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Spontaneous Recovery

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Synonyms

Plasticity

Definition

The natural redevelopment of or improvement in function following insult or injury to the nervous system.

Current Knowledge

Spontaneous recovery generally occurs following acute, nonprogressive, neurological insults such as strokes (either hemorrhagic or occlusive), closed head trauma, tumor resection, and anoxia. The mechanisms underlying behavioral or functional improvements depend, in large part, on the nature of the original pathology.

The many theories about how the brain recovers from such insults may be thought of as falling into one of two broad categories, for example, those that attempt to explain either acute (short-term) or long-range recovery. Some common mechanisms associated with acute recovery likely involve edema and other forms of increased pressure causing temporary suppression of function. Edema can result from a breakdown of intracellular processes or *cytotoxic edema* (as is common in infarcts), a disruption of the blood-brain barrier or *vasogenic edema* (as often occurs in brain tumors and traumatic brain injuries), or as a result of disruption of CSF circulation (*interstitial edema*). Pressure can also be exerted on brain structures by the accumulation of blood, either directly inside brain tissue or simply within the cranial cavity as a result of hemorrhagic lesions. Such lesions may be primary, as might result from hypertension, or secondary, as seen in closed head injury (e.g., subdural or intraparenchymal hematomas). Whether as a result of edema, hemorrhage or brain tumor, the resulting pressure on brain tissue, either local or generalized, can disrupt normal neuronal function. While extensive increased intracranial pressure can be fatal, in nonfatal cases it resolves over time, resulting in at least partial restoration of function. In addition to exerting local pressure on brain tissue, intraparenchymal blood has a toxic effect on neurons themselves until reabsorbed.

Immediately following an injury to the brain, acute changes in metabolic functions and a disruption of neurochemical processes can contribute to behavioral disturbances. In addition to a disturbance of normal neurochemical pathways, an excess release of glutamate can lead to increases

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in intracellular calcium and production of oxygen free radicals, all of which can contribute to excitotoxicity and subsequent cell death. The eventual stabilization of these processes can also provide a basis for recovery.

Another example of short-term spontaneous recovery is that provided by transient ischemic attacks (TIAs) where return of function is thought to result from spontaneous resolution (in whole or in part) of circulation to the affected brain area. While perhaps not strictly constituting "spontaneous recovery," similar effects can often follow the prompt administration of thrombolytic drugs following ischemic strokes.

Functional recovery can continue for days or weeks, possibly for years. The mechanisms behind these long-term changes are less well studied. In the case of strokes, anoxia, or other types of brain injury, it is suspected that different levels of damage may occur. For example, at the nidus of an infarction, there may be a total loss of blood supply (e.g., glucose and oxygen) to the neurons resulting cell death, eliminating all possible recovery utilizing those cells. However, surrounding the area of total infarction, an incomplete loss of circulation may result in cells that are rendered temporarily dysfunctional. This region is often referred to as the *ischemic penumbra* and the phenomenon as the "*idling neuron hypothesis*."

Another theory behind long-term recovery is that over time some revascularization occurs that enables damaged neurons to function more efficiently. The concept of "dendritic sprouting" and the resulting establishment of new synaptic connections that has been offered to explain recovery in such damaged areas, although new, long axonal connections are not thought to develop.

The practices of rehabilitation medicine and cognitive rehabilitation appear to be largely based on other premises. It is believed that, following damage to a portion of the brain, at least two things might happen. The first is that other areas of the brain may assume the function (s) of the damaged cortex or that new neuronal pathways are recruited to carry out a particular function. The consequences of early loss of language or sight appear to support this idea. When language is lost as a result of childhood brain injury, the recovery or reemergence of language skills is usually much better than when such loss occurs later in life. Similarly, individuals who lose their sight appear to develop enhanced acuity of other sensory modalities, allowing them, for example, to become proficient in Braille. Such recoveries or enhanced proficiencies are attributed to a phenomenon known as *neuroplasticity.*

A second premise behind rehabilitation programs is that the patient learns new techniques, strategies, or other compensatory mechanisms to carry out functions impaired by the brain lesion. While these latter two mechanisms of recovery likely occur spontaneously, repeated, facilitated, or guided practice may be useful.

Cross-References

▶ Diaschisis
▶ Ischemic Penumbra

References and Readings

Carmichael, S. T., & Chesselet, M. (2002). Synchronous neuronal activity is a signal for axonal sprouting after cortical lesions in the adult. *The Journal of Neuroscience, 22*, 6062–6070.

Dobkin, B. (2003). *The clinical science of neurologic rehabilitation*. New York: Oxford University Press.

Finger, S., Koehler, P. J., & Jagell, C. (2004). The Monakow concept of diaschisis: Origins and perspectives. *Archives of Neurology, 61*, 283–288.

Fisher, M. (2004). The ischemic penumbra: Identification, evolution and treatment concepts. *Cerebrovascular Diseases, 17*(Suppl. 1), 1–6.

Kennard, M. A. (1938). Reorganization of motor function in the cerebral cortex of monkeys deprived of motor and premotor areas in infancy. *Journal of Neurophysiology, 1*, 477.

Kertesz, A., & Gold, B. T. (2003). Recovery of cognition. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (pp. 617–639). New York: Oxford University Press.

Kohn, B., & Dennis, M. (1974). Patterns of hemispheric specialization after hemidecortification for infantile hemiplegia. In M. Kinsbourne & W. Lynn Smith (Eds.), *Hemispheric disconnection and cerebral function*. Springfield: C.C. Thomas.

Kolb, B., & Gibb, R. (2007). Brain plasticity and recovery from early cortical injury. *Developmental Psychobiology, 49*, 107–118.

Kolb, B., Gibb, R., & Robinson, T. E. (2003). Brain plasticity and behaviour. *Current Directions in Psychological Science, 12*, 1–5.

Reilly, J. S., Levine, S. C., Nass, R., & Stiles, J. (2008). Brain plasticity: Evidence from children with perinatal brain injury. In J. Reed & J. Warner-Rogers (Eds.), *Child neuropsychology: Concepts, theory, and practice* (pp. 58–91). Malden: Wiley-Blackwell.

Robertson, I. H., & Murre, J. M. J. (1999). Rehabilitation of brain damage: Brain plasticity and principles of guided recovery. *Psychological Bulletin, 125*(5), 544–575.