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# The Monakow Concept of Diaschisis

## *Origins and Perspectives*

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The idea that damage to one part of the nervous system can have effects at a distance was popular during the 19th century. Constantin von Monakow, MD, accepted this idea and blended it with the newly formulated neuron doctrine early in the 20th century to account for ipsilateral paralyses and recovery of function. He called his theory of neural depression caused by loss of inputs to structures tied to the damaged area *diaschisis*. In this article, we examine the origins of diaschisis and the goals of Monakow. Credit is given to Monakow for drawing needed attention to the dynamics of the nervous system, remote lesion effects, and recovery of function, even though the fine details or specifics of his theory have had a mixed reception.

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During the 19th century, it was speculated that damage to one part of the nervous system might have temporary depressive effects on the same or opposite side of the brain or even at another level. It was not until early in the 20th century, however, that ideas about remote lesion effects became more specific. Most scientists and practitioners agreed that some temporary "shock" effects could be due to edema and pressure on surviving brain tissue and to changes in blood flow. But now there was another possibility—that simply breaking neuronal ties between regions could also produce temporary impairments of function. The person most responsible for discussing shock effects in this context of the neuron doctrine was Constantin von Monakow (**Figure**). His new theory was called *diaschisis*.

This historical review presents biographical information on Monakow, examines the origins of his new ideas about transient lesion effects, and relates them to cortical localization and recovery of function. It will be shown that the specifics of the Monakow diaschisis theory have always been questioned. Nevertheless, it will be maintained that his more basic notion of temporary depressed neural functioning caused by damage at a distance is still an attractive concept to those hoping to account for recovery of function.

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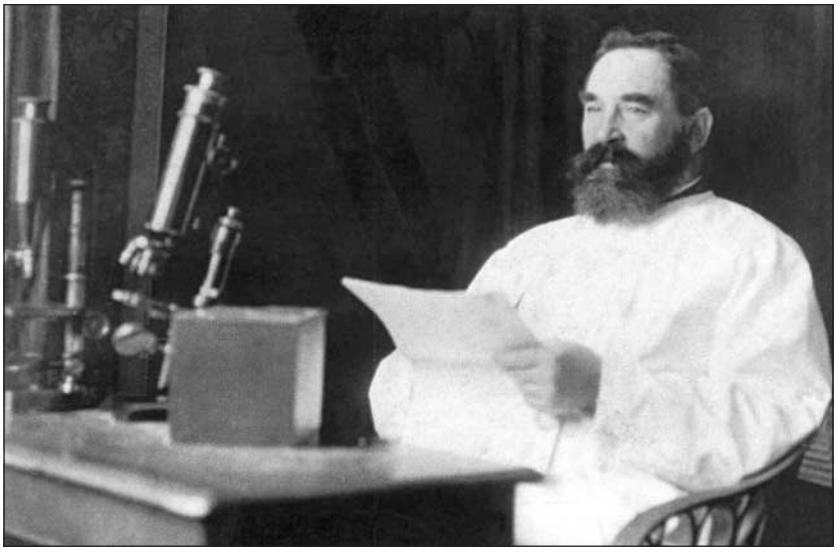
### EARLIER SHOCK THEORIES

Monakow was not the first physician to emphasize that damage to the nervous system could have effects at a distance. Galen[1] contrasted the primary and secondary effects of disease in his treatise *De Locis Affectis*. Writing in Rome, Italy, during the second century, he maintained that interconnections among certain nerves allow animal spirits to travel from one internal organ to another, effectively spreading the message so they can respond in sympathy. The very term *sympathetic nervous system* stems from the idea of Galen that some nerves are especially suited for inducing physiological reactions at a distance from the affected part.

Descriptions of secondary brain lesion effects can be found in many publications from the beginning and middle decades of the 19th century. For example, in 1861, John Call Dalton, MD,[2] an American who attempted to replicate and extend the findings of Flourens[3] on the effects of cerebellar lesions, noted that pigeons with cerebellar damage often



Constantin von Monakow, 1853-1930.

showed severe motor incoordination but then excellent recovery. He thought this finding could be explained in either of 2 ways. The first was that remaining parts of the cerebellum may have taken over for the damaged parts, and the second was that the lesions could have temporarily depressed the remaining parts of the cerebellum. Spared parts of the cerebellum probably mediated motor coordination but only after the remote effects of the lesion wore off.

One of Dalton's contemporaries was Jakob Henle, MD.[4] In 1840, he discussed 3 types of remote lesion effects using the spinal cord as a model. Henle referred to them as symmetrical consent between the nerves of the different sides, ascending and descending consent within the same columns (anterior or posterior), and consent between sensory and motor columns. Within this framework, he systematically discussed how stimulation of one area might decrease or increase the responsivity of another spinal part.

Monakow might not have read Henle,[5] but he was familiar with the ideas of Charles-Edouard Brown-Séquard, MD,[6] who used distant lesion effects to question certain conclusions about cortical localization of function during the 1870s. Brown-Séquard argued that "an irritative action, transmitted at a distance, on a center besieged with that function" may produce "in this same center an inhibitory action."[7(p424)] He distinguished between inhibitory and excitatory reactions at a distance, calling them *inhibition* and *dynamogénie,* and he studied these remote effects not just in the clinic but in the laboratory.[8-10]

Monakow also knew about the theories of Friedrich Goltz, MD.[11] Goltz also wrote about how irritation from a lesion site could functionally disrupt healthy, remaining parts of the brain. Hence, the work of researchers such as Dalton, Henle, and Brown-Séquard, not to mention others, helped to provide some of the zeitgeist for Monakow, who viewed the nervous system as a dynamic entity and wished to know more about transient lesion effects.

## CONSTANTIN VON MONAKOW

Monakow was born in Bobrezowo, Russia, a town north of Moscow, in 1853.[12-22] His mother died when he was 4 years of age, and his father, who was a difficult and religious man, took responsibility for his upbringing. The Monakows moved to Dresden, Germany, for political reasons when Monakow was 10 years of age. They next moved to Paris, France, before finally settling in Zurich, Switzerland, in 1866. Three years later, they became naturalized Swiss citizens.

Monakow was headstrong at home and in the classroom and went on to study medicine in Zurich against his father's wishes. In 1876, while still a medical student, he was encouraged by Eduard Hitzig, MD, who was then head of the Burghölzli Asylum, Zurich, and interested in institutional management, to visit a number of asylums and laboratories in German-speaking countries. Hitzig's suggestion led him to the laboratory of Bernhard von Gudden, MD, in Munich, Germany, where he learned to use the microtome and was introduced to new techniques for studying secondary degeneration after cortical lesions. Many of the early experiments of Monakow were based on what he learned from Gudden and were carried out on young animals.[23]

In 1885, after spending 7 years as an assistant at the St Pirminsberg Asylum at Pfäfers, Switzerland, he established his own research institution in Zurich. Its purpose was to study secondary brain degeneration in humans, and it was paid for by personal funds from his private practice. He became *Extraordinarius* (associate professor) at the University of Zurich a little less than a decade later (against the wishes of the faculty!), and his Hirnanatomisches Institut (Brain Anatomy Institute) was then merged into the university.

Following the model provided by Mieczyslaw Minkowski, MD,[15] his biographer and successor in Zurich, the professional career of Monakow is typically divided into 3 parts. During the first period, which began around 1880, he performed many elegant experiments on developmental anatomy and on how cortical and thalamic areas are related. At this time, he added significantly to what was known about the thalamic nuclei involved with vision and hearing and cortical localization of function. He published his *Gehirnpathologie*[24] (Brain Pathology), a massive text with more than 3000 references, in 1897, near the end of the first period.

During his middle period, which started around 1900, his interest in traditional anatomical procedures for studying localization waned and he became considerably more involved with brain dynamics and functional issues. It was at this time that he wrote about levels of functioning, "chronological localization" (the role of time), the interplay of the parts within the nervous system, and diaschisis. The major publication that completed this phase of his professional life was his *Die Lokalisation im Grosshirn und der Abbau der Funktion durch kortikale Herde*[25] (Localization in the Cortex

and Breakdown of Function Through Cortical Lesions), which appeared in 1914. (Unless otherwise specified, what Monakow had to say about diaschisis comes from this source.)

The final period of Monakow's professional life was strongly affected by the events of World War I (1914-1918) and the Russian Revolution of 1917. Although not in the trenches, he was appalled by the senseless destruction of people and things, which he believed reflected spiritual degeneration.[14] He became depressed and retreated from his neurology and friends in Zurich to the Swiss mountains. There he devoted his time to blending his holistic ideas about biology with his ethical and philosophical beliefs.[14]

Monakow retired from the university in 1928 at 75 years of age but still devoted himself to understanding his hypothetical self-actualizing force *(horme),* conscience, the basis of good and evil, the pitfalls of modern civilization, and spiritual regeneration until his death in 1930. Three years later, a biographical piece was published about him by Swiss novelist Maria Waser,[26] to whom he had turned for help on how best to express his most spiritual thoughts in writing.

## DEVELOPMENT OF DIASCHISIS THEORY

Monakow wanted to differentiate between the deficits that result directly from focal brain lesions and the transient effects of such lesions, which he believed could be attributed to depression of neurally connected but distant parts of the brain. When he coined the term *diaschisis,* it was specifically to describe the injury-induced drop in activity in functionally connected brain areas. Diaschisis was never intended to describe anything more than the depressive effects of breaking connections between functionally related parts of the brain. The word itself came from the Greek *schizien* (to split) and has been variously translated as "splitting off" or "separated."

The Monakow concept of diaschisis was based on the notion of a working unit with parts that must cooperate to assure normal functioning.[25(pp27-30)] But it also had roots in localization theory and in neuron theory, which explained how the different parts comprising these working units are connected. The basic idea to emerge was that damage to one part must have disruptive effects on other parts, which may later wear off and be associated with some recovery of function.

It was during the late 1890s that Monakow began to emphasize the importance of transient, remote lesion effects. At that time, however, the basic mechanisms that he thought could account for these effects differed from the one that would later characterize his diaschisis theory. His evolving ideas can be appreciated by comparing his statements from 1897 with his more mature formulations from 1914.

Monakow attempted to explain ipsilateral hemiplegia in the 1897 edition of his *Gehirnpathologie*.[24(pp292-296)] He wrote that ipsilateral paralyses occur immediately after injury, in a period he called *the reaction period*. He also discussed a *stage of chronic symptoms*, observed after the transient lesion effects diminish.[24(p756)] He reasoned that the reduction of most secondary symptoms is due to the release of pressure, reduction of edema, and other mechanical factors.[24(pp208,209)] He did not use the term *diaschisis* and only hinted at his later theory when he attempted to account for recovery of finger movements, the last function to return on the ipsilateral side.

The term *diaschisis* was introduced by Monakow in 1902. By 1905, when the second edition of his *Gehirnpathologie*[27] was published, readers found a small chapter with a title that could be translated as *shock and diaschisis*. Here he mentioned flaccid paralysis (an initial symptom) preceding spasticity (a residual symptom) due to a shock-like disconnection.[20(p33),27(p245)]

One year later, he gave a talk on aphasia and diaschisis at the Neurological Section of the Stuttgart Naturalist Meeting.[28] Then, from 1906 to the end of World War I, the theory of diaschisis underwent further refinements as it rose to represent a dominant theme in his dynamic and holistic neurology.

As Monakow developed his concept, he made it clear that the type of effect he was talking about was only an "abolition of excitability" or a "functional standstill." He never wanted his theory to be associated with neural inhibition.[25(pp66,67)] In fact, he had a hard time even accepting the concept of inhibitory fibers or inhibitory synapses. In addition, diaschisis was not to be equated with irritation, a mechanism deemed important by some of his predecessors, including Brown-Séquard and Goltz.

Thus, in 1914, Monakow defined diaschisis as:

> An "interruption of function" appearing in most cases quite suddenly . . . and concerning widely ramified fields of function, which originates from a local lesion but has its points of impact not in the whole cortex (corona radiata, etc) like apoplectic shock but only at points where the fibers coming from the injured area enter into primarily intact grey matter of the whole central nervous system. . . . Speaking quite generally, the process of diaschisis may be regarded as being caused by abolition of excitability (functional standstill) due to local disruption of brain substance within one neuron group, which is transmitted to neuron groups closely adjacent to and directly related with the afflicted part of the brain.[29(pp28,29)]

The insights of Monakow led him to argue that localization of symptoms and localization of function should not be equated, as they tended to be by some map makers.[20(p34),30] Diaschisis also gave him the key he needed to understand a variety of neurological problems and phenomena. One riddle that suddenly seemed solvable had to do with acute right-hemispheric lesions impairing speech, at least for a while, in right-handed people. Now, by thinking of effects at a distance,—in this case in the mirror locus on the opposite hemisphere—severe but transient impairments of speech could be better understood.[28]

Monakow was always very insistent about how the word *diaschisis* should be used. He pointed out that diaschisis has only limited focal consequences (such as loss of voluntary movement in one leg), leaving the vital functions intact. In contrast, circulatory changes and edema, which may also be associated with traumatic injuries, have more global and less localizable effects, which may

include changes in respiration, pulse, and consciousness. In 1914, Monakow explained:

> There is no doubt that the development of diaschisis is enormously enhanced by latent circulatory disorders in the affected as well as in other areas of the brain (vasomotor disorders, pressure effects, or accumulation of CSF, toxic factors, etc). These disorders *per se*, however, are never able to bring about a functional disorder in the sense of local initial symptoms.[29(p32)]

Hence, Monakow never denied the importance of edema, pressure on the brain, circulatory changes, or other dynamic events that can follow brain lesions and cause effects at a distance. These factors, he noted, are important, and they may add significantly to the clinical picture. But they can and must be distinguished from diaschisis, which is neurally mediated and has specific focal consequences; a point that is sometimes overlooked in books and articles, especially where the specific term he coined has been used generically and interchangeably with *shock effects*.[31,32]

## LOCALIZATION THEORY AND DIASCHISIS

Monakow was far from convinced that brain lesions could serve as a reliable guide for localizing functions. In part, this was because he thought the highest functions of the brain, such as associative learning, creativity, and abstract thinking, were personal or idiosyncratic. As a result, they may involve a dynamic interplay among different brain parts in different people.

For sensory and motor functions, the situation was different. Monakow believed that after an injury of such an intricate mechanism as the mammalian brain, other structures that are anatomically related to the region destroyed must be affected. Hence, if anything at all is to be gleaned about localization with the lesion method, it must come from the study of residual symptoms after diaschisis and the more general effects of the lesion have dissipated.[30(p238)] But, he warned, even this is dangerous. Since other brain areas would now be deprived of some connections, one cannot dismiss the possibility that alterations in distant areas could be contributing to the residual symptomatology. Regarding the future of localization theory and the role played by diaschisis theory, Monakow had this to say: "However the problem of localization will develop in the future, I am convinced that it will not be able to do without the dissociation of function by diaschisis or a similar conception."[30(p250)]

It is worth adding that Monakow had little use for vicariation theory, which was also popular at the time. On purely logical grounds, he could not comprehend how brain areas could take on new and unusual functions without severely affecting their own specialized functions. He did not dismiss vicariation outright when trying to account for recovery long after brain damage. But he always made it clear that the wearing off of diaschisis is a more reasonable and parsimonious explanation for recovery than any hypothetical "morphological reorganization."

## CAUSES AND TYPES OF DIASCHISIS

Signs of diaschisis, which are most obvious with sudden injuries and strokes, are less noticeable with fast growing tumors and are usually absent with slowly growing neoplasms. Recognizing this difference, Monakow initially reserved the term *diaschisis* for the effects caused by lesions of sudden onset. Indeed, his theory was largely based on cases of trauma, hemorrhage, and other acute types of brain insult.

Monakow introduced different terms for different types of diaschisis. For example, diaschisis could proceed from the cerebral cortex to the spinal cord, hence, *diaschisis cerebrospinalis* or *diaschisis corticospinalis*. This is the type of diaschisis that can affect the spinal cord via the long pyramidal tracts, and it is most likely to be observed in highly evolved organisms whose cerebral hemispheres dominate over lower parts of the brain. Diaschisis could, however, also go in the opposite direction, hence *diaschisis spino-cerebralis, diaschisis bulbo-cerebralis,* and the like.

*Diaschisis corticommisuralis*, another variety, involved the corpus callosum. It can account for how an injury to one hemisphere can alter the functional activity of the other hemisphere, a phenomenon observed with some frequency in clinical populations. Ipsilateral hemiplegia, for example, could be explained with this type of diaschisis.

*Diaschisis associativa*, yet another type, pertained to cortical suppression of other cortical areas via association fibers. It may be involved in forgetting highly specific information, such as a person's address or perhaps the name of a friend's wife. It may also explain why polyglots who become aphasic typically regain the use of their long-used native tongues well before they recover newly learned foreign languages.

Monakow contended that different types of diaschisis could occur simultaneously. But because the varieties may vary in degree, one type could dominate over the others at any given moment in time.[25(pp31-34)]

## SUSCEPTIBILITY TO DIASCHISIS

Because every structure in the brain is directly or indirectly connected to all other structures, all nervous structures can be affected by diaschisis and its effects can be distant and widespread. Nevertheless, based on what he was observing in the clinic, Monakow postulated that different parts of the nervous system might be more or less susceptible to diaschisis. Some brain areas, in fact, seemed much more sensitive than others. For instance, the motor cortex seemed more sensitive than the sensory cortex. And even within the motor strip, the parts responsible for the fingers and toes were more likely to be affected by diaschisis than those controlling the shoulders or hips.[5] Moreover, and with regard to higher cognitive functions, older, more used circuits appeared more resistant to diaschisis than newer and less used ones.[25(p40),33(p29)]

In addition, although Monakow hypothesized that the wearing off of diaschisis is always a passive process, there may be considerable variability in the speed with which it dissipates. In *Die Lokalisation im Grosshirn und der*

*Abbau der Funktion durch kortikale Herde*, it is maintained that diaschisis is likely to be most protracted in older people and of shortest duration in children. Monakow attributed such differences largely to the nature of the connections between the neural units, but he noted that other factors affecting the condition of the brain could also affect the time needed for recovery:

> The different rate in the regression of diaschisis is due to variations in the way individual neuron groups are linked, and in the type of excitability in different tectonic groups (everything else being equal, the groups that are more frequently used as a unit and are fortified by training will recover earlier than others), as well as to variations in the associated disorders of circulation, etc.[29(pp31,32)]

## COMPLICATING THE THEORY

Hans-Lukas Teuber, MD,[34,35] and others have argued that Monakow might have taken away from the elegance of his theory when he went on to postulate that the natural wearing off of diaschisis, which typically subsides in a matter of hours or days, can sometimes be delayed indefinitely. Monakow employed the term *diaschisis protractiva* for remote diaschisis effects that do not undergo diminution, possibly because of interactive factors (eg, vascular problems) that can affect the status of the distant brain area.

Another postulate that bothered some of his followers was that diaschisis can sometimes follow lesions of slow onset. As noted, strokes, injuries, and war wounds provided the bulk of the material he needed for the formulation of his theory, because these acute-onset cases tend to show the most dramatic recovery soon after injury. Nevertheless, Monakow argued that acute onset of damage is not a prerequisite for the theory and that a "slowly creeping in diaschisis" could be due to slowly breaking neuronal connections.[25(p35)]

## RECEPTION OF THE THEORY

The concept of diaschisis proved attractive for several reasons. First, it made intuitive sense. Second, it could account for some troublesome phenomena in a seemingly rational way. Third, it was both economical and parsimonious. Fourth, it did not demand the formation of new centers in the brain to account for recovery of function. And fifth, it did not call for neuronal rewiring or reorganizing, for which the microscopic evidence at the time was virtually nonexistent. Simply put, the beauty of the Monakow concept of diaschisis was that it fit well with both the concept of a static, hard-wired nervous system and with that of a dynamic organ, the interconnected parts of which share responsibilities and cooperate to execute functions.

Nevertheless, the Monakow diaschisis theory was not quickly embraced by the scientific community. His successor Minkowski cited several factors that did not help, including the publication of *Die Lokalisation im Grosshirn und der Abbau der Funktion durch kortikale Herde* on the eve of World War I; the difficulty of the subject matter; the multitude of associations, clauses, insertions, brackets, and footnotes used by Monakow; and his inability to communicate in an easily understandable way. Following a presentation by Monakow in Amsterdam, the Netherlands, in 1907, for example, his Dutch colleague Cornelis Winkler, MD, wrote a letter to him in which he explained that the majority of the attendees did "not quite understand, and neither did I. I was in the more favorable position as I had spoken with you and Valkenburg several times."[36(p2575)]

The ideas of Monakow seemed to have had their best reception in his adopted Switzerland, in part because of its isolation during World War I and the so-called *Monakowsches Kränzchen* (Monakow Circle), which evolved into the Zurich Society of Neurology and Psychiatry in 1904. But it was also picked up elsewhere. In England, Charles Sherrington, MD,[37(p245)] wrote that diaschisis fit well with his own conception of spinal shock, and Henry Head, MD,[38(p93)] used the concept when discussing the dynamics of aphasia and apraxia. Another prominent writer who was drawn to the concept was Kurt Goldstein, MD,[39] the German-born gestalt neuropsychiatrist who emigrated to America in the 1930s.

The most ardent supporter of the ideas of Monakow was, however, another German, Walther Riese, MD,[18] who practiced neurology in several German cities and in Paris before emigrating to Virginia. More than anyone else in the mid-20th century, Riese, who had been a close personal friend of Monakow, made the concept of diaschisis a focal point in his monographs, case studies, and theoretical reviews.[5,40-43] He repeatedly used it to explain transient aphasia after right-hemispheric lesions, functional differences following lesions of rapid and slow onset, and a host of other neurological phenomena. Riese was particularly instrumental in disseminating the Monakow theory in both German and English in the mid-20th century.

## CURRENT STATUS

What none of the early supporters of Monakow was able to do was to combine bedside observations with more direct measures of brain activity. It was not until the second half of the 20th century that investigators dealing with clinical patients began to look at contralateral cerebral blood flow (metabolism) after strokes that left one hemisphere undamaged. One early study was conducted by Høedt-Rasmussen and Skinhøj[44] in Denmark. These researchers noted that damage to one hemisphere can temporarily depress the metabolism of the other, a finding consistent with the Monakow *diaschisis corticommisuralis,* which has been verified many times.[32]

In addition, researchers conducting laboratory animal studies with even more informative and creative methods have generated increased interest in diaschisis as a viable concept. The early cortical electrophysiological studies on cats by Warren Kempinsky[45,46] stand out in this regard.

In the last few decades, a wealth of new studies involving neurotransmitter assays, metabolic measures, and pharmacological agents have been designed to test the theory of diaschisis.[32,47-54] The results of these studies have been mixed, causing debates in the literature.[55-58]

Today, no one who works with clinical patients or brain-damaged laboratory animals doubts that acute brain injuries can have secondary effects. But whether transient, localizable deficits are caused specifically by the disruption of neural connections and can only passively undergo remission, as specified by Monakow, is another matter. At the heart of the problem is the fact that many brain changes

can overlap in the first few days after an injury. They include edema, vascular changes, intracranial pressure, cell death, dispersions of toxic waste products, neuroglial proliferation, changes in transmitter levels, and the like. This complex state of affairs makes it exceedingly difficult, if not impossible, to isolate one event and single it out as the causal factor underlying a transient neurological effect.

From this perspective, the lasting neurological contribution of Monakow may not be in the details of his diaschisis theory. Instead, it may be in drawing much needed attention to transient neurological phenomena and in promoting the more basic idea of remote depressed levels of functioning. Although only a small percentage of publications still use his term *diaschisis* in the title or in its originally specified way in the text,[59-61] it is impossible to ignore the profound influence that Monakow has had on how we think about the dynamics of the nervous system, the limitations of the lesion method, consequences of brain injuries, and recovery of function.

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