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HEMIHYPERTONIA APOPLECTICA: REPORT OF A CASE. DR. A. M. ORNSTEEN.

There are three reasons for presenting this case: (1) Its resemblance to the hemihypertonia apoplectica described by von Bechterew; (2) the presence of signs of optic thalamus involvement, and (3) the presence of multiple areas of cerebral softening in a young person with resultant pseudobulbar palsy.

The illness began eight and one-half years ago when the patient was 26 years old. At that time she had been married four years, had had two normal deliveries and no miscarriages. After a rather restless night she awoke unable to speak; she appeared to be greatly agitated, and although she understood when spoken to and seemed to make an effort to reply, she would point to her mouth and frantically cry out, "ah-ah," the only sound she could emit. This symptom persists to date. About a month later, she suddenly became hemiplegic on the left side; whether or not consciousness was lost I am unable to determine. The paralysis must have been complete because she was bedridden for about two years, becoming greatly emaciated and developing large bedsores on the left buttock and other pressure points on the left side. She was unable to walk unaided in a satisfactory manner at the end of three years. There was left hemiplegia with motor aphasia in a right-handed person.

The condition remained unchanged for the next three or four years, when her facial expression became somewhat fixed with the mouth wide open and a silly smile; there was much drooling, and she began to shriek spasmodically with sudden outbursts of laughter; also ability to get around diminished until she again became bedridden a year ago. She now has urinary and fecal incontinence.

Examination: She shrieks and laughs spasmodically; any form of stimulus, such as asking her to close her eyes, will result in one of these outbursts. She is mute but seems to understand everything said to her, responding correctly with gestures of her head and right arm, although her reactions are greatly exaggerated. She has never been known to cry involuntarily. Memory and orientation are apparently well preserved. Attention and perception are good. Further investigation of her mental processes is impossible, with the exception of writing. It is distinctly agraphic, although occasionally a word can be recognized. She attempts to write from dictation but only her name is partly discernible.

The pupils are irregular, unequal and react poorly to both light and convergence, which may be partly due to the existing primary optic atrophy. Vision cannot be tested, but there is no evidence of gross defect in the fields. Ocular movements are well performed without nystagmus. Volitional power in the frontalis and orbicular palpebral muscles is good. In the orbicularis

oris volitional power is poor, the woman being unable to bring her lips together, or to retract the corners of the mouth, but in a spasmodic outburst the orbicularis oris is actively contracted. The tongue lies motionless in the floor of the mouth, and the patient is unable to move it in any direction. Eating and drinking are interfered with, the soft palate is slightly raised on volition, and she is unable forcibly to bring the jaws together.

The left arm is held extended, and to passive motion the resistance is great, but she is able to raise the arm quickly above the head without much sign of rigidity. The same difference is seen in the left leg, i. e., a much greater tonicity to passive than to voluntary motion. The fingers of the left hand are strongly flexed over the thumb by a spasm, not a contracture. This spasm may be seen to disappear allowing the fingers slowly to extend and occasionally to overextend through spasm of the extensors. The same alternation of spasm of the pronators and supinators of the arm may be seen. It is a slow mobile spasm. The toes, although held mostly in a flexor spasm, occasionally become involved in this mobile spasm. She is able to raise the left leg quickly from the bed; her left hand-grasp, although weaker than the right, is fair. In other words, she has not a hemiplegia, but a hemihypertonia with spasm. The reflexes on this side are exaggerated and a Babinski reflex is obtained. During active movements on the right side, the left arm shows some associated movements.

Voluntary movements and power in the right extremities are good; the movements, as stated in the foregoing, are very active and quick in response to a stimulus. The reflexes are exaggerated and a typical Babinski reflex is also present on this side.

The perception of touch and pain is preserved on the left side, but it is not as keen as on the right. Point discrimination and localization could not be tested for obvious reasons. Thermal sense appears to be normal. If a key be placed in her right hand, with eyes blindfolded, when asked if she recognizes it, she will nod her head in the affirmative and take the key between her fingers and turn her hand as if opening a lock. With the left hand she is unable to recognize the key. A left hemi-ataxia exists. Pressure of the musculature on the left side is more painful than on the right; whether or not she experiences the dysesthetic phenomena of thalamic disease it is impossible to determine. For many months she has complained of pain in the left side.

The underlying pathologic factors are probably bilateral interruptions in the cortical projection systems. A lesion in the right capsule extending into the thalamus and lenticular nucleus may be surmised in explanation of the left capsulothalamostriatal syndrome, namely, initial hemiplegia with regression, hemihypertonia with spasm and hemianesthesia for deep sensibility, associated with a disturbance of emotional tone. A bilateral involvement of the corticopontile fibers for the pseudobulbar symptoms, interruption of the frontothalamic and the thalamofrontal fibers (the anterior thalamic peduncle), loss of cortical inhibition of the primordial emotional reflexes with elevation of affective tone and instability of the emotions were present. The initial loss of speech, before the involvement of the muscles of articulation, must be looked on as the result of a lesion subcortical to the left operculum.

The lesions are probably thrombotic softenings of syphilitic origin. The blood Wassermann test is negative; the spinal fluid was not examined because of unsuccessful punctures. The cardiovascular and renal systems are normal.

Roettiger, in 1921, gave the following points, among others, as characteristic of hemihypertonia apoplectica: Cases follow apoplectic hemiplegia with regression of paralysis; no secondary contractures occur as in capsular lesions; passive movement increases the tonus, voluntary movement diminishes it; movements of the unaffected side are more energetic than usual, sometimes assuming the character of associated movements; frequently forced laughter occurs; occasionally tremor and athetosis and dysarthia are seen.

DISCUSSION

DR. C. K. MILLS: To my mind, the idea of tonicity being due to a withdrawal of inhibition is not correct. Tonicity and other tonectic phenomena are results of actual stimuli discharged from cortical or subcortical regions, these impulses being often markedly interfered with in their transmission, as when the pyramidal system is diseased. Cases such as reported by Dr. Ornsteen may be due to lenticulocapsular lesions.

DR. M. K. MEYERS: This patient was in my service at the Jewish Hospital. I regarded her as having a case of pseudobulbar palsy. I think that she was less emotional at that time. Never, to my knowledge, did she act in the exaggerated manner that she does tonight. She has deteriorated markedly since I saw her last, over a year ago.

A CASE OF TUMOR OF THE OCCIPITAL LOBE. DRS. L. J. HAMMOND and J. HENDRIE LLOYD.

Ferrier believed that the region of the angular gyrus was especially the cortical center for vision, but he claimed that a lesion here did not cause hemianopsia but complete amblyopia of the opposite eye, and possibly partial amblyopia of the eye of the side of the lesion. In order to obtain hemianopsia he thought that the occipital lobe must be impaired along with the angular gyrus. These experiments on monkeys, however, are open to the grave criticism that it is quite impossible to measure the visual fields in a monkey.

Attempts have been made to distinguish the macular fields, or fields for central vision, from the panoramic fields, or fields for wider peripheral vision. It has been asserted, for instance, that in lateral homonymous hemianopsia the blind half sometimes shows a small central area, or half-area in which vision is retained; this is the so-called macular area; and in double hemianopsia this small central area may, it is said, still be active. In other words, the patient is not completely blind. He goes peering about as though he were looking through a knot hole. Those who still follow Ferrier believe that the center for macular vision is in the angular gyrus, but others locate it in the cuneus, on the mesial aspect of the occipital lobe, and believe that the fields for panoramic vision surround it. The latter view is probably correct, for thus the macula, or center of the retina, is represented in the center of the visual cortex. Bramwell, some years ago, recorded a remarkable case of bilateral cortical blindness, of twenty-five years' duration, with preservation of a small macular field in each eye, in which necropsy revealed a small normal area in each cuneus, surrounded by an area of degeneration in each occipital lobe. This would seem to prove that the macula is represented in the cuneus, and that the center for panoramic vision surrounds the center for the macula. In the human subject we are dependent on pathologic lesions, and such lesions are often too gross, or not well enough demarcated, to furnish an exact localization. Such as they are, however, we must avail ourselves of them; therefore, we ought to record those cases as they arise.

In the present case there was right lateral homonymous hemianopsia, which was practically the only localizing symptom caused by a tumor which compressed the left occipital lobe. The patient was a single woman, aged 53, admitted to the service of Dr. Hammond, in February, 1922. About ten months previously she began to have pain in the occipital region. The pain was severe but intermittent, with vomiting, lasting for from two to four days. There were intermissions of two or three weeks. Vision gradually failed, until on admission she could not see to read but she could distinguish faces across the room. There was no paralysis of any limb or cranial nerve, no convulsions and no anesthesia. There was some vertigo, but no forced movements. The head was carried tilted to the right. The mind was rather sluggish, general health good.

Examination revealed right lateral homonymous hemianopsia. There were no visual hallucinations. The gait was unsteady; some dysmetria of the hands was noted. There was no anesthesia, but some astereognosis of the right hand. The pupils were equal, slightly dilated and responsive to light. The Wernicke pupillary sign was lacking; that is, the pupils reacted to light thrown on the blind halves. There was no aphasia. Dr. Moore found choked disks of a high grade, small central vessels, edematous retinas, but no hemorrhages.

The roentgen-ray report was negative, as were the laboratory tests of the blood and spinal fluid.

The Bárány tests showed a lesion in the right hemisphere of the cerebellum, at or about the semilunar lobe. This localization did not agree with the one indicated by the other findings, but unfortunately it was allowed to determine the site of the operation. Dr. Hammond performed an operation over the cerebellum, but found nothing. The patient died about four weeks later.

The tumor was comparatively large, and sprang from the membranes of the left occipital lobe. It lay partly at the extreme posterior end of the lobe in such a way as to cause pressure on the mesial aspect of the lobe and on the cuneus or calcarine region. Hence it had rather unusual value as a localizing lesion. The injury done was almost entirely to the occipital lobe, although the size of the growth was such that it may have made some pressure forward on the superior parietal lobule. The temporal lobe did not seem to be involved.

The main features in this case were: severe headaches, paroxysmal in kind, with nausea and vomiting; papilledema, advancing rapidly to blindness; right hemianopsia; slight astereognosis of the right hand; unsteady gait, not forced or suggestive of cerebellar lesion.

The symptomatology is significant for what it includes and for what it lacks. There was no paralysis, no anesthesia, except the slight astereognosis; no convulsions; no affection of gait or forced movements, except an unsteady gait, partly due no doubt to increasing blindness; no aphasia. Of the positive symptoms the hemianopsia was by far the most significant, the only one that had any localizing value, except the astereognosis, which tended rather to indicate a possible parieto-occipital lesion and hence was a little misleading.

Affections of the form fields were seen, of course, in other than strictly localized occipital lesions. In another case a large tumor in the parieto-occipital region caused hemianopsia to the opposite side, but there were other symptoms that served to distinguish it. Cushing has recently called attention to tumors of the temporal lobe as causes of various types of anopsia. It is easy, of course, to understand that a lesion anywhere in the course of the optic radiations will cause such symptoms; but the point to be emphasized in

the present case is the fact that the lateral homonymous hemianopsia was the only reliable symptom, and that if we had relied on it we should have been led to the seat of the tumor.

We feel obliged to point out that in this case the Bárány tests were misleading. These tests are only tests of the vestibular nerve; the possibility of a misinterpretation of them is incalculable; and the doubt raised by them may be little less than demoralizing to the diagnostician. Why do we attach so great importance to this one nerve? We do not attach localizing value to a mere choked disk. Disturbance of the vestibular nerve, taken by itself, may be no more of a sure guide than a choked disk.

DISCUSSION

DR. C. K. MILLS: The one doubt that might have arisen in Dr. Lloyd's case was whether the lesion was on the surface or in the interior of the occipital lobe. The astereognosis is easily explained by the pressure or direct involvement of a part of the parietal lobe.

To a certain extent I am a believer in Bárány tests, as clear results can be obtained by this method in cerebellopontile angle lesions. Even an alleged Bárány determination of a cerebellar lobe lesion can be carefully scrutinized. The examination of panoramic and macular vision is important, as the former can be present without the latter or the latter without the former.

DR. H. MAXWELL LANGDON: I feel that the Bárány tests are a valuable addition to our methods of brain localization; they are additional evidence, and should be weighed in the same scales with the other evidence. I know of some cases in which the Bárány tests have been almost astounding in the directness of their evidence, and other instances in which their findings did not fit in with other examinations at all. If we could distinguish between direct and transmitted pressure signs, many of our cases could be localized, whereas now we miss them.

When there is exact lateral homonymous hemianopsia, I believe that in ninety-nine cases out of a hundred there is direct and not transferred pressure.

DR. J. HENDRIE LLOYD (closing): I agree that the Bárány tests are of value sometimes in localizing peripheral nerve lesions and pontile angle lesions. In syphilis of the eighth nerve they may show that the functions of the vestibular nerve are abolished, and thus confirm the diagnosis of a lesion of the eighth nerve. But when these examiners get inside the brain stem they are lost. In one case of syphilis of the eighth nerve, the man who made the Bárány tests tried to draw conclusions as to localizations inside the pons. I did not agree with him. Inside the brain stem the course of the vestibular and acoustic nerves cannot be followed with scientific accuracy. When the attempt is made to diagnose a lesion in the right cerebellar hemisphere on data given by the Bárány tests, it is making deductions from insufficient premises.

Dr. Fay, I think, referred to hemianopsia in pituitary tumors. I think they are usually cases of bitemporal hemianopsia due to pressure on the chiasm. That is what is generally taught. I have not seen many such cases.

The question was raised as to the possibility of a lesion of the cerebellar hemisphere causing pressure upward through the tentorium and thus causing hemianopsia. I feel that the case is added proof that the visual cortex is on the mesial aspect of the occipital lobe. I do not believe that macular and panoramic vision are located in different regions of the cortex but that macular vision is localized in the cuneus and panoramic vision surrounds it.

MARKED ATROPHY IN EARLY TABES. Dr. Herbert Fossey.

The patient, a white man, 30 years old, was admitted to the Philadelphia Hospital Aug. 23, 1921, complaining of urinary incontinence and gastric pains. He presented marked emaciation and appeared to be very sick. Examination revealed severe cystitis, pyelitis and an infected penis due to continuous friction of the urinal in addition to the typical signs of tabes dorsalis. He was suffering intensely from gastric crises which had been present for the last six weeks. Two weeks prior to entrance he had been operated on for appendicitis.

The most striking finding was the extreme atrophy in the disease which was of only four years' duration. That his symptoms started one year after the initial lesion is noteworthy.

Déjerine mentions the tabetic type of neuritic atrophy which may be confused with tabes. Lapinsky speaks of an initial neuritis in young people which gives marked early muscular atrophy. According to Marie, tabetic muscular atrophies may be divided into two groups: (1) those appearing late, presenting a symmetrical distribution, rarely marked by fibrillary twitchings; (2) those occurring often in the earlier stages of the disease, usually unilateral in distribution, marked by fibrillary contractions and sometimes by the reaction of degeneration. The first group embraces atrophies confined to the distal portions of the extremities, and recalls the conditions found in multiple neuritis. The second group contains lingual hemiatrophy, localized atrophies of the shoulder, of the back, of the hand, and one-sided involvement of the cranial nerves. They are analogous to lesions of the gray matter. Both the central and peripheral lesions are found, and in the associations indicated in the foregoing. The wasted muscles present the usual histologic change due to degeneration in the lower motor neuron.

Herman Lippman ("Concerning Muscular Atrophy in Tabes Dorsalis") states that "Lapinsky and others have found that section of the posterior root causes changes in the cells of Clarke's column and in the nerve cells of the anterior horn; changes which in their essentials consist of a swelling of the cells and loss of Nissl bodies. From this standpoint he explains the atrophy in tabes. Déjerine thinks that the muscular atrophy of tabes depends on a peripheral neuritis, proceeding slowly to the anterior roots. The muscles which these nerves supply have thin round fibers which in portions fall apart and are filled with pigment. The connective tissues in these muscles are increased and rich in nuclei, the fibers thin and few. Clinically there is fairly symmetrical atrophy of the extremities. Fibrillary twitchings are not observed; the reaction of degeneration is frequently seen. Déjerine is of the opinion, because of these findings, that the spirochete or its toxin at times locates in the peripheral motor nerves. The clinical and pathologic findings are the same whether the neuritis is due to poisoning or to a mechanical injury. It has been known for a long time that in tabes, as in alcoholism, the motor nerves are vulnerable. A toxin which a normal person can withstand may injure the nerves of an alcoholic or a tabetic person. Leyden, Remark, Möbius and others believe that in tabes there is a tendency to paralysis because, owing to the degeneration of the posterior roots, impulses do not reach the anterior horn cells. The resistance of these cells is decreased, and they are easily injured.

An objection to the theory of Déjerine is that in his studies he used the older methods of staining, and the author believes that the newer methods, especially the special cell stains, are necessary to determine the entire picture, especially as regards the anterior horn cells.

The author concludes that muscle atrophy in tabes occurs under these conditions: (1) due to accompanying disease; (2) as a result of the general weakness and anesthesia of the muscles toward the end of tabes; (3) as a result of the peripheral neuritis brought out, not through the poison of tabes, but through other injurious processes; (4) as a result of the localization of the toxin of syphilis in the nerve roots and anterior horn cells, whereby the motor impulses are more or less impaired.

The spinal cord of the patient under discussion revealed the following: The usual degeneration of the posterior roots seen in tabes is very intense in the lumbar region and even in the midcervical region. The nerve cells of the anterior horns of the lumbar region show considerable degeneration of the type of central chromatolysis; that is, a disintegration of the chromatophilic elements, particularly of the center of the cell body with peripheral displacement of the nucleus. This is a common finding in multiple neuritis and would indicate that the peripheral nerves were considerably degenerated. The nerve cells of the anterior horns of the midcervical region show a similar reaction at a distance but not so intense. The nerve cells are possibly not so numerous as one would expect. Intense degeneration of the posterior root from the upper limbs with some central chromatolysis of the nerve cells of the anterior horns indicate that a similar degeneration of the peripheral nerves was present in the upper limbs. Such widespread degeneration of the posterior roots is uncommon in tabes of such short duration and the process had reached a height which is usually seen in tabes only after much longer duration. The action of the spirochete in this case must have been exceptionally virulent, producing within a short time lesions uncommon at so early a period.

DISCUSSION

DR. C. M. BYRNES: I should like to ask Dr. Fossey whether there were any objective sensory changes in his case, and whether the patient had been treated with mercury or arsphenamin before the development of the atrophy. Through the courtesy of Dr. Spiller, I have been studying round cell infiltration in various nervous lesions and I was much interested to find that in syphilitic cords there is not infrequently, contrary to the usual teaching, marked cellular exudate about the anterior roots. In several instances, the anterior roots on both sides were practically embedded in a plastic exudate, sufficient, it seemed, to account for the occasional atrophies occurring in tabes.

DR. HERBERT FOSSEY: The patient had hyperesthesia generally distributed over the extremities. He had received no antisyphilitic treatment prior to his admission.

A CASE OF PERMANENT QUADRANT ANOPSIA, POSSIBLY DUE TO MIGRAINE. DR. J. C. MULRENAN.

John F., aged 28 years, married, complained of defective vision. In 1918 he had had influenza and pneumonia. For several years he had suffered from periodical headaches; otherwise he has been well. His habits have always been good. The headaches occur about once in two weeks and are usually located in the right frontolateral region. During the attack he is pale, but as a rule does not vomit. There have never been hallucinations of vision, taste or smell. One evening in April, 1920, he had a severe headache. The next morning while quietly standing after some heavy lifting, he was suddenly seized with a "feel-

ing of numbness and pins and needles in the left hand, arm and side of the face. Everything became black, but he was not unconscious and did not fall." Immediately afterward there was a violent headache, and he vomited. For about five minutes he was completely blind. After the vomiting vision returned but was blurred, and it has remained so since.

Dr. L. C. Peter reported a refractive error (hyperopia) and left superior quadrant anopsia and a large scotoma for colors in the lower nasal area of the right field. Color fields were concentrically contracted following the form fields. An enlarged blind spot was present in the right eye, otherwise the eyes were normal. Neurologic examination was negative, as was also the medical report made by Dr. G. M. Piersol. Examination of the nasopharynx by Dr. Butler was also negative, with the exception of chronic pharyngitis. Syphilis was denied, and the Wassermann test was negative.

His mother and grandmother had suffered from similar headaches.

DISCUSSION

DR. CHARLES S. POTTS: This patient was in my service at the Polyclinic Hospital. While I at first thought that the attack was probably embolic, and a case somewhat like those described by Dr. Woods in the February number of the *Journal of Nervous and Mental Diseases,* the history of headaches, the fact that the mother and grandmother were subject to headaches of a migrainous type, the negative Wassermann test and the absence of cause for an apoplectic disturbance, made me think that this case bore a similarity to the cases described by Hunt and others occurring in patients subject to migraine.

DR. C. K. MILLS: I think it is a mistake to give out the idea that migraine can in some inscrutable way cause a more or less permanent anopsia or hemianopsia, unless a destructive focal lesion is present.

DR. C. S. POTTS: With reference to what Dr. Mills has said I would state that my belief is that arterial spasm caused the condition. Whether the spasm lasted long enough to cause degeneration, or whether thrombosis occurred in the artery, I cannot say. During an attack of migraine I believe there is arterial spasm, and it therefore might produce the contraction of the arteries that caused the lesion producing this condition. There is no reason to believe that the patient had syphilis; and he was carefully studied from all standpoints and no cause found for an embolism.

DR. ALFRED GORDON: We are accustomed to believe that migraine is a temporary but periodic condition, which comes and goes; but this patient had distinct subjective sensory phenomena, limited to the left arm. He had tingling in that arm and right hemianopsia. It is difficult for me to understand that migraine could cause a lesion of such character. I would attempt to localize the lesion in the posterior portion of the internal capsule and in the optic radiations. This would explain the hemianopsia and the sensory phenomena. I believe that the lesion is vascular. An alternating spasmodic contraction of the blood vessels is a plausible assumption.

DR. H. MAXWELL LANGDON: There is a type of case which ophthalmologists see rather infrequently which may throw some light on what happens to the cerebral circulation in these cases of migraine; namely, spasm of the retinal arteries. Some years ago I reported such a case before the American Ophthalmological Society. It was that of a man who for many years had had attacks of transient monocular blindness, the attacks never being simultaneously bilateral—each eye was affected at different times; the attacks would last from three to five

minutes, during which time vision was completely gone, and then it would return to normal. Unfortunately there was no opportunity to examine the eyes during an attack. One eye eventually suffered from an attack which was permanent, there being a complete collapse of the retinal circulation. The man had advanced arteriosclerosis, and one of the interesting questions to be solved is whether spasm of perfectly normal vessels occurs. Should a spasm occur in normal vessels I do not believe it is as likely to be permanent in its results as when the vessels are sclerosed and are likely to become permanently blocked. Similar cases have been reported when examination was made during an attack, one case in this city being seen by several observers, among them Drs. Zentmayer and de Schweinitz. During the attack the retinal circulation would be completely lost, and then the vessels would gradually fill again until the condition was restored to normal. I think what happened in these cases is what happened in Dr. Mulrenan's case, only that in his case it happened in the cerebral circulation instead of in the ophthalmic.

DR. L. C. PETER: Four or five years ago I saw a case similar to this one. It occurred in a young lawyer who had been over-worked, and who, in the midst of a conversation with his partner, suddenly became aphasic. He did not fall, but was hardly able to stand up. He had left-sided motor and sensory disturbances, which cleared up in a few days. On the day after the attack, he had a left inferior quadrant anopsia which changed slightly in the course of five or six months and finally left quite a defect in this particular area. He did not have a lesion in the cuneiform body, but a lesion somewhere around the capsule, possibly in the beginning of the optic radiation. The condition cleared up entirely, the aphasic phenomena lasting two or three days.

These fields bring to mind a thought of Dr. Cushing, when he referred to the asymmetry in homonymous hemianopsia, the greatest advance, as a rule, being homolateral to the lesion. It has been difficult for me to reconcile the cause usually assigned to this asymmetry with the facts in hand. Associated nerve fibers representing corresponding retinal points may or may not be in close contact after they leave the chiasm, during their course to the occipital lobe. Defective technic may lead to error and apparent asymmetry. The main factor, however, in asymmetry of homonymous hemianopsia is the relative difference of retinal sensitivity in the nasal and temporal retinae.

In the case of Dr. Mulrenan it is not likely that the lesion was in the cuneiform body, because of the motor and sensory symptoms, but far forward, probably in the beginning of the optic radiation of Gratiolet.

TUBEROUS SCLEROSIS. DR. W. FREEMAN.

This paper will be published in full in a future issue.

DISCUSSION

DR. N. S. YAWGER: I have had three epileptic patients in whom I have made the diagnosis of tuberous sclerosis. Dr. Freeman has referred to two of these.

TWO CASES OF IDENTICAL ACHONDROPLASIA WITH EPICANTHUS IN BROTHERS. DR. A. E. TAFT.

Two boys, aged 16 and 9 years, respectively, showed identical anomalies, which were congenital and not progressive. The personal and family histories were negative; the Wassermann test was negative.

Both cases presented the following features: A lock of white hair at the midline of the frontal hair margin; bilateral epincanthus; narrow, high-arched palate with overlapping teeth; complete deafness; "high shoulders"—scapulae in embryonal position opposite lower cervical vertebrae; maldevelopment of elbow joints preventing normal flexion of forearms; radius relatively longer than ulna and maldevelopment of carpals, thus forcing hands into ulnar position; marked lack of muscular development of upper extremities reducing function to a minimum, though other skeletal muscles were well developed Intelligence was normal.