With all these various minor modifications there still remains the one disappointing fact that as soon as the treatment is discontinued the patient relapses, so that in a few weeks the patient gradually passes back into the condition of myxœdema from which he has only too lately been resuscitated. So long as the drug is in use, whether it be by hypodermic injection or the ingestion of the gland or a preparation therefrom, so long the improvement continues; but let it be dropped for a time, then without fail do we see only too soon the reappearance of the disease. But it has now been proved that only a small dose is needed to maintain the condition, and that not at very frequent intervals, and the taking of an occasional dose is surely a hardship not hard to bear even were it necessary for the remainder of the patient’s life. Although at present there are wanting real indications of a permanent cure, it would seem that after a considerable period the dose may be reduced to a mere trifle, and who knows but that it may finally be dispensed with altogether!*

(To be continued.)

General Paralysis Occurring about the Period of Puberty.

By J. Wiglesworth, M.D.Lond., M.R.C.P., Lecturer on Mental Diseases, University College, Liverpool, and Examiner in Mental Diseases, Victoria University.

We are in the habit of regarding general paralysis as in the main a disease of the prime of life—of a time when the fresh vigour of youth has subsided, but before the first touch of decay has laid its hand upon the organism; when the mental faculties are strained to the utmost in the pursuit of wealth or pleasure, or social distinction, or in the keen struggle for existence entailed upon so many of our race. We are not indeed unaccustomed to meet with cases of this disease occurring both before and after this epoch of life, but the association of general paralysis with the period of childhood and puberty has hitherto been a very unfamiliar idea. Nevertheless, scattered cases have from time to time been published which tend to show that the period of life which appears to offer most exemption from all the ordinary causes of the disease may still claim its victims, and that at, or

* Such, too, are the views of Dr. Robert Lundie, as contained in an interesting and popular article, "A New Departure in Medical Treatment," which he has lately contributed to "Chambers’s Journal," May 6, 1893.
about, the period of puberty cases may occur which, both clinically and pathologically, appear incapable of separation from the ordinary forms of the disease with which we are so familiar.

So far back as 1877 Dr. Clouston published a case of general paralysis occurring in a boy aged 16; and in 1881 Dr. Turnbull recorded another one, also in a boy, which started at the still earlier age of 12; whilst in 1883 I myself reported a case in a girl commencing at the age of 15. References to a few other juvenile cases reported by Régis and others will be found in Dr. Mickle's invaluable work on general paralysis.

Attention has, however, more particularly of late been directed to this subject by the publication of Dr. Clouston's work on the "Neuroses of Development," in which two fresh cases are fully recorded under the title of "Developmental General Paralysis," both in girls, the disease in each case commencing about the age of 15. Previous, however, to the appearance of Dr. Clouston's work I had had two more cases of this description under my care, and in March, 1891, I had the pleasure of calling Dr. Clifford Allbutt's attention to one of them in the wards of Rainhill Asylum, and Dr. Allbutt has alluded to these cases in his article on "Insanity in Children" in Dr. Hack Tuke's "Dictionary of Psychological Medicine." Recently another case has been reported by Charcot and Dutil in a boy, which commenced at the age of 14.

I may also in this connection allude to a paper by Dr. Shuttleworth "On Idiocy and Imbecility due to Inherited Syphilis." It is true that Dr. Shuttleworth does not class any of his cases as general paralytics, nor, indeed, allude to this subject in connection with his paper, inclining to the belief, expressed by Heubner, that the "progressive pathological change" observed in his cases was to be referred to affections of the cerebral arteries, the calibre of which, having become narrowed by endo-arteritis, more or less cerebral atrophy was produced. But Dr. Shuttleworth, in a communication on the subject with which he has been good enough to favour me, tells me that cases I., III., and IV., described in his paper, were very like Dr. Clouston's cases which he saw at Morningside; so that the possibility of some of Dr. Shuttleworth's cases being really examples of juvenile general paralysis appears open to discussion.

I will now give a short account of the two cases already
alluded to which I have recently had under my own care, and
will then briefly summarize the leading features of these and
of the other published cases referred to.

Case I.—M. E. M., girl, aged 15, was admitted into Rainhill
Asylum on September 19th, 1888. Her parents were living,
healthy, and temperate, the father being fifty years of age and the
mother forty-two, and they had been married eighteen years.
Patient was the second child in the family, and she was one of
three survivors out of a total of thirteen pregnancies, eight of which
were females and five males; of the eight female pregnancies
patient was the only survivor; two of the others were miscarriages
at five months, two were stillborn, one (an eight months child)
died two weeks after birth, and the other two died aged three and
a half years and seven years respectively of scarlatina. Of the
five male pregnancies, one died aged five weeks in a fit, one aged
two years of some cause unknown, and another, aged two and a
half years, of scarlatina; the other two were living. Thus out of
the total number of 13 pregnancies no less than six were either
miscarriages, still-births, or died within a few weeks of birth—a
history certainly suggestive of syphilis, though no other evidence
of this disease was forthcoming. There was no history of nervous
disease in the family, nor indeed anything worthy of special note.
Patient herself was said to have been a bright child until eleven
years of age, and had passed the third standard at school. At this
time she was running one day in the street when she slipped and
fell, striking her head violently on the kerbstone; she was uncon-
scious for two hours, and was several days in bed with headache.
Some months after this she appears to have developed some weak-
ness of the limbs, for which she was treated in the Liverpool Royal
Infirmary. About a year from the accident, patient being then
twelve years of age, it was noticed that she was getting dull and
losing her memory, and from that time her mind appears to have
gradually faded away, though the downward progress had been
more rapid during the few months preceding admission. During
the three or four years previous to admission she had had several
falls apparently as the result of paresis of her limbs, and on one
occasion fell down a whole flight of steps and had convulsions all
night afterwards. Possibly some of these falls were really
examples of paralytic “seizures.”

The facts certified on admission were:

Her intelligence is very defective. She answers “I don’t know”
to all questions. Her powers of attention and understanding are de-
ficient. She is quite unable to look after herself. She is inattentive
to the calls of nature, and occasionally noisy.

When admitted, though fifteen years of age, she was described as
a well-developed child, and the signs of puberty appear to have been
but slightly marked, though her mammary were partially developed.
She had never menstruated. Her pupils were slightly dilated and
of normal reaction. Her viscera were sound. Mentally her con-
dition was one of considerable dementia. She was quiet and
tractable for the most part, and sat quietly most of the day taking
no interest in her surroundings; when touched, however, she
cried and seemed frightened. She had much loss of memory,
could give scarcely any account of herself, and could only be got
to answer correctly the most simple questions. She was wet and
dirty in her habits. She remained much in this condition for
about three months, when, at the urgent request of her mother,
she was sent out to her care. Five months later, viz., in May,
1889, she was readmitted, and in the interval the disease had
made rapid progress. She was now a complete wreck, both
mentally and physically. She was thin and haggard, and so feeble
that she could not stand unsupported. Her mind was a blank;
she could give no particulars about herself and could not even
tell her own name; she seemed, indeed, to understand nothing of
what was said to her. She continually moved to and fro in her
chair, uttering a crowing, meaningless laugh, and when touched
she cried loudly and continuously. Her tongue was tremulous, as
also were her lips, and her speech was hesitating and ejaculatory.
She was wet and dirty.

From this time her course was rapidly downward. She became
so paralyzed that she was shortly confined to her bed, where she
remained until her death, and her limbs soon became strongly
contracted in flexion. She screamed a good deal at times, but
showed no sign of intelligence. All her evacuations were passed
under her. She became excessively emaciated, literally being
mere skin and bone, and bedsores developed on all points exposed
to pressure. She finally died on August 31st, 1889, being at that
time 16 years of age. No convulsions were noted at any time
during the period she was under observation.

The patient having died during my absence from home, I
unfortunately did not see the autopsy, but the following is
the record of it (19 hours after death):—

Body extremely emaciated, lower limbs rigidly contracted in
flexion. Sores on both elbows, trochanters, sacrum, and heels,
inside of thighs, backs of hands, shoulders, and dorsum of feet.
Cranium.—Skull cap thin and very dense, not adherent to dura.
Superior longitudinal sinus empty. Great excess of subdural
fluid, about 450 c.c. Encephalon, 885 grammes. Right hemi-
sphere, 348 grammes. Left hemisphere, 290 grammes (both un-

Great and general opacity of arachnoid, the white lining along the
course of the vessels being a prominent feature. Vessels prominent,
but no general congestion. Large excess of subarachnoid fluid,
Pia mater and arachnoid stripped readily over the whole of the left hemisphere, except along the superior temporo sphenoidal convolution and the angular gyrus; here extensive decortication occurred, though the adhesion was very slight, the cortex being extremely soft. Great and general wasting of gyri. Ventricles enormously dilated, the cornua especially so. Slight roughening of floor of fourth ventricle. Great wasting of cortex, the grey matter being reduced in thickness to about \( \frac{1}{2} \) normal. Striation for the most part completely absent. White matter very pale, almost no puncta cruenta. Basal ganglia dark-coloured and softish. Brain as a whole firm, though surface layers very soft. Cerebellum rather pale and firm. No macroscopic lesion of pons or medulla, which were fairly firm.

**Thorax.**—Right lung, 6 oz.; left, 4½ oz. Both dry and partially collapsed. Heart, 3 oz., soft and flabby, but otherwise normal. Pleura and pericardium quite normal.


I will now proceed to describe the next case.

**Case II.**—C. K., girl, aged 15, admitted November 17th, 1888. Her friends neglected her and did not visit her, and the history was only obtained with considerable difficulty upwards of two years after her admission.

Father living, aged 50; he was a heavy drinker, but no further information could be obtained about him; her mother died at 39 years of age, of phthisis; she was 18 years of age at the time of her marriage. There were seven children born as the result of the marriage.

1. Girl, aged 24 (who gave these particulars of the history); she had been married three years, and had had one child stillborn at seven months.
2. 3, and 4. All died in infancy of convulsions and teething, etc.
5. Patient.

The sister stated that patient was a bright child, and was in the fourth standard at school when between 10 and 11 years of age. Four years before admission, viz., in 1884, she one day fell down stairs and hurt her head and leg, for which injury she was treated in the Northern Hospital, Liverpool, for two months, the entry in the books of the hospital stating that she suffered from "osteitis of tibia." Her mother then died, and her father being a great drunkard she was much neglected, and depended on neighbours for chance meals. She became thin and weak, and it was thought she was going into a consumption. Six months before admission she was noticed by her sister to be "queer" at times, and she:
became subject to "faints," from which she soon recovered. The medical certificate upon which she was admitted stated that "she sits in one position all day, takes no interest in her surroundings or future prospects, answers questions very unwillingly and stupidly. Her intellectual powers and memory are deficient. She is unable to look after herself; does not go to her food unless fed."

When admitted she was noted to be a girl of fair physical development, but her mammae were small, and she did not display any signs of puberty. She had never menstruated. Viscera sound. Pupils normal. No noticeable paralysis, but appeared awkward on her feet, and tripped over any irregularities on the floor. Knee jerks very brisk. Plantar reflexes normal. Mentally she was in a dull, listless state, with a tinge of depression, taking no interest in her surroundings, although she appeared to understand something of what was going on around her, and when roused could be got to answer a few simple questions, giving her age correctly, for instance; very few particulars about her family could, however, be elicited from her, and her memory was evidently at that time considerably impaired. She was clean in habits. She remained in this sort of semi-stuporose condition for many months, capable of being roused to answer questions, or at times to laugh at trifles, but sinking back again at once into her usual state. Then, rather more than a year after admission, she had several well-marked epileptiform convulsions, and soon after this, viz., in March, 1890, she was noted to be steadily growing worse, her mental condition being one of slowly progressive dementia. She was also at that time becoming unsteady on her legs, and dragged her feet when walking. Sensation appeared normal, but her mind was too dull for reliable data of this kind to be ascertained. She was already beginning to lose flesh. A few months after this, owing to the advance of the paralysis, she was confined to her bed, and her condition assumed the characteristics which persisted for nearly two years—up to the time of her death—with very little change. She was completely paralyzed, and lay huddled up in bed with all her limbs rigidly contracted in flexion, nor could these be straightened by any reasonable force; knee jerks unattainable owing to the rigid contracture. The excitability of the muscles was found about equally diminished to all forms of electrical stimulation. She was wet and dirty. She gradually developed small bedsores over points of pressure—elbows, hips, etc.—but these did not enlarge to any size, and showed a tendency to heal as time went on. Reflex closure of the eyelids when the hand was brought near the face was very marked. At the commencement of her bedridden period she frequently repeated words and syllables spoken in her hearing in an automatic manner, but latterly she was very silent, scarcely uttering a sound, except to cry when disturbed. She was quite fatuous, displaying no sign of intelligence. She ground her teeth for hours together after the
fashion of the most typical general paralytic. For some time before her death she had not sufficient intelligence to protrude her tongue, but at an early period this was markedly tremulous as a whole when protruded, and the tremor spread to the muscles of the lips and face; her speech also was distinctly quavering. She was greatly emaciated. Pupils much dilated; right 7 m.m., left 7.5; sluggish, but contracted to strong light. Optic discs normal. As she lay in bed, with her small wasted limbs and features, she had all the appearance of a child of nine or ten years of age, instead of being, as she then was, in her 19th year. There were no signs of menstruation all the time she was in the asylum. She died on March 3rd, 1892.

Autopsy* (seven hours after death).—Body much emaciated. Great contracture of all the limbs.

Ossium.—Skull cap: thickness and density slightly increased. Slight adhesion of dura to bone. Sinuses moderately full. Inner surface of dura mater coated with an old laminated membrane, which covered both hemispheres, extending down on each side as far as the 1st temporal gyrus, backwards to the tip of the occipital lobe, and forwards to the tip of the frontal lobes; it was evidently of old date, and was adherent to the dura, though it could readily be detached from it. Near its margin it was transparent, of a straw colour, and extremely thin, but towards the vertex of the skull it rapidly increased in thickness, and measured here from 1.5 m.m. to 2 m.m. thick. It appeared quite free from hemorrhage, but was plentifully supplied with blood vessels. In its thicker parts it was very distinctly laminated, opaque, and of a brownish colour. Inner surface of dura smooth and shining when the membrane was removed. Great excess of almost clear colourless serum in subdural space, appearing not only to distend the dura mater, but to float up the brain, as when the fluid was drained off the brain sank to the bottom of the cranial cavity, and it became evident that it did not fill more than $\frac{1}{2}$ to $\frac{2}{3}$ of the cranial space. The encephalon as a whole only weighed 720 grammes. Arachnoid thickened, and showing widespread opacity. Marked adhesion between frontal lobes and across Sylvian fissures, the adhesions showing themselves as thick bands of membrane bridging across the fissures. Slight general thickening, and marked general hyperœmia of pia mater. No decortication on stripping, but the arachnoid showed a tendency to separate from the pia, leaving portions of this behind on the cortex. Great excess of serum in subarachnoid space. Convolutions of cerebrum immensely wasted, the sulci widely gaping, these conditions being particularly marked on the inner aspect of the right hemisphere; the wasting was less marked in the occipital lobes than elsewhere. Surface of convolutions red in colour.

* Pathological notes by Dr. Wynne.
Right hemisphere (unstripped) weighed 287 grammes; (stripped), 265. Left hemisphere (unstripped), 290 grammes.


Cortex purplish red in all parts, of firm consistence and increased vascularity; depth much diminished, and striation very indistinct. White matter slightly yellowish in colour. Ventricles much dilated, containing clear fluid.

Ependyma not granular. Corpora striata, optic thalami, corpus callosum and fornix all unusually small and rather too firm. Cerebellum; cortex small, but not out of proportion to size of organ. Pons small.

Medulla oblongata.—Fourth ventricle slightly dilated; ependyma a little granular at calamus scriptorius.

Spinal Cord.—Smaller and firmer than usual. Membranes hyperemic but not thickened.

Spinal Nerves.—Brachial plexus, great sciatic, and anterior tibials small, but not out of proportion to muscular development; appeared normal.

Microscopical Examination.—Sections from superior frontal and from parietal and occipital lobes (fresh condition) all showed a fine spider-cell formation, which passed deep into the cortex; the nerve cells were much degenerated and distorted, apparently by contraction of the connective tissue elements. Vessels thickened and showed proliferation of the nuclei. The spinal cord showed small patches of sclerosis in the posterior columns in both cervical and dorsal regions. There was marked sclerosis of the posterior part of the right lateral column throughout the cord, and in the cervical region of the left lateral column also; there was also a small patch of sclerosis of this left lateral tract in the dorsal region, but none at all in the lumbar.

The peripheral nerves above noted were examined fresh and after hardening in osmic acid, and staining with picricarnine. No segmentation of the myelin and no nuclear proliferation were detected; the nerve fibres appeared quite healthy, and in teasing out no undue roughness or increase of connective tissue was observed.

Thorax.—Right pleura; local empyema at base. Right Lung, 11oz.; upper lobe in a condition of complete pneumonic consolidation. Left Lung, 6oz. At apex scars of old tubercle. Small bronchi dilated.

Heart.—Weight 5oz., normal.

Abdomen.—Liver, 23oz., congested; capsule not thickened; no scars.

Spleen.—2oz., pale and firm.

Kidneys.—Right, 3oz. Left, 3oz.; healthy.

Other organs healthy.

No evidence of syphilis.
If now we add to the above two cases the six others referred to at the outset of this paper, we get a total of eight cases in which the disease commenced at or about the period of puberty, which may be briefly subjected to analysis. These cases include three reported by Clouston, one by Turnbull, one by Charcot and Dutil, and three by myself.* I have only seen an abstract of the case published by Charcot and Dutil. It may be premised that of the eight cases two were living at the time the cases were reported; in the remaining six the disease had proved fatal, and the diagnosis had been confirmed by post-mortem examination.

1. The age of the youngest patient at the time the disease commenced was 12 years, that of the oldest 16, the average of the whole being 14. It is, of course, impossible to state with precise exactitude the time at which the disease made its first appearance, and probably, if anything, the patients were a little younger than the above figures indicate.

2. The duration of the disease shows a tendency to be prolonged. Of the six completed cases the duration of the shortest case was three years, of the longest six, the average of the whole being 4½ years, which is certainly rather a longer average duration than that of ordinary adult general paralysis. Here, again, these figures probably understate the actual duration.

3. The high proportion of females is a noteworthy feature, five of the cases having been girls and only three boys. Of course from such a small number of cases one must be careful about drawing general conclusions, but it is unlikely that the above proportion is altogether accidental. Having regard to the large preponderance of adult male general paralytics over female, it would appear that in these juvenile cases the disparity between the sexes tends to disappear, the incidence of the disease being more equally divided between them.

4. The mental state is of interest, as indicating an immense preponderance of the demented type of general paralysis. In only one case (one of Clouston’s) were any grandiose ideas present, and these only in a slight degree; in all the other cases the condition appears to have been one of dementia, from first to last; this was very marked in my own cases, a gradual, almost imperceptible failure of mental

* I have not been able to include in this summary all the cases referred to by Mickle, as details of the cases are not given by him, and I have been unable to consult the original papers.
power having been noticed from the first. The cases, indeed, seem to have partaken more of the degenerative than the sthenic type of general paralysis.

5. It seems to be the rule either that the signs of puberty do not appear at all, or if they have commenced that they are arrested, and tend to disappear as the disease progresses, menstruation in the females being absent. And pari passu with the above, and, indeed, as a part of it goes, arrest of the bodily development generally. This has been especially remarked upon by Clouston, and in my own cases the child-like appearance of the patients when the disease was well advanced was a very striking feature, although the last two patients at the time of death were aged 16 and 18 years respectively.

6. The excessive emaciation exhibited by both of my cases was a very striking feature.

7. The extreme atrophy presented by the brains was also very noteworthy. In the case of M. E. M. the brain, with membranes adherent, immediately after removal from the cranial cavity weighed 885 grammes, whilst in the case of C. K. the brain in similar condition weighed only 720 grammes; this, it must be remembered, was from a person then in her 19th year, who was not microcephalic (her head circumference was 20 inches).

8. On the question of aetiology hereditary tendency was distinctly traced in four of the cases, and in the fifth it probably existed (in Turnbull’s case the father of the patient was himself a general paralytic), whilst in two others there was marked alcoholism in one or both parents, so that we may fairly consider that there was a neuropathic taint in 4 of the cases—an immensely higher proportion than obtains in ordinary adult general paralysis.

Next, perhaps, to heredity, syphilis appears to be a cause. In both of Clouston’s last two cases the syphilitic history was well marked, and both patients exhibited in their persons evidence of congenital syphilis. In one of my cases (M. E. M.) a syphilitic taint was suggested by the history, though the evidence was not conclusive. I have already referred to the possibility of some of Dr. Shuttleworth’s syphilitic imbeciles being examples of juvenile general paralysis.

The only other factor which seems to stand out at all prominently is traumatism. Both of my latter two cases had had severe falls on the head, and the disease was said to
have dated from about the period of the accident. It is, of course, necessary to be cautious in accepting this as an aetiological factor, for apart from the tendency of parents to assign an injury as the exciting cause of mental disease, there is the further fallacy that the fall may have been occasioned by a paralytic seizure, which may itself have been the first symptom of the disease. In both my cases, however, the falls appear to have been of a severe character, and I am disposed to regard the injury as a factor in the production of the disease. I may also mention parental neglect as a contributing factor, which appears to have been operative in several of the cases.

I remarked at the outset that these juvenile cases of general paralysis occurred at a time of life which appeared to offer most exemption from all the ordinary causes of the disease. But here we are reminded that the above causes which appear to have been operative in the cases analyzed are also causes generally recognized as operative in adult general paralysis. Heredity, syphilis, traumatism are all regarded as causes of general paralysis, although the relative importance assigned to each by different authorities varies greatly. It might be inferred, indeed, that these causes were more potent when acting in early life, but in the immense number of mental and nervous disorders owning a neuropathic heredity how seldom do we meet with these juvenile cases of general paralysis; and whilst the congenitally syphilitic are fairly common, it seems very rare to meet with general paralysis amongst them, though possibly cases are more frequent than published records would seem to indicate. Dr. Clouston suggests that in these cases the strains of development at puberty may have the same effect as strains and undue outputs of energy in after life have in other cases in causing the disease. And possibly when an individual is strongly predisposed to nerve degeneration by reason of neuropathic heredity, or inherited syphilis, or other cause, there may not be sufficient energy left in the organism to enable it to respond to the great calls upon the nervous system which the onset of puberty entails, and hence a deadly decay may take the place of that development and higher life which is the normal outcome of this epoch.

But the period of adolescence is also by no means free from attacks of general paralysis. I give here in very brief outline the notes of two cases which have been under my
care, in each of which the disease commenced at the age of 18 and proved fatal at 20, which cases I have not included in the above analysis, since they do not in strictness come within the scope of the title of this paper.

John McC., aged 19. Admitted August 21, 1890. Both parents very intemperate. Patient was said to have been naturally weak-minded, but had been to school, and was in the 4th standard when he left. Fifteen months before admission he had a fall on the back of his head, and lay unconscious for four days in convulsions; was said never to have fully recovered his mind after this. When admitted he was in a condition of advanced dementia. He could not be got to answer a single question rationally, and did not appear to understand anything that was said to him; he was also excited and noisy, shouting and chattering, and muttering an incoherent jargon. Was wet and dirty. He died on February 27, 1891, and at the autopsy the brain was found to have typical general paralytic characters. It weighed 1,185 grammes. The arachnoid formed a dense white opaque watery membrane over nearly the whole of both hemispheres, and there was great congestion of the vessels of the pia mater. Great and pretty general decortication occurred on stripping the membranes. There was great wasting of the convolutions, atrophy and darkening of the cortex, and the ventricular floors were very granular.

Alice S., aged 20. Single. Admitted April 8th, 1889. Was formerly a barmaid, and had been deserted by her paramour, and since then had been on the streets. Was confined of a child in the workhouse two years before admission, after which she had an attack of mania, which was clearly the starting-point of general paralysis. When admitted she was in the last stage of the disease, quite fatuous, wet and dirty, could understand nothing, but cried out at intervals. Could not walk or stand. Tongue could not be fully protruded. Died two months after admission, viz., on June 8th, 1889. The brain was typical of general paralysis. There was considerable opacity of arachnoid and injection of pia mater, and the latter membrane was so adherent that it could not be stripped anywhere without decortication. Great wasting of gyri. Ventricular floor granular. Weight of brain, 1,070 grammes.

Between 20 and 30 years of age, as we know, the disease is fairly common, and we all could quote several examples of it.

It is thought by some that general paralysis is becoming more frequent now than formerly, and certainly, with the constant straining effort entailed by modern civilization, we are not likely to see a diminution in the victims of this
disease; and, along with this, it may be that the disease is
tending to appear in the individual at an earlier age than
formerly. Be this as it may, I submit that the above cases
prove that the disease may be met with at a far earlier age
than has until lately been thought possible, and that the
period of puberty, and even childhood, can no longer be
regarded as exempt from its attacks.*

Since the above paper was written I have had another
case of juvenile general paralysis under my care, in a boy,
which proved fatal at 16 years of age. The case bore a close
resemblance to those above described—the progressive
paralysis, with complete fatuity and great emaciation, to-
gether with contractures, being prominent features. The
brain, which weighed 912 grammes, was very typical of
general paralysis, there being great opacity and thickening
of membranes, extensive adhesion and decortication, and
immense wasting, etc. The duration of the disease could
not be ascertained, but he was in a very demented state a
year before his death. His father was English and his
mother Italian. Both parents were very intemperate, and
parental neglect and a condition of semi-starvation were
prominent features in the case.

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de Neurologie, March, 1892 (abstracted in British Medical Journal, May 28,
1892).

* Dr. Percy Smith has kindly sent me notes of a case of a youth of 15, seen
at Bethlem Hospital as an out-patient in 1890, with symptoms closely resem-
bbling those of general paralysis. He was admitted for a short time into St.
Thomas's Hospital, but the symptoms remaining stationary he was taken away,
and the case has been lost sight of. As Dr. Percy Smith did not see the case
again, he is unable to speak decidedly as to the diagnosis.