TUMOR INVOLVING THE CRUS CEREBRI (WITH UNUSUAL ENDOCRINE SYMPTOMS)

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Tumors of the crus cerebri usually give a symptomatology dependent upon the possible positions that the tumor may assume in relation to the three important anatomical divisions of the crus—namely the basis, the tegmentum, and the corpora quadrigemina. The tumor, if small, may involve only one of these, giving sharply defined limited symptoms. But usually, besides the direct symptoms produced by the tumor growth, there are those arising from the pressure exerted by the neoplasm upon more remote cell groups and tracts within one or both of the other crural divisions. Such a growth involving the *basis pedunculi* gives a so-called Weber syndrome, oculomotor palsy of one side with crossed paralysis; involving the *tegmentum,* an oculomotor palsy with crossed choreiform or athetoid movements, Benedikt's syndrome; and involving the *corpora quadrigemina,* presents the syndrome of Nothnagel (1) with ocular muscular palsy, cerebellar ataxia and disturbances in hearing.

There are of course various combinations of these to which are superadded the distant symptoms in the case of larger tumors. Cases heretofore reported conform more or less completely with this theoretical scheme. Unexplained symptoms which have thus far been described in the literature are inordinate laughter, which occurred in 2 cases reported respectively by Hunt (2) and Spiller (3), and reduction of body temperature on the paralyzed side, in the cases of Garnier (4), Mendel (5) and Ramey (6). To these I now desire to add a case in which, apart from the fairly classical symptoms of oculomotor involvement with crossed paralysis, ataxia and incoordination of cerebellar type, there were added abnormally *rapid skeletal growth* and *sexual precocity*. Rhein (7) published a series of 18 cases of tumor of the crura which he had thus far found in the literature, and in none of them were such conditions present. While the tumor here presented was not solely confined to the crus

cerebri but extended down to the pons, yet there will be little difficulty in separating the symptoms due to that part below the crus from the gross picture.

In July, 1913, a boy, fourteen years of age, with a negative preceding history, was hit on the head by a playmate. He fell, and though he arose unassisted, was dizzy for a minute or two afterwards. In August, about 5 weeks later, while running, he fell and struck the back of his head without any apparent after-effects. One week later, or about August 25, the father noticed that the boy's speech was affected, his articulation not being clear and perfect as formerly. At the same time his friends began to notice a gradual change in his gait, which had become unsteady. Coincident with these changes, headache began, located chiefly in the occipital region. With the advent of headache, nausea, though no actual vomiting, also began. With these changes, and indeed as early as any of them, the father began to notice priapism in the boy lasting from two to three hours each night. About the 15th of September his sight began to bother him and this got progressively worse. Since the beginning of August his stature increased two to three inches, that is, within five weeks; the rapid growth involved his extremities also, so that shoes which were bought in the early summer no longer fitted him. He became withal more and more drowsy and at times it was difficult to awaken him for examination. He was admitted to the Neurological Institute on September 25, on the service of Dr. Pearce Bailey. While here he was very unruly and resistive, constantly crying to go home, so that it was necessary to discharge him for a time.

His status on entrance was as follows: A staggering, swaying gait towards the left side chiefly, but also occasionally to the right; occipital headache; nausea; no vomiting at first and no tremor. There was a right facial weakness seen chiefly in smiling, *i. e.*, emotional in character. His eyes were examined by Dr. Holden on September 25 with the following result: Diplopia was present, possibly due to the weak left external rectus. Nystagmus, coarse in character, greater when looking to the left, with the slow component to the right, was constant. Vision 20/30 each; hyperopia; with white and red fields normal. Discs were pink, veins slightly dilated.

On October 26 a beginning papilledema with hemorrhage was first noticed in both fundi with normal color fields.

There was incoördination with ataxia of hands and feet; right greater than left. The reflexes gave a greater right knee jerk, a double Babinski and Oppenheim, greater on the right, doubtful on the left at times; abdominals, right sluggish, left absent; epigastrics likewise; cremasterics equal. Elbow jerk, right exaggerated, left doubtful; asynergia was well marked in the usual movements of equilibration. Hearing unaffected. Weber and Rinne tests showed normal conduction. There was irregular pointing by and adiadochokinesis of the right hand. The cerebrospinal fluid was negative. The penis and scrotum were unduly developed. An X-ray

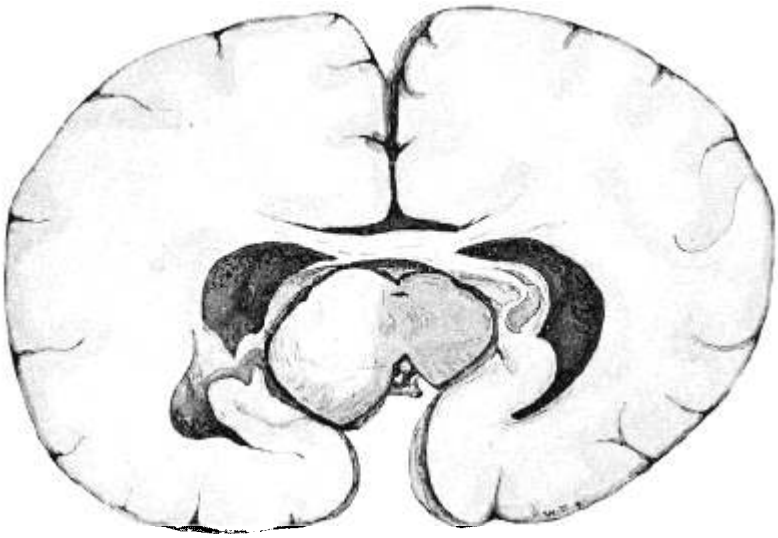
of the skull showed no abnormal sella turcica, nor other pathological condition.

From these findings a general diagnosis of tumor was made without special localization. I personally kept track of the patient while he was at home and noticed gradually an exaggeration of the signs and symptoms. The drowsiness and headaches became more marked. He had two unilateral convulsions involving the right side. Following them there was added an Oppenheim on the left side and a gradual impairment of the motor functions of the trigeminus on the left side. Joint sense was unimpaired. Astereognosis was absolute on the right side, the boy being able to give no information whatever of the object in that hand. A moderate spasticity of the right leg began to appear but no clonus. Finally there was elicited by means of my esthesiometer (8), a slight diminution of cutaneous sensibility of the entire right side. These signs, together with the foregoing status, enabled us to localize the tumor as one involving the crus and pons of the left side, and extending posteriorly to the origin of, but not including, the facial and auditory nerves—at any rate beyond the origin of the motor fifth. The possibility of an enlarged left crus impinging upon the hypophysis or its stalk, was also considered probable.

As the patient became progressively worse with daily attacks of respiratory weakness, verging on the Cheyne-Stokes type, it was imperative that surgical interference be undertaken, albeit there was no increase in the papilledema and scarcely any diminution in vision. He was again brought to the Institute on October 27, but before anything could be done, he died of respiratory paralysis.

In analyzing the symptoms I would like to call attention to several interesting and important points brought out in the examination. First, the astereognosis was probably due to the imperfect sense perceptions from the right periphery and the reciprocal imperfect motor adjustment on the same side, therefore it was no true cortico-psychic astereognosis; secondly, I would like to point out the importance of examining always for the sensory and motor functions separately, of the fifth nerve. In this case this difference possibly marked the limit of the tumor, laterally; the motor root cells lying centrally to the sensory. Thirdly, there is seen the importance of differentiating not only crude changes of sensibility on symmetrical areas of the body, which in this case elicited nothing; but also and especially the finer changes. This gave us one of the requisite signs for localization. Lastly, and most important, I would like to call attention to the symptoms in this case pointing to irritation of either the pineal gland or the hypophysis, those of priapism and of skeletal growth. In none of the 18 cases of tumor of the crus heretofore published, were such symptoms mentioned. As neither of these glands was abnormal, as shown at the necropsy, were they produced

by the pressure within the third ventricle transmitted to the pineal gland or to the hypophyseal stalk, or were they originated by direct pressure of the left crus cerebri (which centrally encroached on the middle line) upon the hypophysis, and superiorly against the pineal? As the ventricles were hardly distended, it is fair to assume that the increased mass of the left crus cerebri was the irritative cause of these symptoms. Furthermore the signs of increased intracranial pressure came on after the growth phenomena had appeared and therefore these could not have depended on this general pressure increase. Another, though very remote possibility, is that the fibers of the commissura habenularum (some of which penetrate and



A transverse section of the brain showing the enlarged left crus cerebri impinging against the stalk of the hypophysis ventrally, and against the pineal gland dorsally. The very slight distension of the ventricles is also to be remarked.

become part of the pineal gland), in their further course from the glandula habenulæ to the glandula interpeduncularis as the tractus habenulæ interpeduncularis, are interfered with in their course through the crus by the tumor, thus affecting the function of the pineal. In such an event, however, all tumors of the crus should show similar symptoms—which they do not. These fibers moreover are presumed to be merely vestigial in character.

These remarks are of course based upon the assumption that interference with either the hypophysis or the pineal gland, or perhaps both, influences the evolution and control of skeletal growth and sexual precocity.

The autopsy by Dr. Casamajor showed a brain very much enlarged, the ventricles only slightly distended, with a pons very much distorted and enlarged, especially on the left side. This enlargement was caused by an extensive pontine tumor mass which reached forward through the left crus cerebri to the left thalamus, and posteriorly nearly to the beginning of the medulla, extending slightly into the brachium pontis of the left side; involving in this extended locus, the left median fillet, the red nucleus with the emerging rubrospinal tract, the left brachium conjunctivum, the left motor fifth root, and compressing the pyramidal tract of the left side as well as by transmitted pressure that of the right side also in lesser degree. The hypophysis was normal in size. The pineal gland was roughly triangular in shape with a large transverse diameter of 12 mm. and its anteroposterior 10 mm. This represents a gland rather large in size although within normal variation. The tumor proved to be a glioma.

The sketch shows a transverse section of the brain giving the relations of the tumor to the pineal gland (which is also reproduced in the picture), and to the hypophyseal stalk.

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