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OF

STEPHEN WALTER RANSON

1880–1942

BY

FLORENCE R. SABIN

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Stephen Walter Ranson, Professor of Neurology and Director of the Institute of Neurology at Northwestern University, died of coronary thrombosis on August 30, 1942. He was born August 28, 1880, at Dodge Center, Minnesota. He was the son of Stephen William and Mary Elizabeth (Foster) Ranson who were of English and Welsh descent. It is clear that he came of a medical family, for his father was a physician and of the six children, three became physicians and one received a Doctorate of Philosophy in Psychology.

Ranson was graduated from the University of Minnesota in 1902. He then went to Chicago University, where he took the Master's degree in 1903 and the Ph.D. under H. H. Donaldson in 1905. He was a Fellow in Neurology at Chicago from 1904 to 1906 and received his medical degree at the Rush Medical College in 1907. After a year of interneship at the Cook County Hospital, he became Associate in Anatomy at the Northwestern University Medical School through the influence of Prof. Arthur W. Meyer. From then on there was no break in his successful academic career. In 1910-11 he studied under Wiedersheim in Freiburg; in 1912 he became Professor of Anatomy at Northwestern University. In 1924 he went to Washington University School of Medicine in St. Louis as Professor of Neuroanatomy and Head of the Department of Neuroanatomy and Histology. In 1926 he spent the summer at Queen's Square Hospital, working with Gordon Holmes and Kinnier Wilson in the clinics. He remained at Washington University only four years and in 1928 was induced to return to Northwestern University Medical School as Professor of Neurology and, more important still, as Director of a new Neurological Institute to be organized in recognition of the value of his research.

Ranson was the combination of teacher-investigator. He trained many students in research as is evident from his bibliog-

raphy, and an exceptional number of his students are now heads of departments in our medical schools. Through his textbook on the Anatomy of the Nervous System he has influenced medical students in practically all of our schools. It is, however, interesting to note that in spite of the marked swing toward physiological interests in his research, this interest was reflected best in the last or seventh edition of his book.

Ranson received many honors. He delivered the Weir Mitchell Oration in 1934, a Harvey Lecture in 1936, the Dunham Lectures in 1940, and the Hughlings Jackson Lecture in 1941. He was a Fellow of the American Association for the Advancement of Science, a Member of the National Academy of Sciences, the American Neurological Association, the American Physiological Society, and the American Association of Anatomists, of which he was president from 1938 to 1940.

In 1909 Ranson married Miss Tessie Grier Rowland of Oak Park, Illinois, who made their home a center of hospitality. There were three children—one son and two daughters. His son, now Captain Stephen Ranson, became a physician, and it must have been a great gratification to him that in 1941 both his son and one daughter, Mary Ranson, collaborated with him in research.

Medical research in this century is characterized by a breaking down of the barriers between different disciplines, barriers which grew up in the last century because the problems then attacked needed the development of highly specialized techniques. In the present phase new types of problems have come to the fore, which need not one but a wide range of these techniques. Nowhere is this new type of research more happily illustrated than in neurology when, at the turn of the century, Sherrington (summarized in 1906 by the publication of his book, "The Integrative Action of the Nervous System") unraveled the mechanism of "how the animal stands," and thus wiped out all artificial barriers between anatomy and physiology.

Ranson began his work at the start of this new era and developed with it. His training had been strictly anatomical. He first discovered that there are more unmyelinated than myeli-

nated fibres in the dorsal roots, and then proved that these unmyelinated fibres were afferent in function and followed their central connections. He studied their relation to the sympathetic system and finally became the acknowledged leader in the field of the physiology of the hypothalamus, the center of control for the sympathetic system and for water balance.

Ranson's first work concerned the question of whether there is retrograde degeneration, as well as direct, and he proved it in the affirmative by the use of the double pathway of the corpus callosum (1, 2).* His next study, published in 1906 and constituting the dissertation for his Ph.D. degree (4), was entitled "Retrograde degeneraton in the spinal nerves," but the subject matter was more significant than the title. The procedure suggested to him by Donaldson was to cut a spinal nerve, allow degeneration and then count both the myelinated axons in the corresponding roots and the cells in the spinal ganglion. Since 1896 (Gaule and Lewin) it had been known that there are more cells in a spinal ganglion than there are myelinated fibres in its root. The studies of Gaule and Lewin, with those of Hatai [1902] and of Ranson, showed from three to six cells per fibre, varying both from nerve to nerve in the same animal and from animal to animal (138). Ranson went on to find the meaning of this fact, namely, that 70 per cent of the cells, known to be smaller than the rest, give rise to unmyelinated fibres. The existence of unmyelinated fibres in spinal ganglia was just becoming known, for example, to Cajal [1906] and to Dogiel [1908], but it was Ranson's contribution to demonstrate how large their number, even more than myelinated fibres, and to work out their peripheral distribution and their central connections. Thus he extended our knowledge, of this afferent system (5-7, 8-13) and, indeed, it was these studies, carried on over a long term of years and showing remarkably sustained interest, that laid the foundation of Ranson's career in neurology.

The conventional way of staining nerves with osmic acid had stressed only the myelin sheaths; but, with the introduction of

* Figures in parentheses refer to the numbers of titles in the accompanying bibliography.

silver methods, axons were brought out. Ranson modified one of Cajal's silver methods, making the so-called pyridine silver technique (7, 10) which permitted the discrimination of axons from connective tissue fibres and from neuroglia. The axons of myelinated fibres stained yellow in the center of clear, unstained rings of myelin, while the axons of the unmyelinated fibres were brown or almost black.

In the study of spinal ganglia (10,13) with the pyridine silver method, Ranson found, as had Dogiel, more variations in the type of origin of the single axon, single, branched, or plexiform, from both large and small cells, than can be related to functional differences, but the common and essential characteristic of all of them was the bipolar division into two branches. Of the unmyelinated fibres the branch which entered the cord was smaller than the one which ran into the peripheral nerves. He found that the spinal nerves carried more unmyelinated fibres than myelinated ones, and far more fibres than could be accounted for by the postganglionic, motor, sympathetic fibres. In the peripheral nerves most of the afferent, unmyelinated fibres were distributed to the cutaneous nerves and only a few to the muscular branches (104,124).

Ranson surveyed these unmyelinated fibres from the ganglia of the trigeminus and the vagus complex (19, 23, 25). In the sensory ganglia of the vagus he found the same predominance of small, unipolar cells giving rise to afferent, unmyelinated fibres. He then was able to complete the study of the two kinds of roots of the vagus begun by the Belgian anatomists, Van Gehuchten and Molhant, first, the efferent roots containing two sizes of myelinated fibres, many small and a few large ones, and second, the more varied afferent roots, containing many more unmyelinated than myelinated fibres, the latter being of all sizes —large, medium, and small (12, 13, 19).

The number and distribution of this extensive, afferent, unmyelinated system could be established only in animals in which the sympathetic chain had been removed (102). This he did both opposite the lumbar plexus and in other animals opposite the brachial plexus. After allowing time for complete degenera-

tion of postganglionic fibres, he made comparative studies of a nerve to the skin and one to muscle. In a cutaneous nerve he found 3.5 residual unmyelinated fibres, hence afferent in type, for each myelinated one. For the vagus complex (125), he found the persistence of the afferent, unmyelinated fibres after elimination of the sympathetic fibres induced by removing the superior cervical ganglion and after cutting the vagus roots as well.

This concept that there are unmyelinated fibres which are sensory in type made necessary a restudy of the sympathetic system (34, 36, 39, 43, 101, 102, 124). Ranson found that all the sensory cells for the viscera were in spinal ganglia or their cranial counterparts. Their fibres were both unmyelinated and myelinated. As Langley had found, Ranson confirmed that when a spinal nerve was cut distal to a spinal ganglion, nearly all the fibres of the corresponding white ramus degenerated, which would not have happened if afferent fibres were running from the sympathetic chain to spinal ganglia (43). When the efferent sympathetic fibres were removed from a white ramus by cutting the nerve roots proximal to the spinal ganglion (39), the visceral afferent fibres remained in the white rami; they are myelinated fibres of all sizes, as well as unmyelinated, and they run not diffusely but in compact bundles.

Structurally, the finding that all afferent cells are in spinal and the corresponding cranial ganglia means that the sympathetic ganglia are entirely efferent in type. Ranson therefore restudied these ganglia. As was well known, the cells are multipolar with exceedingly complex dendrites and with an axon that becomes a postganglionic, efferent fibre, for the most part unmyelinated.

The axons of the preganglionic fibres, on entering a sympathetic ganglion, such as the superior cervical ganglion, form an extensive plexus of branching axons in the intercapsular spaces of the ganglion. They come into synaptic relations with the complex, branching dendrites of the multipolar ganglion cells. In the human being, besides the extracapsular dendrites of the sympathetic ganglia, there are also complex intracapsular dendrites, making large glomeruli of processes often from several

cells. Ranson found that when all the preganglionic fibres entering the superior cervical ganglion had been cut, with resulting degeneration of their axons, there was no evidence for association neurons either within one sympathetic ganglion or between two or more of them (34, 36, 39, 40, 41, 42, 43). It is probable that each preganglionic fibre ends on several sympathetic cells. Huber has pictured one entering axon in relation to seven cells, and Ranson found thirty-two sympathetic ganglion cells to each entering axon (42).

Ranson thus came to the generalization that all the cells of the spinal ganglia are unipolar, with T or Y shaped processes, that is to say, they are afferent in type. This conclusion was reached only after ruling out two puzzling structures, first, the so-called pericellular baskets described by Dogiel in 1908, which might be synapses, motor in type, in spinal ganglia, and second, possibly multipolar cells in these ganglia described by Kiss. Ranson (127) showed that the Kiss cells were artefacts due to shrinkage. For a long time Ranson (58, 59, 60, 63) believed that Dogiel's pericellular baskets might be synapses, motor in type, within spinal ganglia, but he finally saw that the strongest evidence for their existence, the apparent blocking of impulses by painting nicotine on spinal ganglia, was faulty (60). Moreover, it was finally shown that these pericellular networks are probably a reaction to injury (see: Barris, a pupil of Ranson, *J. Comp. Neurol.*, 1934, *59*, 325; also p. 53 in Ref. 213).

Thus it finally becomes clear that Ranson established the fact that a large proportion of afferent fibres are unmyelinated in type, that they arise from the small cells of spinal ganglia and the corresponding cranial ganglia and that these ganglia contain only afferent fibres.

Ranson then proposed to study the pathways of the unmyelinated system in the spinal cord. In this work he combined with a pharmacologist, von Hess (27), and a surgeon, Billingsley (29-33). On studying the entry zone of the dorsal roots, they found that all of the unmyelinated fibres were segregated into the lateral border of the roots and entered Lissauer's tract bordering the substantia gelatinosa of Rolando which made a nucleus

of reception for them (14-17, 20, 21, 26). Moreover, an important structural point became clear, namely, that all entering unmyelinated fibres are short, ending (29) almost completely in their segment of entry with perhaps slight overlapping into the next above. In this characteristic they agreed with the known paths of pain and temperature. As a matter of fact, the concept that the unmyelinated fibres might carry pain and temperature impulses was suggested to Ranson (20) by a parallelism between these two types of sensory fibres, and Head's concept of two types of functional sensory paths, protopathic or epicritic. At that time Head's work had not been refuted. The separation of unmyelinated fibres in the cord is not complete for a few fine, myelinated fibres also enter Lissauer's tract, but the vast majority of myelinated fibres, as had long been known, become the posterior ascending columns. Lissauer's tract as the zone of entry of the unmyelinated fibres proved characteristic of all the animals commonly used in experimental work (15, 20). Also, Lissauer's tract itself contains no long neurons (31), none extending more than two or three segments, and thus (29) represents intersegmental conduction paths.

It was found possible in the lower segments, where the dorsal root bundles are longer, to cut the lateral, unmyelinated bundle and the medial, myelinated one separately (27, 29-33, reviewed in 47). Also Lissauer's tract, and of course the posterior columns, could be eliminated separately. When the medial (myelinated) roots or the posterior columns only were cut, there was no loss in pain and no change in vasomotor reflexes (27, 98). On the other hand, stimulation of the lateral, unmyelinated root fibres, studied in the 7L and 1S segments, gave rise to struggling and to a reflex rise in blood pressure (38, 98). Hence Ranson concluded that the unmyelinated fibres carried pain impulses. These studies, Ranson (98) considered as his best evidence that unmyelinated fibres mediate pain. More conclusive evidence was finally provided by Gasser and Erlanger (*Amer. J. Physiol.*, 1929, *88*, 581) who showed, by means of the cathode ray oscillograph, that some of the fibres that carry pain may be the smallest in the nerve.

It had long been known that the stimulation of the central end of a nerve might give rise to either a fall or a rise in blood pressure. In 1895, Hunt had shown that depressor responses were elicited by weak stimuli, that is to say, had a low threshold, while pressor impulses had a high threshold. In following the pathways for pain and temperature in the cord, Ranson and Billingsley found that destroying Lissauer's tracts and the posterior horns of both sides abolished the pressor reflex but not the depressor. Under these conditions, continued stimuli merely increased the fall in pressure. The pressor pathway ran on both sides but predominantly homolaterally. The destruction of Lissauer's tract did not abolish consciousness of pain but only that part of the pain and temperature mechanism associated with the pressor reflex functionally, and structurally only that part which is intersegmental within the cord. The pressor reflex path for the vessels of the head was found to be in the tractus spinalis N. V., as was shown by cutting the tract, an experiment carried out by Miss M. Wilson, a pupil of Ranson, in 1921.

The pathway for the depressor reflex, demonstrated by stimulating the sciatics with a weak current, was abolished only by cutting both lateral columns, and proved to be predominantly crossed. This pathway has fewer and longer neurons (27, 32, 47) than the pressor path. When the depressor reflex has been eliminated by cutting both lateral columns, a moderate current excites a pressor effect (32), suggesting that, in the intact cord, there is an algebraic summation of pressor and depressor impulses. The structural differences between the pressor and depressor pathways, the former of many short neurons, Lissauer's tract, and the latter of a few long ones in the lateral columns, Ranson thought might account for their marked difference in threshold. But it is now known that differences in the rate of conduction of impulses, as shown by Dr. Gasser and his associates, also enter as a factor.

Ranson and Billingsley (33) were aware that these pathways were not simple, for they found that lesions of the posterior gray matter low in the cord cut off pressor effects induced by

strong stimuli of the sciatic nerves, but that if lesions were made higher up, there was less disturbance, suggesting alternate pathways probably in the gray matter of the cord. It was clear that the main arc for pressor impulses was not complete in the cord. They then exposed the floor of the fourth ventricle (30) and found a pressor point at the apex of the ala cinerea and a depressor point in the area postrema just lateral to the obex.

The peripheral mechanism for vasodilators proved complex and difficult to analyze, both from the obscurity in postulating the mechanism, that is, how a vessel can be made to dilate actively, and from the nature of the nerve impulses associated with the process. As early as 1876, Stricker had postulated dilator fibres, and in 1901 and 1908, Bayliss had proved their existence and shown that for the hindlimbs the cells of origin were in the lower lumbar and first sacral spinal ganglia. He postulated antidromic conduction along nerves afferent in type. Ranson [1922] now proposed to explore the relation of the unmyelinated fibres to this concept. After postulating (49) and finally discarding the idea that there are synapses in the spinal ganglia, Ranson and his associates (50, 51, 53) devised an experiment in which they could separate peripheral and central effects on vasodilators. They placed a dog's leg in a plethysmograph, cut and tied the opposite iliac artery, and pulled it out through an opening in the flank, so that thus they could inject through it directly past the bifurcation of the aorta into the vessels of the opposite leg. Then, in the completely denervated leg, they obtained vasodilation with nicotine. These experiments, they concluded, confirmed the work of Dale and Richards (*J. Physiol.*, 1918, *52*, 110) by which these investigators had shown that vasodilation is a function of the arteries and capillaries themselves, not initiated by nerve impulses but subject to regulation by them. Thus the mechanism for vasodilation proves to be different from that for constriction of the vessels, the latter being mediated directly through sympathetic ganglia, the former being primarily a peripheral mechanism.

These studies on vasomotor pathways made Ranson formulate the concept that the unmyelinated fibres which form the afferent

part of their arcs carry pain and temperature impulses. Since these fibres were the smallest in the nerve, he analyzed a given cutaneous nerve in terms of size of fibre, as well as in the proportion of myelinated and unmyelinated fibres, and compared the data with the known punctate sensibility of the area of skin supplied by this nerve. At first in a study of the median cutaneous nerve of the forearm (123), he found a remarkable statistical parallelism with the studies of von Frey; for example, he found 90 per cent small fibres to be compared with 87 per cent pain points; but with other nerves, such as those for the scalp (138), he found that the correlation broke down completely because there were many more fine fibres, both myelinated and unmyelinated, than there are pain spots and far too few large fibres for the touch spots. Thus, Ranson saw from his own work that size of fibre does not correlate with function, which had been more conclusively proved by direct rather than by indirect evidence by Dr. Gasser (*Research Publications,* Assoc. Nervous and Ment. Dis., 1935, *15,* 35) who showed that rate of conduction and diameter of fibre do correlate with each other, but neither corresponds to function.

It is thus clear that Ranson had established his discovery of an extensive system of unmyelinated afferent fibres, had worked out their peripheral distribution, had demonstrated that their entry zone into the cord is Lissauer's tract and its medullary extension, the tractus spinalis N. V, and had proved that this tract is a part of the mechanism for vasomotor pressor reflexes and that hence these unmyelinated fibres are a part of the mechanism for the conduction of pain and temperature impulses. But it is also clear that the fine, unmyelinated fibres, C types in physiological terms (Gasser, H. S., *J. Neuro-Physiol.,* 1939, *2,* 361), are not the exclusive pathways of pain, since Dr. Gasser (*Research Publications,* Assoc. Nervous and Ment. Dis., 1935, *15,* 35) showed that pain is carried by larger fibres of the B type which are myelinated, and Dr. Tower (Tower, S. S., *Proc. Soc. Exp. Biol. and Med.,* 1934-35, *32,* 590) demonstrated that sensory fibres from the cornea conveying pain are mainly

myelinated (*J. Neuro-Physiol.*, 1940, *3*, 486), the slowly reacting C fibres not being demonstrable.

For a period of years Ranson became interested in the subject of postural contraction or muscle tonus, in part through the stimulus of Sherrington's studies on decerebrate rigidity and in part through his own interest in making as complete a survey as possible of the functional rôle of the spinal ganglia. In the study of decerebrate rigidity, it appeared that there was a perfect example of tonus, involving a type of contraction with marked lack of fatigue and lack of heat production. Moreover, Sherrington had noted that the muscle had also a certain degree of plasticity. There developed then a concept of three different types of activity in muscle, the usual phasic contraction, contractile tonus, and plastic tonus.

Sherrington and Brown had shown that the dorsal roots are necessary for tone—in Sherrington's view through propioceptive impulses. Ranson, defining tonus (69) as "the steady, indefatigable contraction required for posture," felt that Sherrington's concept did not account adequately (64) for the lack of fatigue. He proposed to explore two mechanisms as possibly related to the phenomenon, (a), the question of sympathetic connections, and (b) the question of action through the spinal ganglia either by motor impulses or by antidromic conducton along afferent fibres. The subject proved baffling and Ranson's own studies did not unravel the nature of the mechanism nor why it is not subject to measurable fatigue. This phenomenon has since been explained by the nature and timing of certain impulses through motor nerves.

Hinsey and Ranson (66) found that after complete removal of the left lumbar sympathetic chain, followed after 50 to 75 days by decerebration, there was no difference in tonus on the two sides, as indicated by posture, by measuring resistance to flexion and by the effect of tetanus toxin. This ruling out of the sympathetic system from the mechanism of tonus confirmed the work of van Rijnberk [1917], and is in complete agreement with the studies of Cannon. As has been stated, Ranson became convinced that there are no motor synapses in the spinal

ganglia. Moreover, the absence of any endings of dorsal root fibres in striated muscle, as demonstrated by Hinsey (78), ruled out direct efferent impulses through the spinal ganglia and any mechanism for making antidromic impulses effective on muscle fibres (78), but did not analyze the rôle of afferent impulses in tonus. During these studies, Ranson (101) found that it was practically impossible to de-afferent the hind legs in cats without a certain amount of damage to the cord because the operation could not be done without opening the dura. In this case cutting of the afferent nerves was followed by an immediate loss of tone with subsequent extensor rigidity. For the forelimbs, on the other hand, it was not necessary to open the dura in order to cut the dorsal roots, in which case Ranson found that the immediate loss of tonus was not followed by an overaction of the extensors. This gave to Ranson (101) the evidence that afferent impulses do not play an exclusive rôle in maintaining tonus.

Concerning the central relations of the mechanism for tonus, Ranson and Hinsey (79, 80) made an important advance. Sherrington had shown that in decerebrate rigidity, when the afferent nerves were intact, the crossed extensor reflex was expressed as a slow contraction followed by a prolonged, slow decline, but that when the limb was de-afferented, both contraction and relaxation were rapid. Ranson and Hinsey (80, 81), using the so-called anemic method of decerebration of Pollock and Davis (tying both carotids and the basilar artery), got, on the other hand, a quick response and a slow relaxation. They therefore made transections at different levels of the brain-stem and found that maximum rigidity occurs when the mesencephalon is thrown out (80) and that a cut across the upper border of the mesencephalon, leaving most of the red nucleus intact, gave a quick contraction and quick relaxation; while a cut between midbrain and pons, eliminating the effect of the rostral midbrain, gave the slow reaction of the Sherrington type. They concluded that the rate of response is not due entirely to peripheral, afferent impulses but rather is under central control as well; that in the upper end of the midbrain is a center for regulating tone.

When the transection is low down, the inhibition of this center has been cut off, and the tonic response and relaxation are both slow. Thus they conclude that there is a center in the neighborhood of the red nucleus for tonus, inhibitory in action, that is, mediating cerebral impulses, but that this is not the only center for tonus, since tonus is still present when the hypothalamus is intact as well as the cerebellum. They consider that decerebrate rigidity with intact red nuclei is due in part to the removal of inhibitory influences from the cerebral cortex.

Ranson now began the most important work of his career, the study of the correlation of structure with function in the hypothalamus. He had now [1928] become Director of an Institute of Neurology where, with a large group of associates, his entire energies could be devoted to research.

The experimental approach to the hypothalamus had been initiated in 1909 and 1910 by Karplus and Kreidl who reported excitation of the sympathetic system from stimulation of the hypothalamus. Professor Cannon, in a long series of studies, had shown that the entire sympathetic system acts as an integrated mechanism for the expression of fear and rage. The reactions consist of constriction of the blood vessels, chiefly those supplying the abdominal viscera, producing a rise in blood pressure and causing the blood to flow more rapidly through the brain, heart, and skeletal muscles. At the same time there is poured into the blood an increased amount of adrenalin which reduces muscle fatigue, and the amount of sugar is increased to supply the muscles with an abundant source of energy. There is also a dilation of the pupils and an increased rate and depth of respiration. All of these phenomena are associated with the expression of intense emotional excitement. Other functions are repressed, such as a decrease in secretory activity of the stomach and an abolition of peristaltic movements of stomach and intestine. All of these make an integrated mechanism for the expression of fear and rage. In 1928 and 1929 Bard (*Amer. J. Physiol.*, 1928, *84*, 490; *Arch. Neurol. and Psychiat.*, 1929, *22*, 230) showed that "sham rage" associated with an explosive involvement of the entire sympathetic mechanism had its coordi-

nating center in the hypothalamus. In the same year [1928], Hinsey (Ref. 77; see also page 254 in Ref. 159), working with Ranson, observed that one of the cats with decerebrate rigidity in which crossed extensor reflexes were being studied, when left without any restraint, was restless and finally succeeded in getting to its feet and walking, once even a distance of 15 feet. Moreover, in this cat there were alternate periods of quiescence and restlessness. In the experiments of Bard the animal had been restrained. Hinsey and Ranson found (77, 92) that in this animal the cut had not been strictly transverse in the line between midbrain and diencephalon, but that starting at the posterior commissure, the cut had run obliquely forward to the optic chiasm. Thus was preserved the small wedge of tissue which is the hypothalamus. In this case the entire red nucleus, the medial and lateral hypothalamic nuclei and Luys' body were intact. They had therefore found that the hypothalamus is necessary for the maintenance of the upright position and for the rhythmic movements of walking. Subsequently (in 1930, Ref. 92), Hinsey, Ranson, and McNattin found that indeed only a small part of the hypothalamus need be retained to enable a cat to walk. The cut from the rostral border of the superior colliculus (posterior commissure) dorsally need only pass in front of the mammillary bodies ventrally to retain this function. This small wedge covers the extension of the tegmentum of the midbrain, including the red nucleus, into the hypothalamus. They were aware that the meaning of the hypothalamus for this function could not be solved without determining all its relations to other parts of the mechanism of standing, such as the vestibular apparatus and the cerebellum which this small wedge of tissue might keep intact. In subsequent experiments it was found that destroying both red nuclei in the cat did not eliminate the ability to walk (120, 121, 122).

These observations on the hypothalamus as some part of the mechanism for walking were the starting point of the prolonged study of the hypothalamus made by Ranson and his associates. Their method consisted of a survey of the hypothalamus with the Horsley-Clarke stereotaxic apparatus for placing lesions

and inducing stimulations at exact, reproducible areas in the brain. This instrument was described in 1908 by Horsley and Clarke (*Brain,* 1908, *31,* 45). The following year [1909], it was used by Sachs (*Brain,* 1909, *32,* 95) in a study of the thalamus made in Horsley's laboratory. It was brought from England by Dr. Sachs to the laboratory at Washington University in St. Louis, where, many years later [1924-28], Ranson became familiar with its use. Indeed, Ranson and his associates have made most extensive surveys of midbrain, hypothalamus, and other structures of the forebrain with this instrument. Ranson (131) first made a series of charts with orientation planes for both cats' and monkeys' brains and then a series of studies on the pathways for eye reflexes was undertaken. At the time of his death Ranson and his associates were starting to survey the structures of the basal ganglia and tracts of the forebrain, but their most complete surveys were of the hypothalamus.

Ranson finally summarized the modern work on the hypothalamus as follows: The hypothalamus, which is phylogenetically a very ancient part of the diencephalon, exerts its control over a wide series of visceral functions (131, 132, 214). Two different mechanisms are involved, first, fibre connections with brain and cord by which impulses are relayed to the sympathetic system, giving the physical signs of fear and rage. In this division there is some overflow into the somatic system. The hypothalamus also acts as a thermostat for the regulation of body temperature. Second, there is a tract of unmyelinated fibres from hypothalamus to hypophysis, whose impulses control water balance. Both mechanisms act through glands of internal secretion, the first through the adrenal, the second through the posterior lobe of the hypophysis.

In the explorations of the hypothalamus all lesions and all stimulations were made bilaterally. Ranson and his associates found that the most sensitive zone for excitation of the sympathetic system was the lateral hypothalamic zone in the region of the medial forebrain bundle lying between the internal capsule and the fornix. This zone is the middle region of the hypothala-

mus; the most posterior part of the hypothalamus being the center for emotional expression, and its converse somnolence, while the anterior region near the optic chiasm is a specialized part of the mechanism for the regulation of temperature.

Stimulation of the sympathetic region proper, the zone of the medial forebrain bundle, gives regularly a rise in blood pressure due to contraction of the arteries, increased depth of respiration, and dilation of the pupils, all sympathetic responses. If the stimulus of this area is continued, there is an overflow into somatic motor paths. While occasionally a stimulus of the more medial zone in the hypothalamus may give some response of the sympathetic mechanism, it is the lateral zone that uniformly gives sympathetic responses, and more, that is the integrating center for calling the entire sympathetic mechanism into play as described by Cannon.

No corresponding integrating center for parasympathetic responses was found by Ranson in the hypothalamus. Parasympathetic responses, that is, contractions of the bladder, were obtained from the region farther forward in the forebrain surrounding the anterior commissure. Associated with contraction of the bladder there might be a decrease in rate and depth of respiration and sometimes a fall in blood pressure.

Stimulus of the posterior portion of the lateral hypothalamic nuclei, extending backward into the midbrain, gives the symptoms of "sham rage" and, conversely, destruction of this area gives somnolence. Both in cats and in monkeys, the destruction of the area produces a cataleptic state in which there is plastic hypertonus of the muscles. After extensive studies of both phases of these reactions, Ranson concluded that the normal function of the hypothalamus is a drive on lower centers for maintaining the waking state and that the elimination of this drive favors sleep.

The hypothalamus also acts as a thermostat for regulating body temperature. In the anterior part of the hypothalamus, including the preoptic and supraoptic regions, there is a zone which prevents overheating of the body. Extensive experiments with this zone were published in 1943 after Ranson's death (214).

Following lesions in monkeys which destroyed this anterior region, there was a fatal rise in temperature to as high as 109°. This degree of rise could, however, be prevented and the animals saved by pentobarbital, if given in sufficient doses early enough. The rise in temperature was the result of a central paralysis of the heat loss mechanism. The neural mechanism which protects against chilling, that is, which serves to reduce heat loss and probably also to increase heat production (194) is coextensive with the sympathetic center of the hypothalamus.

The function of the hypothalamus in relation to water balance was discussed in a monograph on diabetes insipidus by Fisher, Ingram, and Ranson, (169) published by Edwards Brothers at Ann Arbor in 1938. The mechanism for the maintenance of water balance is a tract of unmyelinated fibres from the hypothalamus carrying secretory fibres to the posterior lobe of the hypophysis. The main supraoptico-hypophyseal tracts arise in supraoptic nuclei just dorsal to the optic chiasm and pass in the wall of the third ventricle on either side into the infundibulum, to end in the posterior lobe of the hypophysis. Smaller bundles of similar fibres arise near the root of the infundibulum. Lesions which cut both supraoptico-hypophyseal tracts are followed by atrophy of the nuclei of origin, the supraoptico nuclei, and by the death of the secretory cells of the posterior lobe of the hypophysis. There result the typical symptoms of diabetes insipidus with marked thirst and the excretion of a great increase of sugar-free urine. The condition is brought about by the loss of the antidiuretic hormone of the posterior lobe of the hypophysis. Typical diabetes insipidus was induced in 85 cats and in 2 monkeys. In the early work on the function of the hypophysis, some confusion resulted from damage to the hypothalamus as well. These accurately placed lesions and stimulations of Ranson and his group have sharpened these localizations. They did not reproduce Fröhlich's syndrome of adiposity associated with sexual under-development by lesions restricted to the hypothalamus.

In summary, they located the area of the hypothalamus which forms the integrating center for the sympathetic system; they

found the areas that make a thermostat for regulating body temperature, they located the center that has to do with the waking-sleeping rhythm, and analyzed the relation of hypothalamus to hypophysis in the control of water balance.

In 1940 the volume on the Hypothalamus of the Research Publications of the Association for Research in Nervous and Mental Disease was dedicated to Ranson with the following inscription:

In recognition of the distinguished contributions
To knowledge of hypothalamic functions
Made by himself and
By the students he has inspired,
This meeting of the association
Is dedicated by the trustees to
STEPHEN WALTER RANSON

KEY TO ABBREVIATIONS USED IN BIBLIOGRAPHY

Am. Heart J. = American Heart Journal
Am. J. Anat. = American Journal of Anatomy
Am. J. Med. Sci. = American Journal of Medical Sciences
Am. J. Obst. and Gynec. = American Journal of Obstetrics and Gynecology
Am. J. Ophthal. = American Journal of Ophthalmology
Am. J. Physiol. = American Journal of Physiology
Am. J. Rel. Psychol. and Ed. = American Journal of Religious Psychology and Education
Anat. Anz. = Anatomischer Anzeiger
Anat. Rec. = Anatomical Record
Ann. Int. Med. = Annals of Internal Medicine
Arch. Int. Med. = Archives of Internal Medicine
Arch. Neurol. and Psychiat. = Archives of Neurology and Psychiatry
Arch. Ophthal. = Archives of Ophthalmology
Arch. Path. = Archives of Pathology
Arch. Surg. = Archives of Surgery
Bull. N. Y. Acad. Med. = Bulletin of the New York Academy of Medicine
Ergebn. d. Physiol. = Ergebnisse der Physiologie biologischen Chemie und experimentellen Pharmakologie
J. A. M. A. = Journal of the American Medical Association
J. Anat. = Journal of Anatomy
J. Biol. Chem. = Journal of Biological Chemistry
J. Comp. Neurol. = Journal of Comparative Neurology
J. Com. Neurol. and Psychol. = Journal of Comparative Neurology and Psychology
J. Exp. Med. = Journal of Experimental Medicine
J. Lab. and Clin. Med. = Journal of Laboratory and Clinical Medicine
J. Nerv. and Ment. Dis. = Journal of Nervous and Mental Diseases
J. Neurophysiol. = Journal of Neurophysiology
J. Neurol. and Psychopath. = Journal of Neurology and Psychopathology
J. Pharmacol. and Exp. Therap. = Journal of Pharmacology and Experimental Therapeutics
Physiol. Rev. = Physiological Reviews
Proc. Inst. Med. Chicago = Proceedings of the Institute of Medicine of Chicago
Proc. Soc. Exp. Biol. and Med. = Proceedings of the Society for Experimental Biology and Medicine
Psychiat. en neurol. bl. = Psychiatrische en Neurologische Bladen
Psychosomat. Med. = Psychosomatic Medicine
Quart. Bull. Northwestern Univ. Med. School = Quarterly Bulletin, Northwestern University Medical School

Res. Publ. Assn. Nerv. Ment. Dis. = Research Publications of the Association for Research in Nervous and Mental Diseases
Rev. neurol. = Revue neurologique
Rev. Neurol. and Psychiat. = Review of Neurology and Psychiatry
Trans. Am. Neurol. Assn. = Transactions of the American Neurological Association
Trans. Chicago Path. Soc. = Transactions of the Chicago Pathological Society
Trans. Coll. Phys. of Phila. = Transactions of the College of Physicians of Philadelphia.

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(Note: References 213 and 214 were published in 1943 after the death of Professor Ranson.)