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Chapter 51

# Recovery of function: redundancy and vicariation theories

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## INTRODUCTION

Neurologists studying patients and scientists working with laboratory animals have long recognized that recovery of function sometimes appears to occur after brain damage. With more acceptance of the idea that different parts of the cerebral cortex have specialized functions, and with greater survival after brain damage due to improved procedures, 19th-century investigators began to debate the possible underlying mechanisms that might account for what they were observing (Finger, 1978; Finger and Stein, 1982; Finger et al., 1988).

Of course, not everyone agreed that there was recovery in every apparent instance. Indeed, one suggested idea was that subjects were often doing no more than showing behaviors that only superficially resembled those that they were capable of displaying prior to brain damage. For example, a patient or a brain-damaged monkey might still be able to make a tactile discrimination or pick up a cup, but not quite in the same way as before. Hence, it was argued, some reported instances of recovery might be nothing more than compensatory behaviors based on uninjured parts of the brain substituting for the damaged areas in ways consistent with their own specialties (Finger and Stein, 1982, pp. 303–317). With more sensitive tests and more careful observations, it was maintained, this would be apparent.

A second idea rested on a very different possibility – that the lesion had never really destroyed the critical area. The underlying belief in this case was that some of the critical parts for mediating the behavior were only temporarily rendered non-functional by the trauma. From this premise it could be assumed that, if a specialized area or enough of it survived anatomically, there should be some return of the original function as the shock and other distal effects of the trauma dissipated. This idea was central to the thinking of Constantin von Monako(v) (1914), who called his variant of the older shock theory "diaschisis" (Finger and Stein, 1982, pp. 257–270; Finger et al., 2004).

A third theory was based on the assumption that some parts of the brain might be redundant for a given function. Consequently, if one of two functionally equal structures were damaged, the other, perhaps but not necessarily on the opposite side of the brain, would still be present to guide behavior. If no deficit appeared after a brain injury, the "sparing of function" could be due to the redundant structure itself. And if there were a deficit followed by recovery, it could be maintained that the redundant part first had to overcome the distal effects of the brain injury before it again became functional.

The theories of behavioral compensation, "shock," and redundancy share a common feature: none requires rewiring the nervous system in a new way or rearranging the functions normally associated with specific parts of the brain. But during the 19th century, reorganizational theories also came into vogue, and of all the recovery models, the so-called vicariation theories were the most questionable and intensely debated.

The term "vicariation" has its origins in the Latin word *vicis*, from which we get the often-used word "vicarious," meaning substitute. Similarly, the term "vicar" has been used to refer to a priest called upon to fill in for another churchman. In an analogous way, vicariation theorists contended that a brain area not responsible for a specific function could be summoned, reorganized, and possibly even rewired to take over that function after brain damage (Finger and Stein, 1982, pp. 287–302; Slavin et al., 1988; Finger, 1989).

Redundancy and vicariation theories were sometimes viewed as distinct opposites. Nevertheless, these

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two recovery theories can also be considered two ends of a continuum, with most "examples" falling somewhere between the two extremes. For example, redundancy theorists were not always willing to claim perfect equality, whereas vicariation theorists often pointed to substitute structures that might have been involved with the function earlier in life. In fact, most reorganization explanations tended to implicate structures thought to play closely related roles in the function under consideration, veering away from the extremist idea of perfect duplication, as well as the thought that a surviving cortical area associated with one functional system could take on a very different system's unique functions. In reality, the theoretical propositions were rarely black or white; more often than not, they were in shades of gray.

## REDUNDANCY THEORY

In the 17th century, Thomas Willis (1664) looked upon the two sides of the brain as equal, writing that the brain is composed of a double hemisphere and a double substance. He believed this duplicate organization allowed the brain to function even after one side sustained an injury. His thought was not new. In *De Usu Partium*, written in Rome during the 2nd century, Galen had reasoned that the brain is "twinned," like the eyes or the kidneys, so that when one side is damaged the other might still sustain the function in a useful way (Galen, 1968).

During the opening decades of the 19th century, Franz Gall and the phrenologists, who looked upon the cerebral cortex as a collection of specialized organs, continued to rely on the concept of hemispheric redundancy to account for sparing and recovery of function. Like their predecessors, they pondered why unilateral lesions sometimes resulted in profound deficits. If the healthy hemisphere duplicated the injured one, why was there not always a return to normal after unilateral damage? The solution they proposed was that an injury to one side could upset the balance between the two sides. With this logic, they explained the lasting effects of unilateral lesions in some cases, but recovery in others.

In 1844, Arthur Ladbroke Wigan came forth with the novel idea that the skull may house two distinct brains and minds: one on the right side and the other on the left. This represented a significant departure from the time-honored concept of a single mind, which had been tied to the belief in an indivisible, single soul. But, reasoned Wigan, there might be internal conflicts and anarchy with two minds, which is why one hemisphere has to assume a leadership role. The need, as he saw it, was for the two hemispheres to function like an office manager and an assistant. He maintained that this relationship normally develops as a result of experience and education. In the end, at least for healthy people, the two halves of the brain will function as a harmonious unit.

Wigan's thinking about people having two minds had been based on a neurological observation. He observed a man who had one hemisphere severely damaged. Yet this individual still seemed to possess a sound mind.

> *On examining the skull, one brain was entirely destroyed – gone, annihilated – and in its place a yawning chasm. All his mental faculties were apparently quite perfect . . . his mind was clear and undisturbed to within a few hours of his death. He had a perfect idea of his own awful situation.* (Wigan, 1844, pp. 32–33)

Wigan applied his new idea to both madness and amorality. He theorized that disordered states like these arise when the two hemispheres are no longer able to function harmoniously. In fact, not only might a partially damaged hemisphere respond irrationally, it might disrupt higher functions even more than the loss of an entire hemisphere.

In summarizing his thoughts, Wigan wrote:

> *I believe that two brains were bestowed – two perfect organs of thought and volition – each, so to speak, a sentinel and a check on the other . . . The two brains have a subordinate object, no doubt – to provide for the continued exercise of the intellectual faculties when one of them shall be injured or destroyed by disease; but when this is the case the mutilated and helpless victim can scarcely be any longer considered a responsible being – he is reduced to mere animal existence . . . he is incapable of sin, or he is mad – consequently, no longer a moral and responsible agent.* (Wigan, 1844, pp. 297–298)

The views of hemispheric duality accepted by Wigan, the phrenologists, and by most members of the scientific establishment in the first half of the 19th century were in accord with the theories of French histologist François Xavier Bichat. In a book published in 1805, Bichat distinguished between two types of organ. On the one hand, he wrote, there are those that serve "organic life" and the passions (e.g., organs of circulation, generation, and digestion). And on the other, there are those that manage external relationships and understanding (e.g., the cerebral organs and peripheral sensory structures). Bichat maintained that the organs that serve external relations always occur in symmetrical pairs, since duplication allows the organism to deal with the external world equally well with both halves of its body.

Researchers and theorists found no reason to challenge Bichat's (1805) theory of perfectly duplicated cerebral structures prior to the 1860s (see Harrington, 1985, 1986, 1987). Most simply agreed with him and the great anatomist Charles Bell, who had written: "Whatever we observe on one side [of the brain] has a corresponding part on the other; and an exact resemblance and symmetry is preserved in all the lateral divisions of the brain" (Bell, 1811, p. 118). But serious questions began to arise in 1865, when the clinical findings of Paul Broca, Marc Dax, and Gustave Dax showed that the two hemispheres were by no means perfectly identical, at least not for speech functions (Broca, 1865; G. Dax, 1865; M. Dax, 1865; Joynt and Benton, 1964; Finger and Roe, 1996, 1999; Roe and Finger, 1996; see Ch 10).

## CEREBRAL DOMINANCE AND VICARIATION

In the paper he planned to present in 1836, which was based on some 40 patients with left hemisphere lesions and 40 others from the literature, Marc Dax (1865) wrote that left hemispheric damage often affected speech, whereas right hemispheric damage rarely did. He also discussed some examples of recovery from aphasia. Nevertheless, neither he nor his son Gustave, who began to follow up on his findings in the 1850s, directly addressed the possibility of the right hemisphere stepping forth to mediate the recovery process (Finger and Roe, 1996, 1999; Roe and Finger, 1996).

With Paul Broca, it was different. In 1863, he had eight cases of aphasia, all with lesions of the left hemisphere, and all but one having damage in the third frontal convolution of the left hemisphere (Broca, 1863). Statistically, these numbers suggested a rule, yet the notion of hemispheric specialization contradicted Bichat's law, which Broca and those associated with him had always accepted. Jean Laborde, for example, openly doubted that the two hemispheres could be functionally different. In a discussion of a case presented by Parrot in 1863, which involved a lesion of the third frontal convolution on the right side of the brain without loss of speech, he explained that it was hard for him to accept the idea that two parts of the same organ, whose size and detailed anatomy were so similar, might have such remarkably different functions. He added that this "would imply a serious exception to the law of organic duality and functional unity" (see Parrot, 1863, p. 386).

Making the situation even more perplexing, Broca observed that some of his patients showed sparing or recovery of function after damage to the frontal speech area that now bears his name. It was in this context that Broca (1865) postulated that the left hemisphere is normally the more important hemisphere for speech, at least in right-handed people, but that the right hemisphere still has the functional capacity to mediate speech. By moving from a subordinate to a dominant position after damage to the speech region of the left hemisphere, still the speech area on the right side could account for the observed recovery.

The case that more than any other led Broca to think that the right hemisphere could become the leading hemisphere for speech was that of a 47-year-old epileptic woman at the huge Salpêtrière Hospital in Paris. She had been left-handed and, according to Broca, able to read, keep busy, speak fairly well, and even express her ideas without difficulty. But her left sylvian artery was found to be absent upon autopsy, and there was no Foville's gyrus, which included the speech area. "Here," explained Broca in his 1865 paper, "it was perfectly evident that the third right convolution had compensated for the absence of the left" (1986 trans., p. 1069). In fact, Broca compared this woman, who had what he surmised was a congenital defect, to a child born without a right hand. Such a child, he explained, will inevitably grow into adulthood using his left hand skillfully. And so, with the third frontal convolution of her right hemisphere still intact, this woman must have grown up speaking fluently, using what would normally have been a subordinate brain area.

Broca went on to ask himself the same question that the phrenologists before him had asked: why do we not always see sparing and recovery following unilateral lesions, if one hemisphere has the innate capacity to compensate for the other? Indeed, why are there so many aphasics in the Salpêtrière and Bicêtre, as well as wandering the streets of Paris? In translation, he stated the problem as follows:

> *Actually, it seemed that, if the two hemispheres contribute to the function of language, a lesion in only one hemisphere would not be enough to cause aphémie. Just as one can see with one eye, hear with one ear, so one should be able to speak with one hemisphere. Even admitting that the left hemisphere plays a preponderant role in articulate speech (and it is impossible to deny this evidence), it seems that the right hemisphere, when healthy, must always assume the function of speech instead of the left hemisphere that has become powerless because of a lesion . . . How is it, then, that the person who has become aphémique through a partial or total destruction of the third left frontal convolution cannot learn to speak with the right hemisphere?* (Broca, 1865, 1986 trans., p. 1069)

To explain those cases where significant recovery of speech fails to take place, Broca postulated three things. One is that relearning speech is more automatic and easier for a child than it is for an adult ("There are things you can never learn well beyond a certain age"; Broca, 1865, 1986 trans., p. 1069). The second is that caregivers and family members do not spend adequate time trying to teach adults to speak again (an early call for speech therapy). And the third is that cortical lesions caused by strokes and injuries typically extend beyond the boundaries of the speech area to affect intellect:

> *When a lesion is very circumscribed, it could be that language is affected and the intellect remains intact . . . but such cases are rare. More often, the anatomical change is of an extent considerable enough to cause serious impairment to the properly so-called intellect. It follows that most aphémiques have weakened minds, and this condition prevents them from learning to speak exclusively with the right hemisphere, which up to now had played only an accessory role in the function of expression by means of articulated speech.* (Broca, 1865, 1986 trans., p. 1069)

Returning to Bichat, Broca concluded that he was correct when he surmised that the two hemispheres are not "innately" different. But with development, the corresponding speech areas in the two hemispheres grow apart functionally. The clinical data in brain-damaged children and adults reflect this change, as does the emergence of handedness. Thus, on the one hand, Broca's theory of recovery is still tied to the notion of redundancy. But on the other, it is not quite pure redundancy theory, since the two hemispheres develop differently, with the left speech area normally growing into and retaining the leadership position.

## THE BARLOW CASE

Broca's 1865 article on speech is one of the most important papers in the history of neurology. Nevertheless, even his case of the woman with the congenital defect of the left frontal speech area falls woefully short of providing "proof" that the matching region of the healthy hemisphere was mediating her speech. Could it not be argued that a spared part of the left hemisphere, or perhaps even some other gyrus in the right hemisphere, was now mediating fluent speech?

Notably missing from all of Broca's case studies was a second lesion of the homologous part of the right hemisphere – a focal lesion that would have no lasting effect on fluent speech if the left hemisphere were intact, yet one which would render a recovered aphasic patient speechless again – possibly without any further recovery.

In 1877, a case that seemed to meet some of these criteria was described. The salient features of the new case were: (a) loss of speech after a focal left hemispheric injury centered in Broca's area, (b) subsequent recovery, and (c) loss of speech with no recovery after a second lesion situated in the homologous area of the right frontal lobe. The physician presenting the case in the *British Medical Journal* was Thomas Barlow, who had an appointment at a London hospital for sick children.

Barlow's "remarkable" case involved a 10-year-old boy. He initially exhibited a paralysis on his right side and loss of speech, which left him only able to say "haw-haw." But, as stated in Barlow's published report, he exhibited fairly good recovery:

> *In ten days, he was greatly improved. The leg improved before the arm. The speech had returned on the tenth day; but occasionally he made a mistake, gave the wrong name for a boy, and did not seem always quite to understand what was said to him.* (Barlow, 1877, p. 103)

About a month later, he was again able to run errands. But 4 months after his first attack, he suffered another episode that affected the voluntary muscles on the left side of his body, now signifying right-hemispheric involvement. With regard to his speech, Barlow wrote:

> *The only approach he could make to a voluntary articulate sound was "Ah." He could cry vigorously; there was no lack of voice. From his admission, he appeared to understand all that was said to him. When asked his age, he counted ten on the questioner's fingers.* (Barlow, 1877, p. 103)

The boy died less than 2 months after this incident. An autopsy revealed "vegetations" that calcified the heart valves and blocked some of his arteries, two of which were in the brain and caused focal brain damage. The most unusual feature of the case was that the hemispheric lesions were symmetrical; one involved the lower motor cortex and Broca's area on the left side, and the other severely damaged the corresponding areas on the right side. Notably, the circular lesion of the left hemisphere looked older than the one on the right hemisphere.

Barlow did not argue that the right hemisphere took over the speech functions normally mediated by Broca's region. Instead, he attributed the boy's speech problems to innervation of the midline facial muscles, which can be controlled by either the left or the right motor cortex. He theorized that, because of the

redundancy, the right motor cortex took sole control of the bilateral musculature after the left hemisphere's mouth region was damaged. The initial speech problems that abated, he contended, could have been due to distal or secondary effects of the first lesion wearing off. After the right cortical facial region was damaged, all circuitry controlling these muscles was lost, and the boy was left "irretrievably deficient."

Many people cited Barlow's publication, not knowing that it differed in some significant ways from the Great Ormond Street Hospital notes of the case (Hellal and Lorch, 2007). Yet not everyone followed his explanation, which emphasized duplicate or redundant brain parts for controlling the muscles of the mouth, without bringing anything like vicariation into the picture (Finger et al., 2003).

For example, in his widely read *Manual of Diseases of the Nervous System*, William Gowers used language that seemed to suggest some sort of a functional takeover. He wrote that, if the normal speech structures on the left side of the brain are destroyed,

> *the corresponding parts of the right hemisphere may take on the lost functions, and the symptoms of the loss may slowly pass away. The proof of this is that, in several cases of this character, a fresh lesion in the right hemisphere has destroyed the reacquired power, and there has been no recovery . . . If recovery occurs from an organic lesion that destroys the motor speech-centre, the power of speech seldom remains absent for more than a few weeks, compensation by the right hemisphere occurring with great readiness.* (Gowers, 1893, pp. 111, 124)

James Taylor (1905), author of *Paralysis and Other Diseases of the Nervous System in Childhood and Early Life*, was another physician who knew about the case. Like many others at the time (e.g., Bastian, 1898), he first divided recovery into two categories. One was restitution or restoration of function in only temporarily affected parts of the brain. This, he contended, often occurs within a week or two, and it can be accounted for by a renewed blood supply, reduced pressure, diminution of shock, and related factors.

The other mechanism of recovery, Taylor maintained, involves transference of the function from the damaged area to another part of the brain. He called this "functional compensation," and argued that it can do a better job accounting for recovery that takes more time to occur. He further stated that the takeover of a function by a neighboring or related area in the same hemisphere, or by the "corresponding locality in the opposite hemisphere," is most likely to occur in young children. In his words, it is more commonly observed "before the various regions of the cortex are indelibly stamped with a special function, provided that there is a sufficient area of normal cortex remaining" (Taylor, 1905, p. 221).

In this context, Taylor noted that motor aphasias are almost never permanent with unilateral lesions in children less than 10 years of age. Because the boy described by Barlow had already had his 10th birthday, Taylor presented the case as his first example of transference of function from one part of the cerebral cortex to its corresponding part of the opposite hemisphere. To quote: "As an example of compensation by the opposite hemisphere the well-known case recorded by Sir Thomas Barlow is most illustrative" (1905, p. 218).

Since Gowers and Taylor authored the leading English textbooks of neurology and child neurology, respectively, it is easy to understand why their contemporaries would now cite the Barlow case as evidence for vicariation. Indeed, the case would be cited for decades to come as the best proof yet for one area taking over the functions of another (e.g., Henschen, 1920–1922; Nielsen, 1946).

Still, the records show that there were dissenters. The major problem, at least as Henry Charlton Bastian (1898) saw it, was the short time period for recovery after the first lesion. "I very much doubt," he wrote, "whether complete transference of function could possibly have occurred in the short period of ten days." Bastian also wondered about the handedness of the boy. For all we know, he continued, "the right hemisphere might have been the leading hemisphere for speech, and the first lesion on the left side may have merely occasioned some functional disability in the right centre, from which in a very short time he recovered" (Bastian, 1898, p. 322).

Barlow's case was interesting, and certainly worthy of attention, but it did not provide the unequivocal proof Bastian was seeking to convince him that this was an instance of functional takeover. (For more on Barlow's case and what has been discovered with brain scans on recovering aphasic patients, see Finger et al., 2003.)

## RESEARCH WITH LABORATORY ANIMALS

Many laboratory animal researchers rejected the idea that the damaged nervous system may have an unlimited capacity to reorganize, after the theory of cortical localization of function was accepted. Some ardent localizationists also had considerable difficulty believing that even related areas, such as a homologue on the right side, could acquire precisely the same functions as a damaged area.

David Ferrier, the leading British experimentalist of Barlow's day, and the scientist who had encouraged him to write up his case study, was never willing to accept the idea that surviving structures could take on new and unusual functions. As he saw it, the

> *hypothesis is altogether inconsistent with the theory of specific localization of function. If we were to suppose it possible that the functions of the leg centre could be taken up by the neighbouring occipito-angular region, we should have the very remarkable substitution of a motor by a sensory centre. Such a mode of interpretation is no more justifiable than the supposition that the organ of vision may take up the functions of the organ of hearing . . . or perform both functions at once.* (Ferrier, 1886, pp. 368–369)

Many of Ferrier's British colleagues, including Barlow, agreed with him. Consider the case for the opposite cortex taking over motor functions. This idea was tested experimentally by subjecting animals to sequential lesions of the motor cortex, first on one side of the brain and then, after a few weeks, on the other side. The paradigm followed that of the Barlow case, although now the lesions were made deliberately and speech was not a dependent variable. The specific question asked was whether the second lesion would be associated only with contralateral motor deficits, indicative of no takeover of function, or whether bilateral motor deficits would appear, suggesting (but not necessarily proving) vicariation.

Early on, some individuals, including Eduard Hitzig who had identified the motor cortex with Gustav Fritsch in 1870, reported that the deficits found after the second lesion were strictly contralateral (see Carville and Duret, 1875, for a review of early findings). Nevertheless, it was the highly regarded work of Charles Sherrington that proved most harmful to the theory of vicariation.

Sherrington and his colleagues published a series of carefully controlled stimulation and lesion studies on the primate motor cortex early in the 20th century (e.g., Grünbaum and Sherrington, 1901, 1903; Sherrington and Grünbaum, 1901). The most complete paper in the series appeared in 1917, and it summarized findings from 28 great apes (Leyton and Sherrington, 1917).

One part of this paper described an ape that had exhibited considerable recovery of motor function after a well-defined lesion of the left cortical arm area. Yet the researchers reported no return of the defect or worsening of the residual deficit after the right cortical arm area was ablated 2 months after the first operation. "The double arm area lesion showed clearly that the regaining of ability to use the limb could not be attributed to the arm area of one hemisphere taking over the functional powers of the arm area of the other hemisphere after the latter's ablation" (Leyton and Sherrington, 1917, p. 207).

This paper also addressed the possibility that nearby sites on the same side as the damaged area could take over the function. This hypothesis drew even more attention than the opposite hemisphere hypothesis when unilateral lesions were small or when bilateral lesions were followed by some recovery. Friedrich Goltz (1888), who worked in Strasbourg, entertained this hypothesis after he observed the effects of making sequential operations on the same side of the brain in his dogs. Each lesion was followed by the reappearance and eventual remission of symptoms. But others, after conducting stimulation and ablation experiments of their own, questioned this finding (see Dodds, 1877–1878).

Sherrington and his coworkers found that electrical stimulation of nearby cortical areas involved with the elbow, the shoulder, and other parts did not produce arm movements before or after behavioral recovery. They also found that additional ablations near the damaged arm region did not reinstate the deficits. They even went on to examine whether the sensory cortex just behind the motor strip might be mediating the recovery. But neither electrical stimulation nor additional lesions provided any evidence for the somatosensory cortex taking on these motor functions. In short, Sherrington and his colleagues found no evidence for vicariation theory.

Sherrington's reports made many theorists pause, because he was regarded as the most talented experimentalist of the era, and because he worked on apes, which were closer than monkeys to human beings. His work on great apes reinforced the fact that conclusions about underlying mechanisms based solely on clinical observations can be dangerous.

## LASHLEY, FRANZ, AND VICARIATION THEORY

Although Sherrington's work hurt the idea that one part of the brain could substitute for another, it failed to kill the concept of vicariation. The notion still had its share of vocal supporters, although fewer and fewer people continued to believe that recovery could be mediated by structures having nothing in common with the damaged structure.

Karl Lashley, the best-known biological psychologist in the first half of the 20th century, did not deny that there were specialized sensory and motor areas of the cortex. In 1938 he wrote that only focal cortical damage involving the primary visual areas could affect

previously learned, simple visual discriminations. But, he continued, the lost visual habits could be relearned in the same number of trials that it took for original learning. From this repeated observation, Lashley drew two conclusions. First, the visual cortex must play an important role in visual memory under normal conditions. And second, other areas have the potential to mediate visual learning, if the visual cortical areas are damaged.

But where might the new learning take place? To find out, Lashley made focal lesions in surviving cortical areas. Yet he was unable to reinstate the visual learning deficits. In fact, it was only after damaging the midbrain tectum that the deficit re-emerged. These findings led Lashley to conclude that, at least for sensory and motor functions, the cerebral cortex does not have an unlimited capacity for vicariation. Instead, any recovery is likely to be mediated by the system "which is more or less directly concerned with the same function under normal conditions" (Lashley, 1938, p. 741).

It is interesting to note that, although Lashley worked with Shepherd Ivory Franz when he began to study the effects of brain lesions in 1916, he looked upon Franz as a much freer supporter of unrestricted vicariation than himself. Two decades later, he even wrote that some students of the problem of vicariation, "like S.I. Franz, have believed that almost any part of the brain might take over the functions of other parts and that nervous structure sets almost no recovery limits" (Lashley, 1938, p. 740).

## RETURNING TO THE AGE FACTOR

In contrast to the conclusions being reached with mature subjects, analogous work with very young animals proved to be another matter (Finger and Stein, 1982; Almli and Finger, 1984; Finger and Almli, 1984, 1985, 1988; Finger, 1991). In 1876, only 6 years after Fritsch and Hitzig discovered the motor cortex, Otto Soltmann published a series of informative stimulation and lesion experiments on the developing motor cortex of the dog (Soltmann, 1876; Finger et al., 2000).

In one experiment, Soltmann made a lesion of the left motor region in a 6-day-old puppy. This animal still appeared to walk fairly normally. When it was 3 months of age, he electrically stimulated its right motor cortex and found that he was able to elicit bilateral limb movements. When the same experiments were conducted on dogs with lesions made after the first few weeks of life, or for that matter on adult dogs without brain damage, only the contralateral movements were elicited. Soltmann's conclusion was that, if the lesions could be made early enough, one hemisphere could retain control over the movements on both sides of the body.

Some of the paradigms used by Soltmann with his newborn dogs were in themselves "taken over" during the 1930s by Margaret Kennard at Yale University. Kennard shared Soltmann's interest in motor circuitry, and she showed that monkeys and apes could also exhibit considerable functional sparing and recovery after motor cortex lesions early in life (Kennard, 1936, 1938, 1940, 1942; Kennard and Fulton, 1942; Kennard and McCulloch, 1943; also see Finger and Almli, 1988; Finger and Wolf, 1988; Finger, 1999).

Following up on the recovery she witnessed after early unilateral lesions, Kennard reported atypical bilateral effects after the opposite motor cortex was ablated later in life. She also documented motor problems after the somatosensory cortex was damaged, but only in those monkeys that sustained motor cortex damage in infancy. Prior to receiving their additional lesions, electrical stimulation of the somatosensory cortex had also elicited motor responses in these animals.

Kennard's data suggested that some functional takeover might occur in the opposite motor cortex, or in neighboring cortical areas not normally thought of as motor in nature, if the motor cortex is damaged early in life. But in a review of her findings, she made the point that she did not construe her findings to be evidence for vicariation (Kennard and Fulton, 1942).

This was because Kennard defined vicariation in the strictest possible sense – as the takeover of a new and unusual function by an unrelated part of the brain. But, she noted, the somatosensory cortex of the ape contains some large pyramidal cells like those found in the motor cortex. Partly for this reason, she maintained that the motor and somatosensory areas really should be considered a single functional unit. From this premise, she concluded that the "reorganization" does not take place in unrelated parts of the brain, but within the same system – in this case, within the partially damaged "frontal-parieto system." She also postulated that new dendritic growth might contribute to the "within-system" functional reorganization.

## THE TANGLE OF SEMANTICS AND CONCLUSIONS

One of the difficulties facing people interested in sparing and recovery of function is that different authors have used the same terms in different ways, as well as different terms with the same intent (see Almli and Finger, 1988). Sometimes, the choice of words is dictated by theoretical preconceptions, sometimes by what the data suggest, and sometimes by reasons that are just not easy to understand.

The problem is evident in the writings of Margaret Kennard. She argued for "reorganization of function,"

but preferred not to use the term "vicariation," because she believed that reorganization of motor function occurred only within the same, broadly defined system. For those unwilling to accept how Kennard defined her cortical motor system, or strict definition of vicariation, her work could be cited as evidence for functional takeover. Indeed, after the motor cortex had been destroyed on one side of the brain, Shepherd Ivory Franz and his associates had used the term vicariation to refer to the use of pyramidal projections from other cortical areas to mediate recovery (e.g., Ogden and Franz, 1917).

Even today, there is less than perfect agreement about how a functional system should be defined in the brain. This is because it is not always easy to determine what a structure is doing in the normal brain. In addition, theorists can argue over possible degrees of involvement without ever coming to a consensus.

Developmental and experiential histories can make the terms redundancy and vicariation even more problematic (Norrsell, 1988). The prevailing view that both hemispheres might be mediating speech early in life could mean that the right hemisphere is really not "taking over" a new or unusual function after an early left hemispheric injury, at least to some people if not to others. In contrast, with comparable lesions later in life, some individuals might choose to make the case that hemispheric inequality would meet the key demand for some sort of functional takeover, while others would staunchly disagree.

The fact that confusions and contradictions have characterized the topics of vicariation and reorganization of function over their checkered histories should not be surprising when issues of semantics, development, and data from only indirect tests of these theories are considered. What would really be surprising would be if there were general agreement, given how much still has to be learned about normal brain functioning at different times in life, coupled with the problems inherent in brain lesion studies, and the strong theoretical biases held by many researchers.

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