produced in greater abundance than persist in maturity. Sympathetic ganglia, whose neurons project to the smooth muscles of the viscera, and spinal motor neurons that project to the limb muscles seem to go through a culling process as the organism matures. In frogs and chicks, where it was easier to experiment on early embryos, it was discovered that the extent of this culling could actually be increased or decreased by modifying the peripheral organs to which they projected axons. By removing their targets, more cells were induced to die off, and by grafting additional organs (e.g., a supernumerary limb), fewer cells were eliminated. Apparently, these cells were initially overproduced and then found themselves in competition for resources somehow provided by the peripheral target structures.

Early in development the axons of motor neurons grow somewhat exuberantly and nonspecifically and end up overlapping one another on the same muscle cells. Competition ensues to make synapses with these cells, which ultimately only one axon will win, and only those with stable synapses seem to provide molecular signals necessary to keep the source neuron alive. Those branches of axons that fail to establish connections die back, so to speak, and those cells that fail to establish any stable connection will die altogether. Selection mediated cell death, then, turns out to provide a precise mechanism for matching the sizes and distribution of populations of neurons to the sizes and distribution of muscle masses in the rest of the body. From an evolutionary point of view, there need be no correlated change in neural cell production or cell distribution to match changes in muscle size and distribution that have resulted from selection for different modes of locomotion.

The same logic turns out to be used throughout the developing brain, and not just for motor systems, but for sensory systems and even intrinsic systems as well. Consider the visual system, for example. In different vertebrates, the direction that the two eyes face may differ by almost 180° (as in many fish and hooved mammals) to almost 0° (as in owls and humans). In species where the visual fields overlap, there is the possibility of using the nearly redundant information to aid in depth perception, but given the range in possible overlap, one may suspect that it needs to be accomplished differently in different brains. Indeed, the way these connections map onto the visual analyzers in the brain is quite complex. As is the case for many sensorimotor systems, the visual projections into the central analyzers of the brain maintain a topographic organization (although somewhat distorted, as are many world map projections). In animals with binocular overlap (like monkeys and ourselves), each half retina views most of the same visual field as the same-side half retina of the other eye. The projections into the brain of an adult split according to their visual field of origin, so that the parts of the eye that view the left visual field cross over in the midline on their way into the brain and both map onto the cerebral cortex (the folded gray matter sheet that covers each half of the forebrain) on the right side.
What is remarkable about these maps is that they are both separated and overlapping in a complicated way in the visual cortex. It is as though each map was cut into strips (like zebra stripes) and then put together into a single map, interposing each sides’ strips between the other’s, so that points on a strip that represent the same point in viewed space are aligned right next to one another. This allows neurons nearby one another to compare signals, and thereby extract depth information from the slight shifts in disparity that result from the way distance influences the convergence or divergence of lines of sight. One may imagine that such a complicated map organization, requiring such precise alignment, would require very detailed prespecification. Evidence to the contrary was discovered over two decades ago when it first became possible to trace the course of individual input connections at different stages in development. What was found was an early, rather messy pattern of projections in which the two eyes’ maps almost diffused into one another with poor point-to-point precision. Axons tended to branch and fan out in overlapping patterns in the visual cortex. But during development, the degree of overlap and fan out is reduced via competition between axons, and many of what in hindsight we may call misdirected and nonspecific branches are selectively eliminated to produce the final, precisely sculpted pattern.

These examples beg the question of exactly how the competitive processes work. The answer is only incompletely known and almost certainly involves many distinct mechanisms. Most agree on two critical components: (a) in many systems there are growth factors that are provided by the recipient (postsynaptic) cells that are necessary to maintain patent connections and for which axons compete, and (b) what determines whether an axon will be able to hold onto a synaptic connection with respect to competing axons vying for the same target cell has something to do with correlated activity patterns that favor those axons staying connected to the same target cells that tend to fire in synchrony with one another and with the target cell (although antisynchrony with the target cell may also be involved in some cases). The result is that subtle spatial and timing biases in initial connectivity contribute to the relative synchrony or dyssynchrony of signals converging on any particular target, and these biases become amplified by the progressive action of millions of signals and selective elimination events that ensue. Thus some initially rather subtle biases that can be controlled developmentally by some rather generic growth processes are capable of adjusting the development of both large scale and microscale connectional patterning, and in the end produce intricate, appropriately detailed circuits to match.

It now appears that this general principle is at work at all levels of brain development. In fact, the remarkable pattern of maps for different sensorimotor methods and submethods that divides up the cerebral cortex of mammals probably is also only loosely biased by genetic design and yet comes to exhibit a remarkable interindividual and cross-species consistency. A study by
O’Leary (1992), for example, has shown that transplanting immature cerebral cortex from one position to another in the cerebral mantle of rats does not cause the later-appearing sensorimotor maps to track the repositioning. Rather, they develop input and output connections that are appropriate for where they end up. The reason for this was uncovered by O’Leary and colleagues (reviewed in O’Leary, 1992) in studies that showed that early cortical outputs are quite nonspecific with respect to their targets. All areas of cortex initially send outputs to nearly all types of cortical targets, mostly by way of extra branching of their axons. Later in development these branches are pruned so that visual areas only project to subcortical visual processors, auditory areas only project to auditory processors, and motor areas only project to motor structures. Thus it does not so much matter where in cortex the projections originate.

There is even a curious natural experiment that demonstrates this. The blind mole rat (Spalax), has only vestigial eyes. Its lateral geniculate nucleus receives most of its input from midbrain auditory sources, and there is a corresponding shifting of functional boundaries in the rest of the thalamus and in the recipient areas of the cortex to match (Doron & Wollberg, 1994). Where other rodents have visual cortex, the blind mole rat has auditory and somatic sensory cortex, not because the one was eliminated and the others added in any modular sense, but because in the competition for space, the displacements of connection patterns at lower levels produced ramifying effects at all others. Thus, cortical areas’ inputs and outputs are both competitively determined along with the patterns of connections within cortex as well. It is a pattern generation process that is entirely systemic and distributed.

Because much of the information for wiring brains is “rediscovered” rather than genetically inherited in each generation, many species brain differences may not be specified in any detail in their genes. The very same genetic and molecular information may serve very different purposes in different parts of the brain and in different brains. Some of the most convincing evidence for this cross-species generality comes from experiments in which chimeric brains are produced by transplantation techniques. Immature neural cells, harvested before the point in development where neurons have extended fragile axons and dendrites, can be transplanted from one brain to another, both across ages (from fetal to adult) and across species. By placing immature neurons from one stage or species into another, it is possible to probe experimentally for the relative influence of intrinsic and extrinsic signals for development.

As part of an effort to develop alternative fetal cell sources for transplantation treatments for neurodegenerative diseases, Isacson and Deacon (1996) studied the growth of fetal cells from pig brains in the brains of adult rats. The Darwinian features of neural developmental processes had given the authors reason to predict that the signals that controlled neural maturation and connectivity in these different species would likely not be all that dissimilar and so
may allow cross-species transplants to function nearly as well as same species transplants (so long as their immune rejection could be held at bay, which was accomplished with the same sorts of drugs used for other types of organ transplants). It also gave us a chance to observe exactly how the different species’ cells would interact. Remarkably, pig neurons taken from one region of the fetal pig brain and implanted into an adult rat brain grew their axons to the rat brain target structures that were homologous to the normal pig brain targets for those neurons. Cortex cells grew to striatum and midbrain, striatal cells grew to midbrain but not cortex, and midbrain cells grew to striatum and cortex, and so on. Growth into these targets did not appear to be topographical, but rather generic, and could occur even if the transplants were placed in odd sites within the host brain. Most importantly, these newly established connections could restore functions in rats that had lost these functions as a result of experimental brain damage. The therapeutic implications are that neither same species donors nor detailed control of the formation of neural connections are required for functional recovery.

The evolutionary implications are equally significant. Even the incredibly species-specific functions, as are involved in language processing, may have been achieved using the same old developmental information that is present in other primate species, just slightly modified by systemic effects and generic biases. For this reason, understanding how such a uniquely human function arose in the first place may not require any highly specific genetic explanation. One way to describe this is to say that there are no intrinsically prespecified language circuits in the human brain, only connection patterns that have been biased in unique ways so that they are slightly better suited to the unique demands imposed by language. This developmental logic of brain design does not preclude the evolution of localized modular systems, nor does it exclude the possibility of significant species and individual differences that have a precise genetic basis, it just means that there may be more flexible ways of producing these differences.

Combining this developmental logic with data about the quantitative structure of human brains with respect to other species, it may now be possible to construct a remarkably detailed picture of the structural differences that confer human brains with some of their distinctive functional features. Specifically, I will examine how the size of the human forebrain with respect to the brain stem and body affects the connections that control vocal muscles.

THE VOCAL ADAPTATIONS FOR SPEECH

Some of the most striking evidence for innate language specializations involves the speech adaptation itself. The adaptations for speaking include modifications of the geometry of the oral–vocal tract and modifications of the control of oral musculature. The major difference between human and other
primate vocal tracts is the descent in the position of the larynx in the throat of modern humans. This distinguishes humans not only from other primates but also from most mammals (Lieberman, 1984). This feature has played a major part in recent discussions of human fossil ancestry because it can be crudely inferred by analyzing the angles made by the bone of the base of the cranium. For example, australopithecines (the bipedal apes who lived between 4 and 1.5 million years ago and were our early ancestors) had a skull shape indicating a high laryngeal position similar to that of other apes (Lieberman, 1984), but by 200,000 years ago, at the dawn of *Homo sapiens*, an essentially contemporary vocal tract had evolved. Because of this elongation of the vocal tract, a much larger region of the pharyngeal cavity is bordered by the posterior tongue. Various techniques for modelling the effects of this have demonstrated that it allows a significant expansion of phonatory capacity, increasing the range of vowel sounds that can be produced compared with other primates. Most notably, vowels that involve expanding the pharyngeal cavity while constricting the oral cavity (as in ‘beet’) become possible. Additionally, because the epiglottis is also descended, all human vocalizations are less nasalized than in other primates, allowing the articulations of oral structures to have a greater effect as well. But this has come at a cost.

The modern human epiglottis and larynx have descended to the point where it is now becoming a source of danger, since we can much more easily choke on food or drink as a result (Lieberman, 1984). Although some researchers have suggested that this may indicate that speech was only present in modern humans, it is actually testament to a somewhat protracted use of vocalization in our ancestry. For this anatomical change to have evolved to the modern form, the risks imposed by this unprecedented change in vocal tract geometry would need to be offset by its usefulness, and supporting more distinctive phonation during speech seems its only obvious usefulness.

Of course, the changes in geometry of the vocal tract are only the most visible element in a complex web of adaptations for producing and regulating speech sounds. With respect to sound production, there is a far more significant difference between human and nonhuman abilities. We alone among terrestrial mammals seem able to learn new noninnate combinations and variants of vocal sounds. It is not just that other mammals are less adept at controlling and imitating vocal sounds, most other species cannot even begin. Even extensive training and shaping of sound production in other primates has produced at most one or two very crude approximations to a noninnate vocalization. In contrast, human speech depends on our ability to learn by imitation the thousands and thousands of distinct sound combinations that constitute the words of our native languages. Why are most mammals limited to such a fixed and invariant vocal repertoire, and why should it be so difficult for them to control and modulate their vocalizations? I think of my dog in this regard, who has been taught to bark on command for food, but apparently cannot learn to in-
hibit barking when he hears some other dog barking randomly in the distance. This is an incredible difference between humans and other mammals. This difference is also presaged at a very early age in infancy, by babbling. Other mammal species do not pass through a babbling stage in which there is nearly unconstrained play with many of the combinatorial elements of speech.

The closest examples to this human ability can be found among birds and some sea mammals (humpback whales, in particular). Many species of song birds require some learning to fine tune their inherited singing predispositions to match the local “song dialect” sung by their local community. These species appear to begin life with a sort of crude template for the general pattern of species song but need the auditory experience of hearing it sung to arrive at the local adult song variant. However, more developed sound imitation learning is found in birds like mockingbirds, parrots, and mynahs, which demonstrate a remarkable ability to imitate the sounds produced by members of other species (such as human words) or even sounds of the environment. Perhaps even more human-like imitative ability is found in humpback whale courtship song. These songs can extend to 8 minutes in length and exhibit a combinatorial character in which sound elements are recombined into larger phrase units within larger passages, like words within sentences or motifs within a melody. Even more remarkably, the males within an extended social group (a “pod”) all sing versions of the same song during the same breeding season, even though the song changes from one season to the next, so that all sing a slightly variant version the next year. This demonstrates sound imitation abilities that extend through the lifespan, and an acoustic communication system exhibiting some degree of open-ended generativity (although the constant repetition of the same song over and over again by all males during the breeding season demonstrates that this form of communication is quite unlike language).

These other exceptions to the rule provide important hints to what makes this ability special (for a more detailed discussion see Deacon, 1997). Birds and whales apparently do not use the larynx to produce their songs. In birds, a novel muscular structure called a syrinx (located deep in the chest) is the primary sound generator. In whales (and dolphins), muscles that surround the blow holes and muscles of the elaborate sinus system leading to them are probably the major sound producers. This is an important hint, because it indicates a shift in motor control. The laryngeal muscles are visceral muscles, which along with systems controlling swallowing, breathing, and a variety of other automatic motor functions, are relatively autonomous, preprogrammed, and minimally linked with the forebrain and cerebellar systems that underlie skill learning. These other classes of muscles of the syrinx in birds and the deep facial muscles of whales are much more a part of the forebrain motor system, where learning and fine tuning of movement are important.

In this context, human vocal ability is both more surprising and more understandable. Speech, and particularly singing abilities, clearly demonstrate
unprecedented forebrain control of the human larynx. In this regard, we are not just divergent from other mammals but also from all other vertebrates—perhaps the only one with significant forebrain control of laryngeal muscles. This is evidence of prolonged intense selection favoring increased vocal abilities in our ancestors. Achieving articulate laryngeal control was probably a million-year project at the very least. But there is also something else about speech that stands out: the coupling of precisely timed phonation with rapid articulatory movements of tongue, lips, and jaw. Most of the information of the speech signal depends on the highly skilled movements of these skeletal muscles, superimposed on the regulation of laryngeally produced sounds. Apparently, these oral muscle systems have become very much more precisely controllable by forebrain skilled movement systems during the same evolutionary time span as the laryngeal and respiratory muscle systems came under voluntary control.

So what change in the nervous system accounts for this evolutionary shift in motor control? The reason that most mammal species lack direct skilled motor control over vocalizations is that the relevant muscle systems are part of a relatively automatic system called the **viscero-motor system**. Visceral muscles run, for the most part, on autopilot. Very conserved and highly constrained programs of movement are required because functional mistakes could be catastrophic. Automated preprogrammed control of breathing and vocal muscles guarantees unimpeded respiration in a variety of contexts and avoids dangerous conflicts between eating and breathing. In humans, however, it appears that at least some of the margin for safety in these systems has been sacrificed for speech.

The activation of visceral motor programs is not merely automatic. These programmed behaviors must be subject to adjustment according to arousal state. For example, heart rate and breathing rate, along with other autonomic functions, must be adjusted up or down with respect to variation in drive states and level of emotional activation. Because vocalization is produced by viscero-motor systems, it turns out to be relatively automatic and preprogrammed and also a fairly reliable correlate of arousal state. Speech, however, is relatively independent of any particular emotional state or arousal level, although automatic vocalizations such as laughter or sobbing can override and interrupt speech processes in response to high arousal. This indicates that in humans, these two methods of vocalization are independently controlled and in competition for control of final output.

Further evidence for this dual control comes from neuropathologic data (Jürgens, Kirzinger, & von Cramon, 1982). Damage to ventral motor cortex in humans can produce both paralysis of the oral muscles and mutism, even if the damage is confined to the left hemisphere. In primates, however, cortical damage can produce paralysis of oral muscles but not loss of vocalizations. This comparison is not entirely analogous, because mutism in humans often
leaves such automatic vocalizations as laughter and sobbing intact, and pri-
mate vocalizations are probably more homologous to these innate human vo-
cal “calls” than to speech. Nevertheless, this difference in the effect of cortical
damage suggests that the pathways supporting speech vocalization are suscep-
tible to cortical damage, whereas those supporting more automatic vocaliza-
tions are not.

Because of this, it is generally assumed that speech is controlled via de-
scending projections from cerebral cortex to the brain stem nuclei that control
oral and vocal muscles. The question is how this unprecedented difference in
vocal control came about in human evolution. The answer lies in the relation-
ship between brain size differences and the developmental plasticity that de-
termines neural connectivity.

THE EFFECTS OF BRAIN SIZE ON CONNECTIVITY

The most notable difference that distinguishes primate brains from other
mammal brains and human brains from other primate brains is the relative size
of the brain with respect to the body. Although historically, the larger propor-
tion of brain to body has been solely interpreted in terms of general intelli-
gence, a closer analysis, taking developmental information into account, sug-
gests that the significance of these relative size relationships may have more
to do with connectivity patterns and functional specializations and relatively
less to do with intelligence. On the basis of some quantitative facts we know
about human brains as compared with other species brains, we can venture
some reasonably educated guesses about what changes in ontogenetic pro-
cesses caused us to deviate from more typical ape patterns producing our spe-
cial vocal abilities (for a more complete discussion, see Deacon, 1997). To see
why this may be so, it is first necessary to take a closer look at the nature of
these brain size differences.

One of the least questioned facts about brain evolution is the apparent “ad-
vance” in relative brain size among anthropoid primates as compared with
most other mammals. On average, anthropoid primate brains are twice as
large as would be predicted for a typical nonprimate mammal of the same
body size. Superficially, it appears as though primates have added more brain
per their body mass than most other mammals and that humans have added yet
more brain per body than other primates, thus continuing a trend of increasing
encephalization. These apparent comparative increases in encephalization
have long prompted speculation that humans reflect the culmination of a trend
toward increased general intelligence. But a closer look at the nature of this
increase in brain size to body size (referred to as encephalization) suggests
that it may not be that simple.

The question in this case is whether it matters how these proportional in-
creases came about developmentally. Would it matter if proportionately larger
brain size resulted from relatively stunted postcranial growth, as opposed to exaggerated brain growth? Would it matter when during development these quantitative changes first became evident?

To see that such differences do in fact matter, consider dwarfism. Some of the most encephalized mammals and humans on the planet are dwarves. Dwarfism is almost always the result of the stunting of postcranial growth, often the result of some interference with growth hormones or their receptors and seldom affects brain growth as much as body growth. To my knowledge, despite the fact that dwarfism produces extensive encephalization, there is no evidence that stunted postcranial growth contributes to augmentation of any intellectual functions, and likely the opposite. Although no encephalization theories include dwarfism as a major factor, it turns out that selection on postcranial body proportions is common in evolution as well as in selective breeding. In domestic dogs, for example, we can observe large differences in encephalization between large and small breeds. Small dog breeds tend to be comparatively encephalized and large dogs unencephalized. Could this be a model for certain forms of encephalization identified in interspecific comparisons?

Consider the causes of encephalization in primates. We recognize that the encephalization of small dogs is secondary to postcranial reduction because we can compare them with more typical dogs and can compare their patterns of growth. Although we cannot so easily trace the evolutionary “breeding history” of primates, similar information can be gleaned from features of primate brain growth. A distinct difference between brain and body growth in primates and most other mammals was first noted by Count (1947). He showed that during the prenatal growth period, most mammals grow their brains and bodies according to the same pattern—with the same ratio of brain size to body size at corresponding time points, regardless of eventual adult size (see also Holt, Cheek, Mellits & Hill, 1975). The sharing of a nearly identical cranial versus postcranial growth pattern across wide ranges of sizes and shapes is quite remarkable and suggests that most mammals share a very conservative embryologic brain growth plan (Deacon 1990a). The basis for this developmental regularity remains unknown.

One deviant group, however, is the anthropoid primates. Among monkeys and apes, brains and bodies grow along a trajectory that is parallel to but shifted from that of most other mammals. At every growth stage, primates have a higher ratio of brain to body size (Count, 1947; Deacon, 1990a; Holt et al., 1975; Martin & Harvey, 1985; Sacher & Staffeldt, 1974). Conservatism of the growth pattern is still evident, because growth curves still resemble those of other mammals except for this shift. This primate shift cannot be explained in terms of postnatal growth differences, as in small dog breeds, and yet it turns out not to be a case of increased brain growth either. This can be shown by comparing absolute growth rates as opposed to relative growth rates. When brain and body growth rates are compared between species on the same time
scale, primate and nonprimate species differ in total body growth rates but not brain growth rates. For example, prenatal brain growth in humans, macaques, cats, and pigs proceeds at essentially the same rate, whereas body growth rates for macaques and humans are the same during the early fetal period but are significantly below that for cats and pigs. The clear implication is that primate encephalization is the result of a reduction in postcranial growth processes, not an increase of brain growth (Deacon, 1997; Holt et al., 1975). So, primate encephalization is in this one respect analogous to the encephalization produced by dwarfism. But this comparison ignores one crucial factor: timing. Dwarfism is mostly expressed in postnatal brain and body growth patterns, whereas the primate shift in brain and body proportions can be traced back to very early embryonic stages.

In contrast to the effects of dwarfism, which only show up relatively late in gestation after most stages of brain development are completed, primate body growth reduction is a factor through all stages of brain development. This would be of little significance were it not for the fact that the developmental wiring of brains is influenced by the context in which it occurs. Most of the plastic adaptation of neural populations and connections to the structure of the brain and body in dwarves occurs before the point at which brain and body proportions begin to diverge significantly from the general trend, so it has minimal impact on patterns of neuronal populations and connection patterns. Consequently, dwarf and nondwarf patterns of brain organization should not differ by much. But in primates, even though the locus of the evolutionary change in growth is postcranial, the disproportionality of brain and body proportions with respect to other mammals is present from the very earliest point at which cell populations are differentiating and neurons are extending their axons. Consequently, the reduction in primate postcranial proportions exerts a major influence over patterns of neuronal development at all levels.

One likely effect of this shift in proportions is a change in descending motor projections. Tracer studies of corticospinal projections (Sokoloff & Deacon, 1990) have suggested that nonprimates, such as cats and rats, lack direct cortical motor projections to the brain stem output nuclei that control the muscles of the face, jaw, tongue, and larynx, although they do have indirect projections that terminate in the brain stem premotor reticular nuclei. Primates, however, exhibit both direct and indirect projections to the brain stem skeletal motor nuclei, including the motor nuclei controlling facial muscles (facial motor nucleus), jaw muscles (trigeminal motor nucleus), and tongue muscles (hyoglossal nucleus), although not the visceromotor nucleus that controls laryngeal muscles (hyoglossal nucleus). In immature brains, corticospinal projections initially extend collateral branches to all brain stem nuclei. These are quickly culled from most brain stem nuclei, to be replaced by local projections. The preservation of some of these cortical projections in primates is probably a consequence of the larger size of the primate forebrain with respect
to these nuclei and the competing local premotor projections (although no direct evidence is available to prove this effect). These direct cortical brain stem projections almost certainly enable far more articulate control of orofacial muscles in primates, as compared with most other mammals.

Humans also follow the early primate brain growth trend, but in addition show a further unique modification. Human brains continue to grow as though in a larger species. The locus of this shift is not postcranial and not a segmental alteration of proportions as in the primate-versus-mammal shift. Instead, it results from yet another kind of developmental alteration that involves the brain. Quantitative comparison of brain region volumes in primates and humans indicates that the expansion is largely confined to structures on the dorsal half of the embryonic brain, including structures destined to become cerebral cortex, dorsal thalamus, tectum, and cerebellum (Deacon, 1997). Brain stem structures and body structures remain at roughly chimpanzee proportions.

This differential cortical expansion with respect to other structures during early development has many consequences both for cortical organization and for cortical–subcortical relationships. With respect to vocal control, it further extends the difference between cerebral cortex and brain stem nuclei during the phase when neural connections are still being formed. Extending the developmental logic by which more extensive descending cortical projections lead to more direct cortical control over brain stem nuclei in other primates, the even larger human cerebral cortical projection appears to have led to retention of cortical projections to the visceromotor nuclei that control vocal muscles. This accounts for the unique vocal abilities in humans.

Humans alone have dual control of vocal as well as oral muscle systems. This is crucial for speech, which requires precisely correlated respiratory, vocal, and oral muscle control. With the visceromotor systems also under control of the cerebral cortex, the entire facial–oral–vocal complex can be controlled from the same locus. But what happens to the more automated vocal patterns directed by arousal systems? One likely effect is that there has been a relative reduction of connections carrying preprogrammed vocal expression of emotional tone, and thus a reduction in the number of these call types in humans and a reduced influence of emotional arousal over vocalization. This appears to be the case. Compared with chimpanzees, humans have comparatively fewer innately prespecified vocalizations, and those we have, such as laughter, sobbing, and screams of fright, tend to require high arousal to be generated. In contrast, speech sounds are generated in low arousal conditions, without any preestablished correlations with emotional states. But there is also a subtle indication that some aspects of the preexisting vocal programs have become recruited for use by cortical vocal systems. The prosodic features of speech, which tend to encode speaker arousal, emotional tone, and attentional factors as shifts in fundamental frequency and rhythmic variation, seem to reflect the major dimensions of automatic call structure. So it appears that vocal
muscles are under dual control, with limbic, midbrain, and brainstem inputs playing a background role and cortical inputs playing the leading role, except where arousal is extremely high.

CONCLUSIONS

The evolution of vocal communication in humans appears to be the result of large-scale quantitative changes of brain versus brain stem nuclear proportions and the ways this affects “plastic” mechanisms for specifying neural connection patterns. Because these mechanisms depend on competitive processes that sculpt initially widespread, relatively generic projection patterns, the relative sizes of different brain regions with respect to each other as well as with respect to peripheral systems can significantly influence final circuit organization. Although vocal projections are not the only connections likely to be affected by the significant deviation of human brains from primate proportions, they may include some of the most unusual departures from other mammalian patterns.

Because it derives secondarily from developmental responses to quantitative changes in the brain, some estimate of the age of the human vocal adaptation can also be derived from fossil material. The size of the brain with respect to the body began to shift from ape proportions roughly 2 million years ago and reached modern proportions by at least 200,000 years ago. This suggests that human vocal abilities have been improving for nearly 2 million years and that skilled vocal communication of some form has long been part of hominid societies.

Besides simply accounting for the shift to voluntary fine motor control of phonation in humans, this analysis helps to explain a number of features of the human vocal adaptation: the ability to coordinate tightly the precise movements of muscle systems in the face, mouth, larynx, and diaphragm; low number and variety of innate human calls; reduction in the need for emotional arousal to initiate vocalization; the appearance of early, undirected vocal “play” as in babbling; and the simultaneous expression of emotional tone and arousal level along with symbolic content in speech, that is, prosody. But in addition to explaining some of the interesting functional correlations of normal human speech, it may also shed light on disorders of vocalization as well. For example, we may expect that the very plasticity that made this shift to cortical control of vocalization possible in the first place may also make vocal control more susceptible to individual variations and more resistant to disturbances than if it were more genetically “hard wired.” We may expect to see interaction effects from the two independent competing sources of input control (i.e., limbic arousal and cortical volitional), as appears to be the case in stress-induced mutism and stuttering.

Although an understanding of the nature of neural plasticity in response to damage is of critical concern to those who are attempting to augment recovery
from neurologic insults, it may be misleading to treat the underlying mechanisms as reparative. Understanding that these mechanisms evolved as an expedient for fine-tuning neurologic circuitry during development by taking advantage of contextual information, and the fact that they may only be available for response to damage as an incidental functional side effect can help to focus in on how best to augment the desirable and avoid any undesirable effects.

REFERENCES


**CONTINUING EDUCATION**

**Evolutionary Perspectives on Language and Brain Plasticity**

1. Which of the following is true concerning the development of the brain?
   a. Brain development is dependent solely on the genetic code found in the brain cells
   b. Human brain development is known to involve processes not seen in other animal species that lack higher cognitive functions
   c. Early patterns of cell connections are relatively nonspecific, only gaining specificity as the organism develops
   d. Cells appear prespecified for the functions they later serve.

2. Cell survival within the nervous system:
   a. Is affected by the experience of the organism
   b. Is affected by the functional connections made with other cells
   c. Is affected by competition among cells for axon targets
   d. All of the above
   e. None of the above

3. When immature neurons are transplanted into the brain:
   a. These neurons take on the function of where they were transplanted to, rather than where they came from, in the brain
   b. These neurons take on the function of where they came from, rather than where they were transplanted to, in the brain
   c. These neurons are nonfunctional
   d. These neurons are unable to connect to the preexisting cells, but connect with themselves
   e. These neurons make no connections at all

4. The physical lowering of the larynx in human ancestors had the following ramifications:
   a. Speech became less nasalized
   b. The range of speech sounds that could be produced increased
   c. The larynx became less effective in terms of its function of protecting the airway
   d. All of the above
   e. None of the above
5. Deacon suggests that the fine-grained neural control for speech is an outgrowth of:
   a. Larger brain-to-body size ratio in humans relative to apes
   b. Development of a dual system for vocalization
   c. More extensive descending cortical projections to nuclei that control vocal muscles than in apes
   d. a and b
   e. b and c