

On hypertrophy of the brain in imbeciles / by Fletcher Beach.

Contributors

Beach, Fletcher, 1845-1929.
Royal College of Surgeons of England

Publication/Creation

Lewes : H.W. Wolff, steam print. works, [1881]

Persistent URL

<https://wellcomecollection.org/works/u9tfwz7e>

Provider

Royal College of Surgeons

License and attribution

This material has been provided by This material has been provided by The Royal College of Surgeons of England. The original may be consulted at The Royal College of Surgeons of England. where the originals may be consulted. This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.



Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
<https://wellcomecollection.org>

296
64

6

HYPERTROPHY OF THE BRAIN IN IMBECILES.

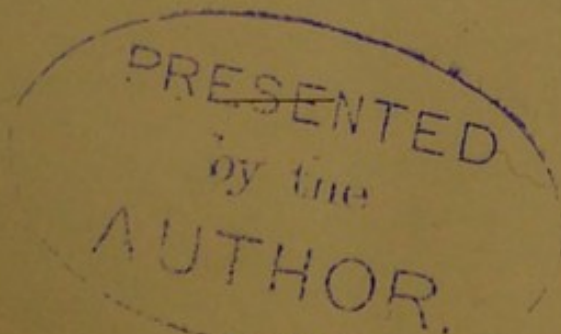
BY

FLETCHER BEACH, M.B., M.R.C.P.,

Medical Superintendent of the Dagenham Asylum.

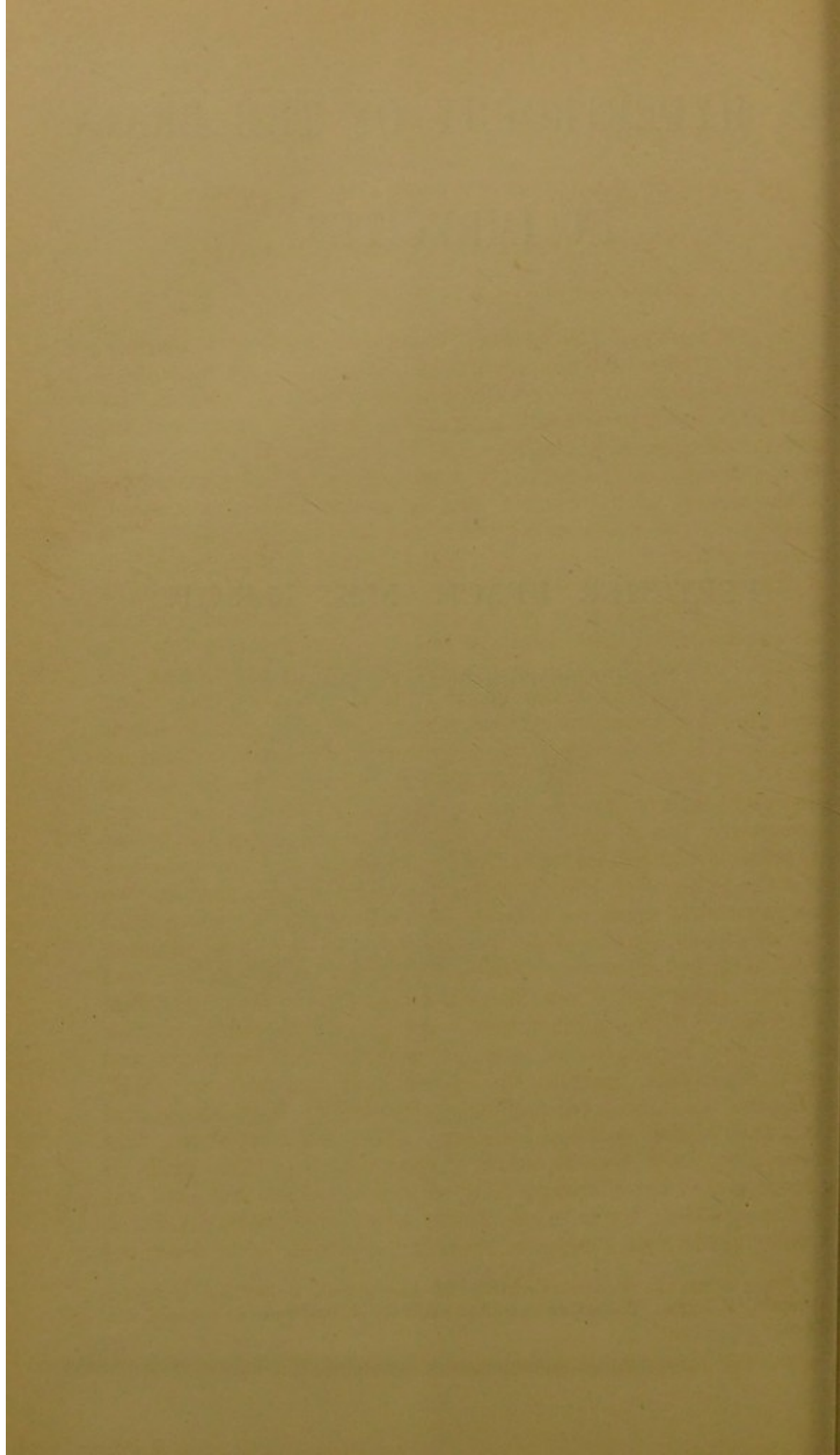


[Reprinted from "The Journal of Mental Science," April, 1881.]



Lewes:

H. W. WOLFF, STEAM PRINTING WORKS.



ON HYPERTROPHY OF THE BRAIN IN IMBECILES.

By FLETCHER BEACH, M.B., M.R.C.P.,

Medical Superintendent of the Darenth Asylum.*

Hypertrophy of the Brain is a comparatively rare disease, and has attracted little notice in England. Laennec first drew attention to its occurrence in children, and to its similarity to chronic hydrocephalus in many of its symptoms, and the likelihood of its being mistaken for it. The cause of the disease is obscure. Brunet defines it "as an increase in the weight of the organ due to a disorder of nutrition, leading to an alteration in the nervous substance." The process is not one of mere increased growth, but the nutrition of the organ is modified in character as well as increased in activity. According to Rokitansky the augmented bulk is not produced by the development of new fibrils, or by the enlargement of those already existing, but by an increase in the intermediate granular matter, most probably due to an albuminoid infiltration of that structure. My own observations lead me also to the opinion that the disease, as seen in imbeciles, is due to an increase in what appears to be granular matter, for in some sections, which I have in my possession, this is seen. The change is accompanied by an increase in the number of the blood vessels, and the presence of a large number of leucocytes. Andral states that in two post-mortems made by himself, the white matter resembled the white of egg hardened by boiling. In one post-mortem only have I noticed any peculiarity in the white matter, and in that case I find I have recorded that it was of a "peculiar whiteness." The parts affected are chiefly the white matter of the two hemispheres, sometimes the corpus striatum and optic thalamus, rarely the pons and cerebellum. MM. d'Espine and Picot consider the affection to be a congenital one, and in this opinion I concur. They hold, however, that imbecility only results when hypertrophy of the brain is accompanied by sclerosis. My experience does not lead me to this opinion, for in none of the seven post-mortems which I have made was sclerosis present. Brunet, too, describes

* Read before the Medico-Psychological Association at Bethlem Hospital, December 1, 1880. The paper was illustrated by photographs and cranial outlines.

two cases of hypertrophy of the brain without sclerosis which he has seen in idiots. Both d'Espine and Picot and Brunet describe two forms of the disease; in the one the hypertrophy is simple, in the other accompanied by sclerosis. The principal symptoms are headache, at times intensified, excitement followed by coma, blunting or arrest of development of the intelligence, difficulty in walking, and convulsions. The symptoms are said to be less marked in children than adults, because the brain is less compressed, the cranial cavity increasing in size as the brain enlarges. I proceed to relate particulars of some cases which have come under my observation.

CASE I. A. H., aged 15 years, was admitted into Clapton Asylum June 19th, 1875, and died April 20th, 1876. There was no history of neuroses or of hereditary disease in the family. His parents were of temperate habits, sound, physically and mentally, and were not connected by consanguinity. His mental deficiency is said to have been noticed after a series of convulsions with which he was seized while teething, at the age of nine months. They soon ceased, and he had none afterwards. There were two other children, one older and one younger than this one, whose mental condition was unaffected. A. H. on admission was a well-nourished boy, of dark complexion, with a large head, square in shape, and with well-marked frontal prominences. Circumference 23 inches, transverse diameter $12\frac{1}{2}$ inches, antero-posterior 13 inches. He was bright-looking, good-tempered, and willing to work. There was no loss of sensation or motion. No sign of rickets. Mental capacity fair for an imbecile. He could use many words, but his articulation was defective. He went to school in the Asylum, worked as a tailor, and assisted in household work. During the ten months that he was under training he made good progress in school and shop. He was an imbecile who, had he lived, would have made good progress. His general health was good, and until the illness, from which he died, he was only under treatment for minor ailments. About a week before his death he took to his bed, complaining of pain in his right shoulder and arm. Two days afterwards he was seized with a convulsive tremor, his limbs at the same time being rigid. From this he recovered, but four days afterwards I was called to him, and found him breathing stertorously with his tongue half protruded. His breathing, while I was with him, became laboured, and in about five minutes he died.

At the post-mortem, 32 hours after death, on removing the calvaria the cranium was found to be increased in size anteriorly and posteriorly, and there were well-marked frontal prominences. The dura mater was congested, and very adherent to the skull cap over the greater part of its surface. The sinuses were very full of blood. The brain

weighed 62 ounces. On removing the dura mater the subarachnoid fluid was seen to be increased in amount over the whole of the convex surface, and over a space $1\frac{1}{2}$ in. in length and nearly $\frac{1}{2}$ in. in breadth, close to the right of the longitudinal fissure, and corresponding in position with the posterior part of the frontal convolutions was a layer of pus. The membranes did not appear to be thickened. The vessels were all much congested, and the pia mater injected. The convolutions were simple. They bulged upwards, and the sulci correspondingly were not as well marked as usual. On slicing through the brain transversely the white matter was seen to be exceedingly increased in amount. The puncta vasculosa were well-marked. The lateral ventricles were dilated and contained a quantity, though not large in amount, of fluid. Ependyma thickened and presenting the so-called "ice plant" granulations. Brain substance firm. No sign of tubercle, tumour or sclerosis. The white matter on being subjected to microscopical examination was found to present a uniform granular appearance. There were a large number of leucocytes present, and a great increase in the number of vessels; from these the leucocytes had evidently escaped.

CASE II. A. C., aged 12 years, was admitted into the Clapton Asylum May 11th, 1876. His mother was hysterical, and had an epileptic fit when pregnant with her eldest child. The maternal grandmother died of apoplexy. On the father's side there was a history of phthisis. There was no history of consanguinity, and the parents were temperate in their habits. The mother had a fright when pregnant with A. C. and became unconscious. When two years old, while teething, he had a fit, and he has had them ever since. He was always dull and sleepy, and used to "bob" his head forward. His head was large when born, but the projections on the forehead have since come on. There are six other children who are quite healthy; three have died, but not of nervous diseases.

A. C. is a fairly grown boy, with a fresh colour, but vacant look. He is a low type imbecile. His head is large, square in shape, and there are well-marked frontal prominences. Circumference 22 inches, transverse diameter $12\frac{1}{2}$ inches (with calipers $4\frac{3}{4}$ inches), antero-posterior diameter $14\frac{1}{2}$ inches (with calipers $7\frac{1}{2}$ inches). Width of forehead between external angular processes of frontal bone 4 inches. He complains at times of headache, and points to the right temporo-parietal region when asked where the pain is situated. There is a very slight depression, the size of a sixpence, in the region of the anterior fontanelle. His head is prominent posteriorly. He walks slowly, and with a tottering movement, hanging his head slightly forwards, and with his left shoulder depressed. He cannot stand long at a time. Soon he begins to lean backwards and would fall if not

supported. He has little grasping power in his hands, which are cold and blue. Pulse 88, regular and weak. No sign of rickets. Sensation very deficient. He answers questions slowly, and there is a long pause between question and answer. He suffers much from epileptic fits, and, altogether, is gradually deteriorating. Lately he has been getting much weaker on his legs and falls about more. About every two months he loses his speech, and becomes very weak and helpless. This lasts for about 10 days, and then he slowly recovers.

CASE III. W. J. E., aged 14 years, was admitted September 4th, 1876, and died April 13th, 1877. There was a history of phthisis on the father and mother's side of the family, and of epilepsy, apoplexy, and insanity on the mother's side. The father and mother were first cousins. The case is a congenital one. The mother suffered very much from pain in the head when pregnant with the patient, and used to bang her head against the wall. The child had a fit after a fall on the head when he was twelve months old. He is said to have had "inflammation of the brain," and afterwards was convulsed at times. One brother of the patient died of bronchitis and convulsions, and another had a series of fits from which he recovered. The boy was fairly nourished, and presented no signs of rickets. His head was large, with well-marked frontal prominences—circumference $21\frac{1}{2}$ inches, transverse diameter 13 inches, antero-posterior 14 inches. He was dejected in appearance, subject to fits, and of low mental capacity. He made no progress in school. His general health was good until a week before his death, when he was taken ill with vomiting and diarrhæa, convulsions came on and he died exhausted. At the inspection the brain was found to weigh 52 ounces. It was congested. The anterior portions of the cerebrum quite filled up the cranium, which in the right frontal region was thinned and diaphanous. The ventricles contained some fluid, but a quantity had drained away when the brain was removed from the skull. There did not appear to be an increase of the white relatively to the grey matter, except in the central part, but it seemed to be a general hypertrophy of the whole brain.

CASE IV. A. H. S., aged 5 years, was admitted into the Clapton Asylum June 23rd, 1875. Parental history good. The mother ascribed his condition to a fright when pregnant with him. He had a large head when born, and never noticed things like other children. He was a fairly nourished boy, not epileptic, unable to speak, and of small mental capacity. No sign of rickets. He went to school in the Asylum, but made little progress. He suffered from diarrhæa a short time before his death, which occurred suddenly, preceded by unconsciousness, on September 22nd, 1875. At the autopsy the brain was seen to be congested, and the pia mater injected. The subarachnoid fluid was increased and slightly turbid. On slicing through the brain the white matter was found to be considerably increased in amount. The lateral ventricles were slightly dilated, and contained

fluid. A large quantity escaped on removing the brain, which weighed $49\frac{1}{2}$ ounces.

CASE V. A. G. D., aged 8 years, was admitted July 14th, 1876, and died December 20th, 1876. The father was intemperate, and had separated from the mother. No history of nervous disease or of insanity in the family. The case was congenital, and said to be due to the mother flying into violent passions with the father during her pregnancy. The child had a "peculiar shaped" head from birth. He was a well-nourished child, with a large head, and had fair use of his limbs. No sign of rickets. He could say a few words, but usually motioned for what he wanted. Disposition passive. Mental capacity small. Not epileptic. He made little progress in school. About a week before his death he vomited, his face became flushed, and unconsciousness supervened. He remained in this state till death took place. When the post-mortem was made the brain was found to weigh 53 ounces. Convolutions simple. Large vessels congested, and pia mater injected. No disease of membranes. On slicing through the brain the white matter was found to be increased in amount, both relatively to the grey matter and absolutely. It was of a peculiar whiteness. Brain substance firm.

CASE VI. P. J., aged 10 years, admitted July 5th, 1875. No history obtainable. The patient was a well-nourished boy, tractable and well-behaved, not epileptic, able to speak, and of fair intelligence for an imbecile. His head was large, but no sign of rickets in the body could be detected. He made some progress in school, and was put to work as a shoemaker. His health was generally good, but bronchitis came on, of which he died, February 10th, 1876. On examining the brain the convolutions were found to be very simple, and the white matter in excess of the normal amount. No sign of congestion, and no excess of fluid. Weight of brain 55 ounces.

CASE VII. F. D., aged 11 years, admitted May 14th, 1875. The only information obtainable about him was from the certificate which accompanied his admission, and which stated that he was an "idiot from birth." He was a well-nourished boy, with coarse features, and repulsive appearance. Head large. No sign of rickets. He was a low type imbecile and had little intelligence. He went to school in the Asylum, but made absolutely no progress. About three days before he died diarrhœa came on, which weakened him considerably, and caused collapse and death on February 11th, 1876. His brain weighed 49 ounces. The calvaria was thicker than normal, especially anteriorly and posteriorly. Dura mater congested. Vessels loaded with blood. Convolutions in the parietal and temporo-sphenoidal regions simple. White matter of brain relatively much increased. No excess of fluid in ventricles, but in the outer wall of the right lateral ventricle was a cyst as large as a pea, containing clear fluid. No meningitis.

CASE VIII. P. D., aged 11 years, admitted December 9th, 1875, and

died March 14th, 1876. No history obtainable. He was well-nourished, with a large head, and well-marked frontal development. He was of a listless disposition, not epileptic, and though he could say a few words, his answers were quite irrational. No sign of rickets. His mental capacity was small, and though he went to school he made little progress. He was blind. Three days before death he was seized with convulsions, unconsciousness came on, and continued till his death. No convulsions had occurred previously. At the inspection the cranium was found to be thicker than normal. The dura mater was much congested and closely adherent to it, but easily detached from it was a thick false membrane which entirely covered the convex surface of the brain. On detaching the membrane purulent fluid escaped. The brain weighed $49\frac{1}{2}$ ounces. The whole convex surface was covered with a layer of pus, which extended over the under surface of the anterior and middle lobes of both hemispheres, shading off gradually posteriorly. On slicing through the brain transversely the white matter was seen to be increased in amount. The lateral ventricles were dilated and filled with fluid. The brain substance was softened and the vessels everywhere congested.

Andral states that there are two periods in this disease. In the first, the chronic stage, the symptoms are slight; in the second, unless the patient has been previously carried off by the intervention of some other disease, those characterising an acute affection appear, and the patient dies of convulsions, of symptoms indicative of compression of the brain or acute hydrocephalus. In the cases just related the patients died in convulsions in 3, in a comatose state in 2, while the remaining two were carried off by diarrhœa and bronchitis. The symptoms differ according as the cranium enlarges contemporaneously or not. If it does not enlarge, then hyperæmia added to hypertrophy is likely to lead to epilepsy. If, however, there is simultaneous development of cranium and brain, there may be few or no symptoms. The latter, no doubt was the condition in seven of the eight cases related; in the remaining one I think hypertrophy is gradually progressing, accompanied at times by hyperamia. I suspect, too, some fluid will be found in the ventricles.

None of my cases presented signs of rickets. Dr. West states that hypertrophy of the brain is associated with that condition, but he goes on to say that, as the health improves, the rickety deformity of the limbs gradually disappears. As my cases, with two exceptions, were not admitted till after the age of 10 years, and were in good bodily health at the time, any rickety deformity which may at any time have been present would no doubt have disappeared. With reference to

the connection of hypertrophy of the brain with phthisis, I find that in two of the five cases in which I have been able to obtain particulars, there was a history of that disease in the family.

As to the weight of the brains removed. In order to estimate the extent of the hypertrophy so far as the circumstance of weight is concerned, it is necessary to ascertain the average weight of the *healthy* brain in individuals of the same age as those above mentioned. I know of no tables which give such particulars. A table giving the average weight of the brains of a number of persons in a robust state of health, who have died suddenly is much required, and would be of great importance. Failing this, I turned to Dr. Boyd's well-known tables, to some drawn up by Dr. Sims, and which appeared in the "Medico-Chirurgical Transactions" of 1835, to those of Dr. Crichton Browne and Mr. Crochley Clapham, and to the table drawn up by myself as a result of the examination of 100 brains of imbecile children. I selected from these tables the average weights of brains of individuals of the *same age* as those whose histories I have related, and compared them with the weights of the hypertrophical brains, and I found that the latter far exceeded the former. I append the results in the following table. The weights are given in ounces.

TABLE showing weight of hypertrophied brains and average weight of brains of individuals of the same age.

Initials of Name.	Age.	Weight of Hypertro- phied Brains.	Average weight of Brains, Dr. Sims' tables.	Average weight of Brains, Dr. Boyd's table.	Average weight of Brains, Dr. Beach's tables.
A. H. S.	5	49½	39	40.23	40½
A. G. D.	8	53	40	45.96	39
P. J.	10	55	40	45.96	40
F. D.	11	49	44	45.96	41
P. D.	11	49½	44	45.96	41
W. J. E.	14	52	44	45.96	41
A. H.	15	62	44	48.54	42

Dr. Sims' tables include males and females, while those of Dr. Boyd and myself are of males alone. These are more useful, as the cases of hypertrophy of the brain which I have related all occurred in males. Dr. Boyd's tables do not give the weights for each year, but during certain periods of years. Thus 40·23 is the average weight from 4 to 7 years, 45·96 the average weight from 7 to 14 years, and 48·54 the average weight from 14 to 20 years. During this last period according to Dr. Boyd, the highest average weight is obtained. The tables of Dr. Crichton Browne and of Mr. Crochley Clapham include all ages under 20. Those of Dr. Crichton Browne give 43 ounces, and those of Mr. Crochley Clapham 42 ounces, as the average weight of the brains of males during that period. It is unnecessary to go through the table in order to point out *seriatim* the great difference between the weight of the hypertrophied brains and the average weight of brains of persons of the same age, as a glance at it will render this evident. I would remark, however, that the weight of the brain of A. H., not only far exceeds that of the other hypertrophied brains, but greatly surpasses the average weight for his age.

The diagnosis of hypertrophy of the brain from chronic hydrocephalus chiefly rests on the history of the case and the form and size of the head. Dr. West remarks that "the symptoms of chronic hydrocephalus generally come on earlier and soon grow more serious than those of hypertrophy of the brain and the cerebral disturbance is throughout much more marked in cases of the former than in those of the latter kind." My distinctive diagnosis of hypertrophy of the brain from chronic hydrocephalus rests on the following points:—

In hypertrophy of the brain the head does not attain so large a size as in chronic hydrocephalus. The head of my first case measured 23 inches in circumference, that of the second 22 inches. I have three cases of chronic hydrocephalus now in the asylum, and their heads measure $23\frac{1}{2}$, $25\frac{1}{2}$, and $25\frac{3}{4}$ inches respectively.

In hydrocephalus the increase in the size of the head is most marked at the temples; in hypertrophy above the superciliary ridges.

In hypertrophy the head approaches the square in shape; in hydrocephalus it is rounded (see outlines). In hydrocephalus there is often an elasticity over the late closed fon-

tanelle; in hypertrophy there is none, and there is often a depression in that situation.

In hydrocephalus the distance between the eyes is increased from the fluid inserting itself between and distending the sutures formed by the frontal and ethmoid bones; in hypertrophy this is not the case.

As to the treatment, all that one can hope to do, is to keep the patient in as healthy a state as possible, and treat any active symptoms which may arise.

