THE NEUROLOGY OF JEAN CRUVEILHIER*

by

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TODAY Jean Cruveilhier is best remembered as an early professor of pathology, as the author of the two-volume atlas *L'Anatomie pathologique du Corps Humain,* and for his theoretical views on the role of phlebitis and inflammation in general disease processes.[13] Works dealing with the history of pathology emphasize his views on inflammation and mention several conditions of which he gave the first or an early description—peptic ulcer, multiple sclerosis, and progressive muscular atrophy.[19,23,26] To those interested in the nervous system, Cruveilhier's *Anatomie pathologique* is a storehouse of fascinating cases equalled only by Morgagni's *De Sedibus.* The magnificent illustrations make the work unique in all the literature of pathology for clarity and accuracy of presentation as well as aesthetic appeal.

His theories of disease processes epitomized by 'phlebitis dominates all pathology' have not survived the development of medicine in the century since his death,[1,19] but his descriptions of certain neurological conditions both clinically and pathologically are as accurate and interesting as any case report today. His illustrations surpass many subsequent ones in detail and beauty. In addition to the superb illustrations of many of these conditions, Cruveilhier has added extensive clinical material for most of the cases and summarizes these with his own comments on the patients' histories, physical findings, and autopsies. He organizes this material in such a way that certain patterns useful in arriving at diagnoses can be made. Again and again he stresses the importance and absolute necessity of correlating autopsy findings with events in the history and clinical examination.

This extensive commentary, not present in the other early nineteenth-century illustrated works of neuropathology, sets his atlas apart from the others. Contemporary books by Hooper, Bright, and Carswell contain superb illustrations of important central nervous system lesions, but none of these writers displays the clinical acumen and insight that Cruveilhier does in arriving at accurate clinical-pathological correlations.[7,11,22] These clinical observations by Cruveilhier have not received the attention they deserve in several works dealing with the history of neurology and neurosurgery.[21,29,35]

Certain problems arise in evaluating a work of this type. For one thing it was published over a thirteen-year period from 1829 to 1842 and reflects certain changes in the author's opinions that are difficult to summarize. Also, the work is devoid of any literary organization. Diseases of different types and different organs are included in the same divisions of Livraisons. In sections on spinal cord disease, Cruveilhier makes digressions to discuss hemiplegia and speech disorders.

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Another problem common to all works of this period is the lack of understanding of many disease processes such as infection and neoplasia that are so fundamental to present-day medicine. For most writers of this period, inflammation was the basis for such diverse diseases as apoplexy, infarction, tumours, and even epilepsy.[2,6,13,24,32]

The illustrations are, nevertheless, clear enough and many of the case reports contain enough information to allow us to arrive at our own diagnoses. More important is the fact that Cruveilhier presents this material in a way that shows his understanding of certain basic differences in neurologic conditions. For the most part, he is able to distinguish between patients with hemiplegia from a stroke and from a tumour. In cases of spinal cord disease, he distinguishes between lesions of the cord itself and lesions compressing the cord.

We shall also see where he is successful and where he fails to bring together the knowledge of the different disciplines of anatomy, physiology, and pathology that today form an integral part of the whole field of neurology.

A biography of Cruveilhier has been written by Delhoume, and his life is summarized in many works on the history of medicine.[1,17,21,26] He was born in Limoges in 1791, the son of a military surgeon. Although he wished to enter the church, he began medical studies in Paris in 1810 at the insistence of his father. A perhaps apocryphal story has it that he was so repulsed by his first sight of autopsies that he gave up medicine and returned to theology.[23] Once again, his father intervened, and he returned to his medical studies. He received his degree in 1816 and settled in Limoges. In 1823, he was appointed Professor of Surgery at Montpellier through the influence of Dupuytren. In 1825, he returned to Paris as Professor of Descriptive Anatomy. In his will, Dupuytren left funds to establish a professorship in pathologic anatomy, the second such chair created. Cruveilhier was the first to be appointed to this chair in 1836 and held it for the next thirty years. In addition to his work in pathology at the Charité and particularly at the Salpêtrière, he was involved in a very active clinical practice in Paris. He died in 1874.

In this paper, I shall discuss some interesting cases of neuropathology presented by Cruveilhier. Many of these represent the first reports of these conditions. I shall consider Cruveilhier's relation to the new neurophysiology that was developing at the time he wrote. We shall see which aspects he made use of as a clinician and which aspects of experimental work had no applicability to the problems he encountered in the clinic. I shall also discuss Cruveilhier as a clinical neurologist in the sense of a physician capable of organizing different findings of a patient in such a way as to arrive at a diagnosis based on neuroanatomy and neurophysiology. In doing this, we shall look briefly at the works of some other men interested in clinical neurology in the first half of the nineteenth century.

Certain conventions of early nineteenth-century neurology must be tacitly observed if we are to gain any insight into Cruveilhier's method of evaluating signs and symptoms. Gall died in 1828, the year before the first volume of Cruveilhier's atlas appeared. His influence can be seen again and again in the writings of Cruveilhier and others of this period.[37] Phrenology had received some objective support from Bouillaud in 1825 when he re-emphasized Gall's contention that the faculty of speech was located in the frontal lobes.[6] Bouillaud based his opinion on actual post-mortem

studies of the brain; he also stressed the necessity of examining the brain in all cases before arriving at conclusions of localization. Because of Gall and Bouillaud, the location of the faculty of articulation as it was called received a considerable amount of attention. Many writers opposed to phrenology went to great lengths to cite cases with speech deficits with lesions in places other than where predicted by the phrenologists.[24,33,37] Cruveilhier states (Livraison 33, plate 2): 'The faculty of articulation of sound does not have a special location in the brain. It ceases whenever there is a large destruction of cerebral tissue whatever the site is—thalamus, striatum, pons, or centrum ovale of either hemisphere.'

As did others of this period, Cruveilhier felt that the loss of speech could occur from loss of memory of things, loss of memory of words, or the inability to articulate sounds.[6,24,37] Most writers agreed that speech loss was due to this last deficit. This represents a loss of a specific motor function, and it is interesting that even Broca in 1861 continued this view of speech loss as a motor rather than a cognitive defect.[8,9,37]

A major figure in French neurophysiology of this period was Flourens. Born in 1794, he was a contemporary of Cruveilhier. His work was summarized and published in 1824.[18] Based on experiments in birds and frogs, he concluded that the cortex was involved in vision, volition, and sensation but not for the initiation of motor function. For Flourens, the cortex was involved in intellectual functions as a unit without regional specificity. The highest centre of motor activity was the striatum. This concept was to influence neurologic thought until 1870; it can be seen in the early writings of Hughlings Jackson.[34,37] The other major observation of Flourens was the role of the cerebellum in the regulation of motor activity. Although today we accept this as Flourens' lasting contribution, it was not as highly regarded by the clinical neurologists we shall discuss.

For Gall, the cerebellum was the seat of sexual functions. This position is maintained by Ollivier in his work on the diseases of the spinal cord.[31] He reports several cases of quadriplegia with associated priapism. This he attributes to an injury of the cerebellum at the time of the fall that produced the cervical fracture and quadriplegia. A more general work on diseases of the brain by Lallemand was published at this time.[24] Although patients with cerebellar lesions are presented, no correlation is made between this site of a lesion and any of the symptoms encountered.

Cruveilhier does not accept the cerebellum as the seat of the faculty of sexual desires, nor does he seem to be influenced by Flourens' work on this part of the brain. He presents several cases of cerebellar tuberculoma. One patient, a soldier aged twenty-one, found to have a tuberculoma of the cerebellum, claimed to have no sexual desires or interests for two years. Although Cruveilhier does not mention Gall by name, he says that the 'absence of sexual desires is against the ideas of certain physiologists that the cerebellum is the seat of this faculty' (Livraison 18, Plate 2).

Cruveilhier reached no conclusion himself about cerebellar function and attributed no specific symptoms to lesions there. Although he mentions irregularity of gait and ataxia in patients with cerebellar tuberculomas, he seems to attribute this more to the tuberculous process than to the location of the lesions. When he discusses tumours arising from the dura of the petrous bone, he says: 'I have not observed particular symptoms from compression of the cerebellum.' (Livraison 8, Plates 1, 2, 3.)

This lack of appreciation of cerebellar function is seen in other places (Livraison 26, Plate 2). Cruveilhier presents a very detailed history and physical examination of a twenty-six-year-old woman with an acoustic neurinoma that is splendidly illustrated. Almost all of the signs and symptoms of a tumour of the cerebellar pontine angle are presented and discussed—all except signs of cerebellar deficit. No mention is made of any defect in balance or co-ordination even though the illustrations and description of the tumour show that the brain stem and cerebellum were compressed by the tumour.

I can offer no explanation for Cruveilhier's failure to appreciate Flourens' work unless he felt as Ollivier did that work done in birds ought not to be applied to man. Why the experimental work of Magendie was so readily accepted and that of Flourens not, will require further study of the interrelationships of the different members of the Paris School of Medicine.

The outstanding development of this period in the understanding of the functional organization of the nervous system was the work of Bell and Magendie.[4,5,27] Without entering into the question of priority of these two men, I would note that both of them are always mentioned together in the contemporary French works dealing with the physiology of the spinal cord. It is certainly to the credit of such men as Ollivier and Cruveilhier, writing only a few years after the papers of Bell and Magendie, that they fully appreciated and accepted the significance of the revolutionary concept of a separate path for input and output in the nervous system. Ollivier published a book on diseases of the spinal cord in 1824.[31] He uses this new physiologic principle to explain many of the findings in the cases he presents. It is of interest to compare this work with Cruveilhier to see the differences in their approaches to diseases of the spinal cord.

In keeping with the tradition of nosology established by the Paris School, Cruveilhier presents a very cogent classification of spinal cord lesions that produce paraplegia. He lists four categories (Livraison 35, Plate 6): (1) paraplegia by alteration of the cord itself; (2) paraplegia by compression; (3) paraplegia by inflammation; (4) false paraplegia due to rigidity of the joint.

Such a grouping is not very different from that used by a neurologist today to establish a differential diagnosis of paraplegia. Cruveilhier adds that the distinction between extrinsic and intrinsic lesions is important since there is a chance of benefit from surgical intervention in the former.

To make this distinction, Cruveilhier suggests that compressive lesions are painful and intrinsic lesions are not. 'I conclude that painful paraplegia is always due to compression and non-painful paraplegia due to diseases of the cord itself.

'One concludes that paraplegia from compression is painful only when it develops slowly. Paraplegia from fracture or subluxation is not painful. Only after severe compression and disruption of the cord does pain disappear, and this may produce confusion in the symptoms.' (Livraison 35, Plate 6).

In the first half of the nineteenth century, the organization of the ascending and descending tracts of the cord was not understood. It was believed that some sensation was carried in the posterior columns. This correlated well with the differentiation by Bell and Magendie of the functions of the anterior and posterior spinal roots.[4,5,27]

Their ideas were extended to the cord itself; the anterior portion of the cord was believed to be motor in function and the posterior portion sensory.

Although the decussation of the descending corticospinal tracts was known, both Cruveilhier and Ollivier were confused by cases with loss of sensation contralateral to the paralysis and the lesion in the cord. This had to wait for Brown-Séquard's work in 1851 for clarification.[10]

The distinction between motor and sensory function of the anterior and posterior halves of the cord is seen in both Ollivier and Cruveilhier. As an example, Ollivier presents a case of a soldier shot in the lumbar region. He had no detectable motor deficit but was numb in the region of his buttock, inner thighs, and genitalia. He interprets these findings as the result of a lesion of the posterior cord or posterior roots without reference to the fact that the entrance wound was below the level of the cord.

Cruveilhier presents a case of a sixty-year-old woman with progressive paraplegia and spasticity but no loss of sensation (Livraison 32, Plates 1, 2). This is explained by the autopsy findings of a tumour arising from the dura anterior to the spinal cord at the lower thoracic level. In Cruveilhier's opinion, this tumour resembled those that arise from the cranial dura. The plate shows a characteristic spinal meningioma probably the first one presented in the literature.

Another aspect of spinal cord organization that was not well appreciated at this time was the relationship between the different levels of the cord and the total integration with the entire central nervous system. In 1812, Legallois reported his experiments on animals in whom he had sectioned the spinal cord at different levels.[25] Although not aware of the reflex arc, he found that local phenomena for isolated sections of the spinal cord were possible but that these were not integrated with the rest of the nervous system.

For Ollivier, this served as the basis for explaining a case of a man who was found to have a complete division of his spinal cord from a bullet wound but who in life had had normal function of his legs! Cruveilhier makes no mention of Legallois but indicates that he has some awareness of the segmental arrangement of the spinal cord. He states that weakness of the legs can be produced by lesions at the thoracic as well as the lumbar level of the cord (Livraison 32, Plates 1, 2).

Both Ollivier and Cruveilhier seem aware of Legallois' experiments in which he localized the control of respiration to the spinal cord at the cervico-medullary junction. They both present cases of high cervical fractures with death from respiratory failure and not from cardiac arrest just as Legallois had shown experimentally (Livraison 25, Plate 4).

What remains as the major difference between Ollivier and Cruveilhier in their writings on the spinal cord is the attempt of Cruveilhier to form a classification that would be of value in prognosis; this is certainly not the approach we usually expect from a pathologist. Ollivier on the other hand grouped his cases in a more typical fashion according to the types of lesions and not by the mechanisms of production of symptoms. In this sense, Ollivier's book is more the work of a pathologist and Cruveilhier's that of a clinician.

One further aspect of the signs and symptoms of spinal cord disease deserves

comment and serves to emphasize Cruveilhier's position as an early neurologist as well as a pathologist. Only after the different tracts of the spinal cord were delineated in the second half of the nineteenth century did it become necessary to study the different modalities of sensation. Before this time, sensation was considered as a single function except at the peripheral level where Müller had begun to make separations.[30] No mention is made by Ollivier of his method of testing sensation. Cruveilhier goes into considerable detail about this. He points out that loss of sensation is not the same for all modalities (Livraison 38, Plate 5). There are patients who will not perceive a very hard pinch of the skin but who feel quite well a tickle with a feather. He also tests for perception of both cold and warmth. He is aware that paraesthesias may be present in patients who have lost all sensation on objective testing. He comments on another alteration of sensation in which the perception of the stimulus is delayed for 15–30 seconds. He also observes summation of stimuli when patients who do not perceive a single pin stick feel the pin if they are stuck three or four times in rapid succession.

Although Cruveilhier could not at this time bring these observations on sensation together into a meaningful theory, one must conclude that he had a considerable amount of experience with and appreciation of spinal cord problems.

This is borne out when we consider some of the diseases of the spinal cord that he distinguished as separate entities. Several patients are presented in whom paraplegia existed and in whom a 'grey degeneration or transformation' was found throughout the spinal cord, brain stem, and cerebellum (Livraison 32, Plates 1, 2; Livraison 38, Plate 5). The lesions were small, multiple, and denser than the tissue of the spinal cord. Cruveilhier felt that this was a new disease entity. Since then, writers have considered these cases as the earliest descriptions of multiple sclerosis.[19,21,29] Although the lesions seem appropriate for this diagnosis, the absence in the histories of exacerbations and remissions and the absence of visual symptoms seem unusual. These criteria, so important to the diagnosis of multiple sclerosis today, are strikingly absent in Cruveilhier's cases. In one case, blindness and paraplegia existed in a woman found to have this grey degeneration; this may be a case of Devic's disease although the history is not an acute one.

Two cases of young women aged eighteen and twenty-one years are presented to show paraplegia due to inflammation and meningitis, the third type of paraplegia in Cruveilhier's classification (Livraison 32, Plates 1, 2). The cases described showed a gradual onset over several weeks of weakness with ascent of the dysfunction up the neuraxis. Recovery took place over several months with gradual improvement. Both of these cases are presented as recoveries from meningitis. To the modern reader, they seem more like ascending polyneuritis of the type described by Guillain, Barré, and Strohl.[20]

To conclude this discussion of the spinal cord, I will only mention some of the other cases that show the extent of Cruveilhier's interest in diseases of the spinal cord. Superb illustrations of cases of Pott's disease (Livraison 4, Plate 4), hydatid cyst (Livraison 35, Plate 5), apoplexy in the cord (Livraison 3, Plate 6), and subluxation of C1 on C2 are presented (Livraison 25, Plate 4). At a later date, Cruveilhier presented an early description of the pathologic findings in progressive muscular atrophy.[14]

If we are to support our contention that Cruveilhier was a competent neurologist, we must look at other types of lesions that he presented. If we consider Cruveilhier's cases of congenital and neonatal malformations and spinal cord disease, several important aspects are found. Among the many congenital conditions that Cruveilhier presents, several stand out because of the accuracy of his illustrations. A case of congenital absence of the cerebellum is presented (Livraison 15, Plate 5). This is well illustrated, but there is no discussion of the significance of this lesion.

He presents a typical case of infantile hemiplegia (Livraison 8, Plate 5). The illustration of the brain shows extensive atrophy in the distribution of the middle cerebral artery. The brain stem shows the characteristic atrophy of the corticobulbar and corticospinal tracts. The associated changes in the skull and the development of the frontal sinuses are also shown in great detail.

Four sections are devoted to hydrocephalus and spina bifida. Two cases of meningomyelocoele with the clinical courses are discussed (Livraison 6, Plate 3). The first patient was observed from the age of three days until death from meningitis two weeks later. The autopsy description includes observations of the flattened gyri and sulci, massive ventricular dilatation, and pus in the ventricles which Cruveilhier assumes spread there from the spinal subarachnoid space through 'a large opening so well described by M. Magendie'. This is probably one of the earliest references to the foramen of Magendie which was originally described in 1825.[28]

The second case is of even more interest in terms of Cruveilhier's early observations of neurologic lesions. Again, it is a case of meningomyelocoele with death from meningitis. The autopsy disclosed the meningocoele, the boney anomalies of the spina bifida, and an associated diastometamyelia. It is the description of the cervical region and the posterior fossa that is of particular interest. '. . . the upper part of the cervical region, considerably enlarged, contained both the medulla oblongata and the corresponding parts of the cerebellum which was elongated and covered the fourth ventricle which itself became longer and wider.' Cruveilhier goes on to mention two other cases with '. . . this type of descent of the elongated medulla and cerebellum into the upper part of the spinal canal.' This is certainly a description of the abnormality that Chiari reported more than fifty years later and is now known as the Arnold-Chiari malformation.[12] Cruveilhier also comments on the presence of pus in the ventricles but not over the convexities of the brain; this, too, is one of the well-known findings in this type of communicating hydrocephalus.

Cruveilhier summarizes his observations on spina bifida and makes the profound observation that the children remained healthy so long as the meningocoele remained closed (Livraison 16, Plate 4). Once it is opened, fever, convulsions, paraplegia, and death ensue in several hours to a few days. This leads him to conclude, not unwisely, that the celebrated case of Sir Astley Cooper, successfully treated by repeated punctures of the sac, was only a fortunate exception.

Another major group of lesions of the nervous system that Cruveilhier deals with is cerebral vascular disease. In the first half of the nineteenth century, this consisted of two types—haemorrhage and infarction; the concept of embolism was not appreciated. Wepfer in 1658 was the first to point out the relationship between cerebral haemorrhage and the commonly observed clinical phenomena grouped together under

the heading of apoplexy.[36] The appreciation of infarction or softening as it was called was a more recent event. In 1820, Rostan published his work that marks the beginning of interest in this aspect of cerebral vascular disease.[33] A work by Lallemand appeared over the next few years.[24] He too presented cases of softening and cited earlier cases from the literature particularly from Morgagni. Rather than maintaining the separate nature and etiology of apoplexy and softening, Lallemand regarded them as part of the same disease process which also included inflammation. For Lallemand, softening was a type of inflammation and was between apoplexy and inflammation in the same disease spectrum.

This was by no means a universally accepted position. Rostan did not consider inflammation and softening together. Andral, writing at the same time as Cruveilhier, did not believe as Lallemand did that softening was always associated with inflammation.[2]

In Cruveilhier's writing, we see an attempt to distinguish between these two conditions by clinical and pathological criteria. He points out the differences that are today still valid between red and white infarcts (Livraison 33, Plate 2). His clinical distinction between these two conditions is based on his opinion that haemorrhagic lesions have a more abrupt onset in their clinical presentation than softening. He also feels that in haemorrhage the motor system is principally affected in contrast to softening where mental changes are also noted. Although his clinical criteria today do not seem satisfactory, it is clear that Cruveilhier appreciated the fact that two different processes were involved.

Elsewhere, he details the method by which he arrives at a diagnosis of apoplexy and how he successfully identifies the site of the lesion (Livraison 5, Plate 6). He describes a man aged fifty-two who suddenly became unconscious. When Cruveilhier examined him, he found a complete right hemiplegia and sensory loss. The tongue was deviated towards the paralysed side. He adds that the patient was known to have frequent headaches for some time before the ictus.

Cruveilhier makes the diagnosis of a very large apoplectic cavity in the left thalamus with probable communication of the clot with the lateral ventricle. These were essentially the findings at autopsy; also found was an old cystic lesion in the region of the insula. In discussing his reasoning in arriving at the precise diagnosis, he says he favoured a thalamic haemorrhage because this is the most frequent lesion in this type of case with the sudden onset of hemiplegia. It is haemorrhage that produces the most profound hemiplegia. He goes on to say that he diagnosed a communication with the ventricle because of the coma and unresponsiveness of the patient. His prognosis had been grave because loss of consciousness was present from the onset of the illness: 'There is an enormous difference between apoplexy with loss of consciousness at the moment of the attack and an apoplexy without loss of consciousness.' To support this, he points out that this patient had an old scar from a previous haemorrhage. He presents other cases with scars in different locations to indicate that apoplexy can occur without being fatal.

I believe that this case represents Cruveilhier at his best as a clinical neurologist. It also shows the type of reasoning that would be used in the next fifty years in the development of the field of neurology with accurate diagnosis based on anatomical and temporal relationships.

Cruveilhier is also aware that the variations in the clinical course of patients with apoplexy depends more on the location and size of the lesion than on differences in the disease processes. In the same section, he presents another case of an elderly woman who developed a sudden hemiparesis but no loss of consciousness. At autopsy, she had a large haemorrhage in the centrum ovale. Cruveilhier explains the partial weakness, preservation of consciousness and sensation in a case with a large clot by pointing out that the lesion was located in the white matter. He goes on to say that: '. . . an apoplexy 10 times smaller located in the corpus striatum or thalamus would have produced a complete motor paralysis and perhaps even [loss of] sensation.'

Cruveilhier distinguishes another type of haemorrhagic lesion but admits that one cannot always be sure of the diagnosis. This is the brain stem haemorrhage. He characterizes it by sudden collapse of the patient, complete loss of movement and sensation, and rapid death (Livraison 21, Plate 5). Although the bilateral loss of function is often seen with lesions of the brain stem, he cautions that one cannot assume that every rapid demise of this sort is due to a pontine haemorrhage. Another interesting note in these cases which we recognize today as related to hypertension is the inclusion by Cruveilhier of a description of the heart at autopsy. Like Rostan, Cruveilhier also noted the frequency of left ventricular hypertrophy in cases of cerebral apoplexy.

The final group of cases I should like to consider is brain tumours. It is here that Cruveilhier sets himself apart from his contemporaries and shows sophistication and insight into the mechanisms of neurologic disease. In no book of this period or before are so many cases of intracranial tumours presented. They are not just case reports, but represent a cogent analysis of the mechanisms by which symptoms are produced. This important contribution of Cruveilhier has been passed over in the usual works on the history of neurologic disorders.[29,35] To this discussion are added illustrations of tumours that have never been surpassed in beauty or accuracy. In no other work until the twentieth century do we find such an attempt to define the various syndromes produced by tumours specific to certain locations. Although there is no histologic confirmation of the nature of the tumours, the presentations are so accurate that little difficulty is encountered in recognizing the nature of them. When Cruveilhier discusses tumours in the brain, he includes such lesions as tuberculomas, abscesses, as well as true neoplasms.

Harvey Cushing was the first to call attention to the accuracy of Cruveilhier's observations on brain tumours. In his classical works on acoustic neurinoma in 1917 and meningioma in 1938, he included portions of Cruveilhier's reports and reproduced some of the illustrations.[15,16]

I have already referred to the case of an acoustic neurinoma (Livraison 26, Plate 2). Other than the lack of awareness of cerebellar symptoms, Cruveilhier reports the findings of his examination of the patient and correlates them accurately with the autopsy. What I believe is of the utmost significance is the manner in which he organizes the different signs and symptoms. These are due to compression of two types: (1) direct compression of the structures adjacent to the tumour; (2) indirect compression of the remainder of the brain—in other words, the effects of increased intracranial pressure. It is just such a consideration that is used by neurologists and

neurosurgeons today when they evaluate patients with intracranial masses. Cruveilhier adds that this distinction is applicable not only to cases with tumours of the acoustic nerve but to all cases with intracranial tumours. The patient with the acoustic neurinoma was a woman aged twenty-six whose symptoms began at the age of nineteen. She gradually and progressively suffered from increasing headache, loss of hearing, and loss of vision. Also noted were facial spasms, paraesthesias and numbness of the face, rigidity of the extremities, and anosmia.

Cruveilhier's diagnosis of a tumour at the base of the brain was fully substantiated by autopsy. The illustration shows a tumour of the eighth nerve with destruction of the petrous bone. In correlating the symptoms with the autopsy findings, Cruveilhier relates the deafness, facial spasms and numbness, and the rigidity and weakness of the extremities to direct compression of the appropriate cranial nerves and the brain stem. Loss of vision and smell and the severe headaches are attributed to the generalized effects of increased intracranial pressure. At the end, he presents the first description and still a fairly accurate one of the syndrome of a cerebellopontine angle tumour. 'Amaurosis with loss of the olfactory sense and the sense of hearing on one side, accompanied by numbness of the facial muscles on the same side, characterizes tumours which originate on the posterior face of the petrous bone and particularly those tumours originating in the internal auditory passage.' (Cushing's translation[15]).

Another fascinating case of a brain tumour that Cruveilhier presents, again for the first time, is that of an eighteen-year-old female with an epidermoid or, as he calls it, a pearly tumour arising from the base of the skull beneath the third ventricle.[8] The tumour is well illustrated, and the displacement and destruction of the adjacent brain are also shown (Livraison 2, Plate 6). What concerns Cruveilhier is the fact that the girl was relatively free of symptoms until shortly before her admission to the hospital, although the tumour had obviously been present for a longer time. This he explains by pointing out that there is a great difference in the effects of a mass that develops slowly and allows the brain to accommodate to the compression and a mass such as a haematoma that develops acutely. This is another point emphasized by Cruveilhier in many places that still forms a basic principle used to evaluate patients with diseases of the nervous system.

The final group of tumours that I wish to consider is the meningiomas. Cruveilhier deals with these lesions in several places (Livraison 8, Plates 1, 2, 3; Livraison 25, Plate 2; Livraison 33, Plate 3). Although he refers to them as cancerous or carcinomatous tumours of the dura, the many illustrations clearly show that he is referring to meningiomas and that he appreciates the many forms they take both in origin and location.

He points out that these tumours can arise from the dura or the arachnoid. Although they can arise anywhere in the skull (or the spinal canal, *vide supra*), special locations exist in which they are more frequently found. He lists the olfactory groove, sella turcica, and posterior superior surface of the petrous bone as preferred sites (Livraison 8, Plates 1, 2, 3). He points out that symptoms result from compression of structures adjacent to the tumour if these portions of the brain are necessary for observable functions. He seems quite aware of certain 'silent areas' in the brain. If compression is gradual, the brain accommodates to the mass and undergoes atrophy. If the portion

of the brain compressed or atrophic is not necessary for motor, sensory, or intellectual functions, no symptoms result. The frontal lobe is such an area. Again, he dismisses Gall's views of cerebral localization and cites several cases with extensive compromise of a frontal lobe and no perceptible intellectual deficit.

To distinguish between symptoms of a tumour and those of apoplexy, Cruveilhier emphasizes that those produced by a tumour have a 'marche graduelle' and this progression allows one to distinguish a hemiplegia caused by a tumour from the sudden hemiplegia of apoplexy. One other point that Cruveilhier makes about the occurrence of symptoms from tumours is that they develop not from the tumour itself but from the changes that result in the surrounding brain. He is also aware that tumours produce epilepsy more often than haemorrhagic lesions. These are still statements that form the basis of our present-day approach to the evaluation of patients with neurologic disease.

If we make exception for his concepts of inflammation and irritation, the following passage clearly shows Cruveilhier's views on brain tumours and their clinical effects. It also serves to establish his place as one of the founders of neurology not only for his descriptions of neuropathology but his understanding of the dynamic processes that produce signs and symptoms of neurologic dysfunction.

> Carcinomatous tumours of the dura mater are lesions rather frequently found in the elderly. The symptoms that result are sometimes of no consequence, sometimes the effect of compression, and other times the effect of irritation. They are of no consequence when the tumour is not very extensive or located in such a way as to cause only slight compression of the brain. There are symptoms of compression when the tumour has acquired a certain volume. I will remark that this compression is a consideration of the volume of the tumour and its location. The question of location must always be given great consideration when it is a question of estimating the effects of the compression. The effects of compression are at the same time general, that is to say they act on the entire brain, and local, that is to say they act on a part of the brain. These general and local effects vary according to the site. The local effects can predominate over the general and the converse. There is irritation when the portion of the brain against which the tumour lies becomes congested. This can be transitory and then the effects are temporary or epileptiform and then the effects are lasting (Livraison 33, Plate 3).

In this review of Cruveilhier and some of his contemporaries, I have necessarily selected only certain aspects of a very large work for emphasis. In doing this, I have obviously neglected Cruveilhier's views on inflammation, phlebitis, and certain general characteristics of disease processes which today are not accepted. I have not discussed many of the other types of cases of neurologic disease that Cruveilhier presents. Brief mention should be made of some of them since they emphasize the special interest that he took in diseases of the nervous system. There are illustrations of a vertebral artery aneurysm (Livraison 28, Plate 3), purulent meningitis (Livraison 6, Plates 1, 2), thrombosis of the superior sagittal sinus (Livraison 8, Plate 4), tumours of the calvarium (Livraison 3, Plate 4; Livraison 21, Plate 1), and several lesions of peripheral nerves (Livraison 1, Plate 3; Livraison 35, Plate 2). For the most part, these are simply case reports. They do not contain discussions of pathophysiology that I have tried to present in this paper. I do feel that there is sufficient material in *L'Anatomie pathologique* that allows us to make this type of selection without creating too biased a view of Cruveilhier's role in the history of neurology.

Although some of the cases I have discussed are unique and original reports of these conditions, it is not for this reason alone that Cruveilhier is important. Of more significance today is his method of establishing appropriate correlations between the patient's findings and the autopsy results. From this, he was able to develop several classifications that show his understanding of the mechanisms by which neurologic lesions produce symptoms, as well as an awareness of the organization of the nervous system that was clearly in advance of his times.

I have discussed cases of spinal cord disease that produced paraplegia by compression of the cord, by intrinsic lesions of the cord, and by inflammation. These groups are still seen today. In dealing with cerebral vascular disease, he seemed aware that it often was part of a systemic process as evidenced by the changes seen in the heart. He was aware of the significance of the rate of development of symptoms and the meaning of their severity in terms of localization of the lesion. With brain tumours, Cruveilhier established syndromes of diagnostic value for masses in the cerebellopontine angle as well as meningiomas in other frequently encountered locations. Most important of all was his understanding of the effects of a brain tumour—the direct effects of compression and the remote effects of increased intracranial pressure. It is here that Cruveilhier establishes himself as one of the founders of modern neurologic diagnosis.

It is interesting to note that neurology at the Salpêtrière, so well known for its later nineteenth-century figures like Charcot, had a true foundation established by the first professor of pathology there—Jean Cruveilhier.

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REFERENCES

1. Ackerknecht, E. H., *Medicine at the Paris Hospital (1794–1848)*, Baltimore, Johns Hopkins Press, 1967.
2. Andral, G., *Précis d'Anatomie pathologique*, Paris, 1829.
3. Bailey, P., 'Cruveilhier's "tumeurs perlées" ', *Surgery Gynec. Obstet.*, 1920, **31**, 390–401.
4. Bell, C., *Idea of a New Anatomy of the Brain*, London, 1811.
5. Bell, C., 'On the nerves: giving an account of some experiments on their structure and functions which lead to a new arrangement of the system', *Phil. Trans. R. Soc. Lond.*, 1821, **111**, 398–424.
6. Boillaud, J. B., *Traité clinique et physiologique de l'Encéphalite ou Inflammation du Cerveau*, Paris, 1825.
7. Bright, R., *Reports of Medical Cases selected with a View of illustrating the Symptoms and Cure of Diseases by Reference to Morbid Anatomy*, London, 1827–1831.
8. Broca, P., 'Perte de la parole; ramollissement chronique et destruction partielle du lobe antérieur gauche du cerveau', *Bull. Soc. Anthrop. (Paris)*, 1861, **2**, 235–38.
9. Broca, P., 'Remarques sur le siège de la faculté du langage articulé, suivi d'une observation d'aphémie (perte de la parole)', *Bull. Soc. Anat. Paris*, 1861, **36**, 330.
10. Brown-Sequard, C. E., 'De la transmission croisée des impressions sensitives par la moelle épinière', *C.r. Soc. Biol., Paris*, 1851, **2**, 33–34.
11. Carswell, R., *Pathological Anatomy—Illustrations of the Elementary Forms of Disease*, London, 1838.

12. CHIARI, H., 'Uber Veränderungen des Kleinhirns, des Pons und der medulla oblongata in Folge von congenitaler Hydrocephalie des Grosshirns', *Dtsch. Med. Wschr.*, 1891, **27**, 1172.
13. CRUVEILHIER, J., *L'Anatomie pathologique du Corps humain*, Paris, 1829–1842.
14. CRUVEILHIER, J., 'Sur la paralyse musculaire progressive atrophique', *Bull. Acad. Med.*, 1852–1853, **18**, 490–502, 546–83.
15. CUSHING, H., *Tumors of the Nervus Acousticus and the Syndrome of the Cerebellopontine Angle*, Philadelphia, Saunders, 1917.
16. CUSHING, H., EISENHARDT, L., *Meningiomas. Their Classification, Regional Behaviour, Life History, and Surgical End Results*, Springfield, Ill., Thomas, 1938.
17. DELHOUME, L., *L'Ecole de Dupuytren: Jean Cruveilhier*, Paris, Baillière, 1937.
18. FLOURENS, P., *Recherches expérimentales sur les Propriétés et les Fonctions du Système nerveux dans les Animaux vertébrés*, Paris, 1824.
19. GARRISON, F. H., *An Introduction to the History of Medicine*, 4th ed., Philadelphia, Saunders, 1929.
20. GUILLAIN, G., BARRE, J. A. and STROHL, A., 'Sur un syndrome de radiculonévrite avec hyperalbuminose du liquide cephalorachidien sans réaction cellulaire', *Bull. Soc. Méd. Hôp. (Paris)*, 1916, **40**, 1462–70.
21. HAYMAKER, W. and SCHILLER, F. (eds.), *The Founders of Neurology*, 2nd ed., Springfield, Ill., Thomas, 1970, pp. 322–25.
22. HOOPER, R., *The Morbid Anatomy of the Human Brain*, London, 1828.
23. KRUMBHAAR, E. B., *Pathology*, New York, Hoeber, 1937.
24. LALLEMAND, F., *Recherches anatomico-pathologique sur l'Encéphale et ses Dépendances*, Paris, 1820–1824.
25. LEGALLOIS, C. J. J. C., *Expériences sur le Principe de la Vie notamment sur celui des Mouvements du Coeur et sur la Siège de ce Principe*, Paris, 1812.
26. LONG, E. R., *A History of Pathology*, New York, Dover, 1965.
27. MAGENDIE, F., 'Expériences sur les fonctions des racines des nerfs rachidiens', *J. Physiol. exp. Path.*, 1822, **2**, 276–79, 366–71.
28. MAGENDIE, F., 'Mémoire sur un liquide qui se trouve dans le crâne et le canal vertébral de l'homme et des animaux mammifères', *J. Physiol. exp. Path.*, 1825, **5**, 27–37.
29. MCHENRY, L. C, Jr., *Garrison's History of Neurology*, Springfield, Ill., Thomas, 1969.
30. MÜLLER, J., *Handbuch der Physiologie*, Coblenz, 1840.
31. OLLIVIER (D'ANGERS), C. P., *De la Moelle épinière et de ses Maladies*, Paris, 1824.
32. PRICHARD, J. C., *A Treatise on Diseases of the Nervous System. Part the First: Comprising convulsive and maniacal affections*, London, 1822.
33. ROSTAN, L. I., *Recherches sur le Ramollisement du Cerveau*, Paris, 1820.
34. TEMKIN, O., *The Falling Sickness*, 2nd ed., Baltimore, Johns Hopkins Press, 1971.
35. WALKER, A. E, *A History of Neurological Surgery*, Baltimore, Johns Hopkins Press, 1951.
36. WEPFER, J. J., *Observationes anatomicae ex cadaveribus eorum, quos sustulit apoplexia*, Schaffhausen, 1658.
37. YOUNG, R. M., *Mind, Brain and Adaptation in the Nineteenth Century*, Oxford, Clarendon Press, 1970.