EMIL DU BOIS-REYMOND

References


ÉDOUARD BROWN-SEQUARD (1817–1894)

Born as he was on the British island of Mauritius of a French mother (Charlotte Séquard, a vivacious young lady) and an Irish-American father (a Philadelphia captain who was lost with his ship [piracy?] soon after the marriage), Brown-Séquard was a British subject who spent much of his life traveling back and forth between Mauritius, France, England and the United States. In Paris they called him Brown. This was natural, as he was born Charles Édouard Brown and did not take his mother's name until 1846, and not until 1858 did he legalize it. Like Claude Bernard, he went to Paris with the intention of becoming a dramatic author. But he, too, soon destroyed his plays and enrolled as a medical student. By 1842 he was working under Trousseau and Rayer, the ablest clinicians of the time; again like Claude Bernard he preferred not to settle down to practice but to continue his physiological investigations. When in 1843 his mother,
He was interested in natural phenomena and turned to the study of the diencephalon, regarding the function of the diencephalon as fundamental to those of Sir Charles Bell and of later French scientists. In his studies of the nervous system of mammals, he found some of the cord to the brain regions in which cord was succeeded by white and gray matter.

His second interest was Claude Bernard's idea that the sympathetic nervous system was the key to the coordination of the body's functions. All this time, the conditions of the animals were being closely observed and recorded. The experiments involved in these studies were conducted in the United States, under the guidance of Dr. T. H. Chamberlain.

He boarded ship from New York and arrived in the United States in 1853. His first task was to establish a laboratory and a research center. He spent the next few years studying the nervous system of the horse, and his findings were published in a series of papers. These papers were later collected and published as a book titled "The Nervous System of the Horse." The book was well received and earned him international recognition.

In Philadelphia, delivering babies at the hospital, he gave birth to a child in July, 1853, and immediately named it "Barnes." The child grew up to become a prominent physician and surgeon in his own right.

Portrait, courtesy of the National Library of Medicine, Bethesda, Maryland.
He was interested in digestion, and would swallow sponges, pull them up, saturated, by a string, and analyze the gastric juice. Soon he turned to the nervous system. His doctoral thesis (1846) foreshadowed the discovery always associated with his name: the syndrome following hemisection of the cord. The ideas in vogue regarding the functions of the different parts of the spinal cord were those of Sir Charles Bell in England, extended by F. A. Longet in France, who maintained that all sensation was carried in the dorsal columns. In his thesis, Brown-Séquard stated that after sectioning the dorsal columns of the cords of cold-blooded vertebrates, birds and mammals, sensation in every case persisted in the parts situated below the section. He also commented on the ease with which he had found sensory impressions to be transmitted from one side of the cord to the other. The thesis was shortly followed by a series of papers in which he clearly established that hemisection of the cord was succeeded by sensory loss on the opposite side of the body and retention and even increase of sensation on the same side.

His second important neurological observation completed Claude Bernard's discovery of vasomotor nerves, for Brown-Séquard was the first to show, in 1852, that stimulation of the cervical sympathetic nerve in the rabbit causes blanching of the ear.

All this time (from 1843 to 1852) he was living in desperate straits. His experiments were carried on in his apartment, and his animals were housed there. To reduce the need for much food he drank coffee incessantly; some eighteen hours of his day were spent writing, reading, experimenting; he became seriously ill from an infection following a wound in the dissecting room. Realizing the poor state of his health and position, and having become involved in revolutionary activities, he decided that he must go to America. He knew hardly a word of English.

He boarded ship armed with a letter from his young friend and partisan, Broca, addressed to the University of Pennsylvania: "... Brown-Séquard ... has imposed upon himself incredible sacrifices ... and today has nothing left save an honorable character, profound erudition, and scientific articles which everyone can appreciate." In Philadelphia he eked out his earnings giving lectures, delivering babies at cut-rate prices, teaching French. The year not having brought him an appointment, he was again on the high seas in July, 1853, accompanied by his new American wife. Again
Paris was unheeding, and he and his wife continued on to Mauritius, there to find, in May, 1854, an epidemic of cholera which was to take the lives of 8000 people. Immediately he helped organize a hospital. He ingested material vomited by victims to test the efficacy of opium as a cure. Imagining that himself had the symptoms—as the story goes—he took so large a dose of laudanum that he almost died.

Fortune for once favoring him, he received an offer of a professorship, again on Broca's recommendation, from the Medical College of Virginia, which he accepted. But he was to stay only about four months. To the faculty he had a "surplus of honesty" (he disapproved of slavery), with a lack of energy; his lectures were "not very unlike an attack of spasmodic asthma"; the agony of trying to make himself understood was, if anything, topped by the agony of his listeners, trying to comprehend. His demonstrations, by contrast, were "like wonders wrought by a stage magician." Something was wrong, for in Paris this short wiry person had always been in constant motion and he had had a great gift of elocution.

Back again in Paris, in 1855, his practice as a neurologist began in earnest with the loan by Rayer of an electrical stimulator which he proceeded to apply with great skill to human patients. But observing convulsions in the guinea pigs upon the spinal cords of which he had performed various operations, he spent much of his subsequent life in the attempt to discover the causes and treatment of epilepsy. Later on, he was instrumental in introducing bromide for epilepsy, as suggested by Locock in 1857. His inquiring mind early led him into another field, endocrinology. Addison had published in 1855 his observations on the clinical effect of disease of the suprarenal capsules, and a year later Brown-Séquard showed how fatal adrenalectomy is. Toward the end of his life Brown-Séquard became uncritically enthusiastic over organotherapy—he had repeatedly injected into himself crude extracts of animal tissues—so that his career ended on a note of extravagant claims: attempts to isolate an effective testicular hormone to counteract senescence made him a laughing stock in some quarters. But he had greater vision than they knew.

In 1858 he undertook a course of lectures in several University centers in Great Britain, and in 1860 was appointed physician to the newly founded National Hospital, Queen Square—the second appointment on the first). Here Hughlings Jackson's practice came. But £200 he insisted £10,000 to see a right person to advise.

Restless, his wit in 1864. Now a chain delivered the address: "I would urge upon creatures, endowed with whose sensibility is ing a carrot (even you are cutting the animals for experiments selfish enough). In 1868 he was a twelve-year-old son's rank in Paris—his libertarian lean.

The next eight years in Europe, were his of a daughter. He was.

Another sailing ship really cared for, naturalization papers Bénard's successor at filled until his death.

During his Paris physiology and pуб. 1876, dealt with his certain regions was previously made (in ulceration due to the corpus striatum or theing, contended that fault. He dwelled o
appointment on its professional staff (Jabez S. Rainskill received the first). Here he remained for about 4½ years. The young
Hughlings Jackson fell under his spell. Honors and a lucrative
practice came. But asked to see a patient in Liverpool for a fee of
£200 he insisted that his ordinary fee would do; offered a fee of
£10,000 to see a boy in Italy, he declined, saying he was not the
right person to advise on the case.

Restless, his wife having died, he again set out for America, in
1864. Now a chair at Harvard was waiting for him. In 1866 he de-
livered the address opening the Medical Lectures at that school.³

"I would urge upon you," he said, "to make good use of those low
creatures, endowed with so little sensibility,—the frogs, the fishes,
and the turtles; to which list I might add the rabbits, animals
whose sensibility is indeed so dull, that they will hardly stop
eating a carrot (even when not particularly in need of food) while
you are cutting their flesh . . . " Arguing that the use of such ani-
mal for experimenting was for the good of mankind he said, "I
am selfish enough to prefer mankind to frogkind, rabbitkind, etc."

In 1868 he was back in France, probably for the sake of his
twelve-year-old son. But since he could not be accorded profes-
sorial rank in Paris—he was still not a French citizen, not to speak
of his libertarian leanings—he returned to America again, in 1870.
The next eight years, in New York, though interrupted by trips to
Europe, were his unhappiest: his second wife died after the birth
of a daughter. He acquired a third, the widow of a painter.

Another sailing ship returned him to Paris in 1878, the only city
he really cared for. Claude Bernard had just died. Taking out nat-
uralization papers he became, finally, a real Frenchman, also Ber-
nard's successor at the Collège de France, an office he happily ful-
filled until his death from apoplexy fifteen years later.

During his Paris years he founded three journals devoted to
physiology and published hundreds of articles. One of them, in
1876, dealt with his observation that cerebral-cortical ablation in
certain regions was succeeded by gastric ulceration, an observation
previously made (in 1844) by Moritz Schiff, who considered the
ulceration due to local vasomotor paralysis (from lesions of the
corpus striatum or cerebral peduncle); Brown-Séquard, disagree-
ing, contended that contraction of gastric arteries and veins was at
fault.⁶ He dwelled on his view that neural activity at one level is
always colored and conditioned by what is happening at another, remote level, a notion independently elaborated by Sherrington later on. His public discourses became famous: at the International Congress of Medicine in Paris in 1867—the first of its kind—he was the most eagerly awaited speaker; however, owing to the sudden illness and death of a fellow Mauritian, he did not appear.

Brown-Séquard left an enthusiastic group of young workers, the best known of whom were d’Arsonval and François-Franc; upon his passing, the great French school of experimental physiology—belonging to Magendie, Flourens, Claude Bernard and himself—was never the same again.

J. M. D. OLMSTED

References


JOANNES GREGORIUS DUSSEUR DE BARENNE (1885-1940)

The sudden death of Professor Dusser de Barenne on June 9, 1940, occurred at a time when international communications were seriously disrupted, and many of his colleagues in Europe were therefore long unaware that his brilliant career had been brought to a close. Death occurred in Dusseldorfer, a small town in the Netherlands, occupied by German forces in 1940. Dusser de Barenne was a graduate of the Catholic University of Louvain, Belgium. After studying at the University of Paris and the École Normale Supérieure, he received a doctorate in medicine from the University of Paris in 1908. He became a professor of neurology at the University of Paris in 1913 and served as the director of the Neurological Institute of the University of Paris from 1917 until his death. He was a member of the French Academy of Sciences and the Royal Netherlands Academy of Sciences. Dusser de Barenne is known for his contributions to the field of neurology, particularly in the areas of neuroanatomy and neurophysiology. He is also remembered for his work on the Pathological Physiology of the Nervous System, which remains an important reference work in neurology. He made significant contributions to the understanding of the role of the nervous system in the regulation of body functions and the mechanisms by which diseases affect the nervous system. His research and teaching were widely recognized, and he was a respected figure in the field of neurology. Dusser de Barenne's contributions to the field of neurology and his dedication to the advancement of medical knowledge have left a lasting legacy. His death was a significant loss to the field of neurology, and his contributions continue to be celebrated and studied today.
fourth ventricle. In the third ventricle they were not so pronounced as in the previous case; how-
erver, they extended somewhat further downward into the upper region of the calamus scriptorius.

We are dealing with an independent, inflammatory, acute nuclear disease in the region of the
cerebral nerves, which leads to death within 10 to 14 days. The focal symptoms consist of the corre-
sponding ocular muscle palsy, which develop rapidly, progress, and finally lead to almost total
paralysis of the ocular muscles. But even then certain muscles are spared, such as the sphincter
iris and the levator palpebrae. The patient’s gait
becomes staggering and shows a combination of
stiffness and ataxia, reminiscent of the ataxia of
the alcoholic. The general appearance is very
striking. It consists of impairment of conscious-
ness, which is either somnolence from the onset
or a terminal state of somnolence preceded by
a longer period of agitation. In addition, involve-
ment of the optic nerves by inflammatory changes
of the discs were characteristic of all three cases.
There was always a severe injury before the onset
of the disease. One patient had sulphuric acid poi-
soning followed by pyloric stenosis. The other
two patients misused alcohol to an unusually high
crage. The question of whether the appearance of
delirium tremens in these last cases is to be re-
garded as a complication or as another one of the
general symptoms of this disease can be asked but
cannot be answered. In any case, there was not a
simple delirium tremens, but one complicated by
the symptoms of poliomenaephalitis. It might be
pointed out that the characteristic disorientation
could also be demonstrated in the first patient,
whose appearance was very far from that of de-
lirium. After the first case, which could not be
understood at all during her lifetime, I was able
in the second and third cases to make the diag-
nosis in spite of the variation in the general
symptoms. This probably gives justification for
presenting these cases as a specific disease picture.

X

Brown-Séquard Syndrome

THE BROWN-SEQUARD syndrome represents a turning point in the history of
neurophysiology. Before Charles Edouard Brown-Séquard performed his classical ex-
periments in the mid-19th century, the doc-
trines of Sir Charles Bell were dominant
and all sensory impulses were believed to
ascend the spinal cord ipsilaterally in the
posterior column. Crossing to the opposite side
was believed to occur only when the sensory
impulses reached the brain.

Brown-Séquard, the son of a French-
woman and an American sea captain, pursued
his career as a physician and investigator
mainly in Paris but also in the United
States. By demonstrating that hemisection
of the cord in animals produces sensory loss
on the opposite side of the body with reten-
tion of sensation on the ipsilateral side, he
challenged the idea that the posterior col-
umn was the sole sensory pathway and provid-
ed concrete evidence of sensory demascula-
tion in the spinal cord.

References

1. Olmsted, J.M.D.: “Charles Edouard Brown-
Séquard (1817-1894).” in Haymaker, W. (ed.):
Founders of Neurology, Springfield, Ill: Charles C.
Thomas, Publisher, 1953, pp 269-269.
2. Olmsted, J.M.D.: Charles Edouard Brown-
Séquard, A Nineteenth Century Neurologist and
Endocrinologist, Baltimore: Johns Hopkins Press.
1946.
3. Schiller, J.: Claude Bernard and Brown-
Séquard: The Chair of General Physiology and
1956.

ON THE TRANSMISSION OF SENSORY
IMPRESSIONS BY THE SPINAL CORD*

By M. Brown-Séquard

Four years ago I announced in my inaugu-
ral thesis (Research and experimentation on the physi-
ology of the spinal cord, pp. 22 and 25.—Paris,
January 3, 1846) that section of a lateral half of

*Translation of De la transmission des impressions
sensorielles par la moelle épinière, Compt Rend Soc Biol
11:192-194, 1849.

the spinal cord does not destroy sensation in the
parts of the body which receive their nerve supply
from the portion of the cord separated from the
brain. This finding was similar to those of
Schoeps, Van Deen and Stilling and contrary to
the statements of Kürschner, M. Longe and other
physiologists. Since that time I have had occasion
to do this experiment more than sixty times either in my courses, or particularly in the process of studying all the circumstances of the phenomenon, or finally to satisfy many people's curiosity. This is what I have seen:

1. Immediately after cutting a lateral half of the cord in the dorsal region of a mammal, sensation appears very diminished in the hindlimb on the side of the section. Feeling is completely lacking in the other hindlimb. Sometimes I have found sensation intact or nearly so in the lower limb corresponding to the side of the section, while the opposite hindlimb was either insensitive or very slightly sensitive.

2. After five to ten minutes of rest following the operation, one always finds the hindlimb corresponding to the side of the section very sensitive. In many cases, or even in most cases, this limb appears noticeably more sensitive than normal. This fact is certainly very curious; but there is another fact, even more unexpected: the hindlimb of the side opposite the section is insensitive or very slightly sensitive. It follows from these facts that cutting a lateral half of the spinal cord, far from causing loss of sensation in the parts caudal to the section on the same side, renders them hyperesthetic. At the same time, if there is loss of complete sensibility in the other half, some sensibility is produced on the opposite side of the body, caudal to the section.

Eighteen months ago, we showed to the Biological Society of London a guinea pig in which we had cut a lateral half of the cord at the level of the eleventh dorsal vertebra. Everyone present could verify that the sensitivity of the hindlimb on the side of the section was very great. The animal was sent to M. Rayer, who had an autopsy performed by our late colleague, M. Désir. At the next meeting, M. Désir showed the Society the portion of the cord where the cut had been made. One could recognize that it was at the indicated place and really involved the designated half of the cord.

In the meeting of December 1, 1849, we showed a guinea pig in which the right lateral half of the cord had been cut before the eyes of several members of the Society. The section was at the tenth dorsal vertebra; the animal had lost much blood. The operation, carried out in half darkness, had been long and very painful. In such circumstances, one ordinarily finds the two hindlimbs paralyzed for voluntary movement and sensation for some time after the operation. This is what occurred in this case. But at the end of five or six minutes, voluntary movement returned in the left hindlimb and sensation in the right hindlimb. About twelve minutes after the operation, sensation was already in the right hindlimb and all in the left. An autopsy was then done by M. Claude Bernard, with the meeting still in progress. The Society verified that the right lateral half of the cord was cut transversely at the indicated level.

Schoors Van Den and Stilling have observed perfectly that the hindlimb on the same side as the section of a lateral half of the cord does not lose sensation. To this fact we add the following: 1. Generally this section leads to a momentary decrease in the sensitivity of the corresponding hindlimb. 2. At the end of a certain time (from three to fifteen minutes) after the section, the sensitivity of the corresponding hindlimb appears markedly increased.

3. The hindlimb of the side opposite to that where the section is made loses its sensitivity completely in large part. The spinal cord thus appears to have, at least in part, a crossed action with respect to the transmission of sensory impressions. This is so true that, if, after having cut one lateral half of the cord in a mammal, one then cuts the other half some centimeters away from the first section, one finds the two hindlimbs insensitive or very slightly sensitive. We cannot examine here the questions that these experiments raise; we will make them the subject of an extensive report. Nevertheless, we believe it is necessary to say that if the transmission of sensory impressions occurs partly in the posterior columns of the cord, it occurs mainly in other areas of this nerve center. In fact, not only is sensation not lost after section of the posterior columns, but it is even markedly increased in the parts of the body which should be insensitive according to the erroneous theory that the systematic physiologists persist in supporting despite the contrary evidence and despite the rejection of Charles Bell.

Some clinical phenomena may be viewed as experiments of nature and serve to stimulate investigation into the mechanisms of normal function. An example is Argyll Robertson's pupillary syndrome.

Argyll Robertson was a pioneering ophthalmic surgeon who made several important contributions to our basic understanding of ocular mechanisms. In 1869, the same year that Johann Friedrich Förster described his well-known form of miosis, Robertson reported miosis of another type, characterized by absence of the pupillary light reflex with retention of pupillary constriction to near vision. Robertson noted the association between the pupillary syndrome and disease of the spinal cord. Later, the Argyll Robertson pupil was found usually to be the result of neurosyphilis, and it became a cardinal sign of the disease.

Robertson reasoned that a lesion in the oculospinal (sympathetic) nerves was responsible for the syndrome. The matter is still unsettled despite much disputation. Experimental production of the Argyll Robertson pupil has not been achieved, and current theories attribute its etiology to lesions in the hypothalamic regions or in the midbrain tegmentum rostral to the third-nerve nuclei. With decline of neurosyphilis, the Argyll Robertson pupil has become an uncommon finding, but the research stimulated by Douglas Argyll Robertson's description has contributed greatly to our present knowledge of the optic reflexes.

References