fear of any tuberculous tendency in the latter. We acknowledge, in fact, that the members of one family throw back to different ancestral strains, either on the father's or the mother's side, and that they do this not only in external appearances but in tendencies to disease, and this is still one of the points which wants close and careful investigation.

The different tendencies of an individual in either childhood or age may be regarded as a kind of reversion to different ancestors at various periods of life. This has been beautifully put by Oliver Wendell Holmes in his novel, *The Guardian Angel*. The heroine of this story, Myrtle Hazard, runs away from home, and, floating down the river in a boat, sees at dusk, in an old Indian burying place, a curious vision. Around the burial mound various figures seem to flit, some thin and shadowy, others more substantial, but still misty, while others again appear almost as distinct as living men and women. She recognises, as one does in a dream, that these are her ancestors—the solid ones closely related to her in blood, while the thin and shadowy are far removed. Each ancestor has an existence in herself, more or less definite, according to the nearness or remoteness of blood. It is not necessary to trace our heroine through her various adventures, but at one time she develops suddenly and unexpectedly, under provocation, the characters of an Indian squaw, from whom she has descended, and nearly slays a schoolfellow while they are acting together. Later on another ancestress who was a famous beauty appears in a dream, and for some time afterwards the heroine feels this beauty living within her and directing her actions, while, later still, another ancestress, who has been a great saint, drives the capricious beauty out and leads the heroine in the way she should go.

As I have already said, Haeckel believes that, during intra-uterine life, the foetus rapidly runs through the development of the race up to mankind; and I think we may in after-life trace the progress of the individual through various ancestors more and more slowly as he advances in years. In infancy and childhood we frequently notice how rapidly the features change; and friends will say at one visit how like the child is to some member of his father's family, while a week or two after they will notice a resemblance to someone of his mother's family. If this Society can gather together notes as to whether an individual presenting an unwonted reaction to medicine happens to resemble at the time someone either of his father's or his mother's family who had a similar peculiarity, a great step might be made. This, however, is not all; we have next to try to trace why medicine acts differently upon different families. It is just possible that this, also, is a case of reversion to a very far-off period; indeed, before mankind had ever reached the level of the monkey. Some years ago it occurred to me that one might possibly gain some information regarding the peculiar differences in the action of drugs on gouty and other individuals, by comparing the effects of certain poisons or remedies upon such animals as rabbits, which excrete their nitrogenous waste in the form of urea, and pigeons, which excrete it in the form of solid urates. Now, Weir-Mitchell has shown that pigeons have a singular power of resistance of the action of opium, and can take large quantities of it without presenting any symptoms of narcosis. Dr. Cash and I made some experiments upon the subject, and we found that opium in moderate doses appeared to have hardly any action upon pigeons; yet, on examining more closely, we found that it had a very marked action, only it did not produce sopor, but caused the temperature to fall greatly; instead of acting as a narcotic it acted as an antipyretic. We did not try the converse of this by giving antipyretics, such as antipyrin, to pigeons, and seeing whether they acted as soporifics; but in patients we certainly do find that occasionally, as in acute inflammation, opium seems to have an antipyretic action, whereas antipyrin certainly acts at other times like a soporific. In order to track these actions further back still, and find out on what changes in the circulation, blood, nerve cells, and nerve fibres, the action of these various drugs depends, a large amount of experimental work in laboratories is required.

Until lately there has been no opportunity for young men generally to do such work in this country, and those who wished to undertake it were obliged to go abroad. Now, how-

ever, owing to the wise action of the Colleges of Physicians and Surgeons, opportunities are being afforded for such investigations, but all such researches will give only one side of the subject, and will remain valueless unless clinical observation can be brought to bear at the same time on therapeutic studies. The members of this Society, by the fact that they have founded it, have shown their desire to forward the progress of medicine. I have to-night tried, in a very imperfect way indeed, to consider some of the methods by which I have thought their objects might be to some extent attained, and even if I should have failed to show you how to work, this evening may not have been uselessly spent if I have succeeded in directing your attention to the search after methods of attaining the objects which we all have at heart—that of securing health and ease to our patients with quickness and certainty.

REMARKS

ON

TWO CASES OF PANCREATIC DIABETES:

THE ONE EXCEPTIONALLY ACUTE, THE
OTHER MARKEDLY CHRONIC.

By VAUGHAN HARLEY, M.D., M.R.C.P.

(WITH COLOURED PLATE.)

As cases of pancreatic diabetes are, comparatively speaking, rare, and the disease is at present attracting much attention, especially on the Continent, I think it well to put on record two exceptionally instructive cases, to which my attention was called by Professors Heiberg and Torup while I was working experimentally on the nature and causation of pancreatic diabetes in the Physiological Institute of Christiania.

I do so all the more readily from the cases not having been hitherto published, notwithstanding that they present us with interesting typical examples of the two opposite forms of the disease, inasmuch as the one occurred in a youth, and ran its fatal course in the brief period of eleven days, while the other occurred in a man aged 64, and lasted four years. Both the cases are all the more clinically valuable, seeing that the signs and symptoms of each were not only carefully recorded during the patients' lives, but the *post-mortem* appearances were equally carefully noted immediately after their deaths.

The signs and symptoms of diabetes, either depending upon or associated with pancreatic disease, are known to present in several respects marked differences from those usually manifested in cases of diabetes unconnected with a derangement of the pancreatic functions, the onset of pancreatic diabetes being usually exceedingly sudden, for the characteristic signs of thirst, voracious appetite, and polyuria, accompanied by a saccharine condition of the urine, in general, set in without any premonitory symptoms whatever. It differs still further from the other forms of diabetes, inasmuch as it not only usually limits its attacks to the young, and often apparently healthy, but in the rapidity with which it marches on to a fatal termination. The nervous prostration, muscular weakness, and emaciation with which it is invariably associated, is quickly followed by a comatose condition, precipitately ending in a lethal collapse. With the above symptoms is associated the presence of undigested fats in the stools. These being the most characteristic signs and symptoms of pancreatic diabetes, I will at once proceed to give the histories of two cases which may be regarded as typical examples of the two most striking varieties of the disease. The first is that of a medical student who died in Christiania in September, 1890, while I was working there, and for the history of which, as well as for the specimen from which the accompanying coloured drawings were kindly made for me by Dr. Julius Nicolaysen, I am indebted to Professor Heiberg. I must also acknowledge my obligations to Professor Gulberg, who kindly prepared the microscopical sections from which the drawings were made.

Y. K. aged 23, had as a child enjoyed good health, although

he was pale and somewhat scrofulous-looking. At school, he always held a good place in his class. As he grew up he was less mentally bright and his eyes assumed a dull appearance, which with his large hanging lips, gave to him a somewhat stupid look. On entering the university at the age of 22, he did not seem either mentally or bodily strong, but as he did not complain of having anything wrong with him, he was accepted as a soldier for summer service—entering upon it in June, 1890. The ordinary drill, however, exhausted him to such an extent that he was discharged after a fourteen days' trial. In August he was attacked with catarrhal tonsillitis, which weakened him very much, making him complain of languor, headaches, and sleeplessness. At the same time it was noticed that, not alone was he drinking a great deal of water, but was constantly complaining of hunger. This led to his urine being examined for sugar, and the discovery that he was diabetic. Almost immediately afterwards he became very restless, although at the same time drowsy, and was constantly asking to be taken out of bed; but his weakness was so great that he could not sit up for more than a few minutes at a time.

His thirst became very marked, but not so his appetite. The quantity of urine passed varied from 140 to 212 ounces (4 to 6 litres) in the twenty-four hours, and contained on an average 2 per cent. of sugar. His tongue was dry, and his breath had the characteristic apple-like odour.

His mental and physical weakness became day by day more marked, and he rapidly drifted into a comatose state, although up till the very last day of his life—which only lasted eleven days after the sugar was discovered in the urine—he could be temporarily roused out of his stupor.

At the necropsy, the liver, heart, lungs, and spleen were found perfectly normal in appearance. The kidneys were normal in size, and their capsules easily stripped off; but their surface had a reddish-grey colour, and was marked with yellowish striæ. On section the cortex had the same reddish-grey colour, the pyramids being of a bright red. On microscopic examination the epithelial cells of the tubules were found to be finely granular, and the addition of liquor potassæ showed fatty degeneration. After hardening, sections showed some of the tubuli to be devoid of cells.

The pancreas alone showed any marked pathological alterations. Its head was converted into an irregular-shaped mass, of about the size of a duck's egg, which adhered closely to the duodenum (Fig. I.). Nothing remained of the body and tail of the gland but a thin flat fibrous band.

In the head of the pancreas was a cavity of the size of a cherry, filled with greenish-yellow semi-purulent fluid. The surrounding tissues were of a greyish colour, and filled with numerous small abscesses, varying from the size of a pea down to little specks, scarcely recognisable to the naked eye. All of these contained the same yellowish green-looking semi-purulent fluid. (Fig. II.)

The fluid when examined microscopically showed numerous leucocytes, and micrococci arranged in groups or short chains. Some of this fresh purulent fluid, when inoculated into rabbits, caused local abscesses at the seat of the inoculation. Cultivations were made with the fresh pus, and the gelatine tubes rapidly liquefied and deposited a yellow sediment. Added to this agar-agar tubes showed a yellowish-gold band at the stræ of inoculation. So that the purulent fluid in the pancreas evidently contained the staphylococcus pyogenes aureus.

In hope of also finding in it tubercle bacilli, some tubes to which glycerine was added were inoculated, but they only yielded cocci; no bacilli being found on careful search. Also pus stained for tubercle bacilli gave negative results.

The common bile duct was traced behind the head of pancreas, and was found to be quite unaffected by it. The duct of Wirsung could be traced into the substance of the gland, where, however, it became lost.

On microscopic examination of the head of the pancreas, the lobules were found to be atrophied (Fig. III. A), and the gland cells fatty. The interlobular connective tissue was greatly increased (C), and pressed on the atrophied lobules. Scattered throughout the sections were small abscesses containing leucocytes (B). Their walls were formed by the hypertrophied interlobular connective tissue. In some parts the glandular lobules were found to consist of merely a mass of

fat globules, in which no structure was visible. But after dissolving out the fat, remnants of glandular (B) and connective tissue (A) were found, as shown in Fig. IV. In none of the sections, however, could healthy gland tissue be detected.

For the notes of the history of the next case I am indebted to Professor Sophus Torup. It is in every respect the very antithesis of the preceding, and consequently is most instructive by way of contrast to it. The patient was a rich banker, who died at the age of 64 from diabetes, with cancer of pancreas. The case possesses some very special features, the first being that it is very unusual to meet with diabetes from pancreatic disease in persons of so mature an age; the second being that it ran a far more chronic course than is in general met with; while the third is that it was only towards its termination that it presented the two most characteristic combined signs of pancreatic diabetes, namely, extreme emaciation and fatty stools. It appears probable, therefore, that it began as some other form of diabetes, and only in the end, when treatment had no effect on the quantity of sugar, developed into one of pancreatic diabetes. In addition to these important features, the case has a further exceptional clinical interest, inasmuch as during the whole four years the disease lasted careful quantitative analyses of the amount of sugar present in the urine were repeatedly made. So that from its very beginning in 1882, until its fatal termination in 1886, we have a reliable record of how the quantity of saccharine matter varied according to the different conditions of the patient.

The family history of the patient was that his father died from a urinary vesical calculus. One of his father's brothers died of diabetes, complicated with cancer of the stomach. Another brother also died of cancer. The patient, however, always considered himself healthy until the summer of 1882, when he had an attack of boils. His urine had been for some reason or another examined for sugar in 1881 without any having been found, but after the attack of boils it was found to be saccharine, the amount of sugar present being 1 per cent. In the summer of 1883 the sugar had increased to 5 per cent., so he went to Carlsbad and took a course of the waters. On his return the amount of sugar had greatly diminished, being only 0.5 per cent. During the winter it again rose, to 6 per cent., and as it had risen to 7 per cent. in the summer of 1884 he returned to Carlsbad, with the result that the quantity of sugar was reduced for a second time to 0.5 per cent.

As in the summer of 1885 the sugar had once more increased, he took a third course of the Carlsbad waters, with the effect that the sugar fell to 1 per cent., which, although twice as much as on the two former occasions, was regarded as satisfactory. Except that he now began to complain of being very thirsty, he felt well and vigorous, and there was no perceptible loss of flesh. Having no pain, or any other symptom of disease about him, he did not diet himself after returning from Carlsbad until the sugar again increased in the urine, when he found it easy to reduce its amount to from 1 to 2 per cent. by a strict albuminoid dietary.

In the beginning of 1886 he began to lose his appetite, and got sensibly thinner, while the sugar at the same time little by little increased, till by the summer time it had risen to 9 per cent., so he again went to Carlsbad. On this occasion his visit was, however, unattended with any beneficial results. On his return home he gradually lost more and more flesh, and his strength steadily decreased. His appetite equally notably diminished.

In the month of August a deep-seated, slightly irregular-surfaced tumour was detected in the middle line, about midway between the ensiform cartilage and the umbilicus. It was slightly mobile, and somewhat tender on pressure. At the same time, as quantities of unemulsified fats of the food were noticed in the stools, there could be no doubt of the existence of pancreatic disease. By September the skin of the whole body had assumed an icteric tint, and this became more and more marked up till his death on October 5th.

Necropsy.—Skin deeply jaundiced; rigor mortis marked; scarcely a trace of subcutaneous fat; the pericardium contained a little yellow bile-stained serum; the heart was normal in size, very pale, and devoid of fat; the valves were normal. The pleural cavities contained 100 c.c. of bile-stained serum. The lungs were emphysematous, but were otherwise normal. Most of the lymphatic glands were normal, but a few showed

signs of commencing caseous degeneration. The abdominal cavity contained a considerable amount of yellow, bile-stained, somewhat turbid serum, with here and there fibrinous coagula floating in it. The peritoneum, more especially towards the lower abdomen, had a thin fibrinous deposit on its surface. The stomach was normal; the intestines slightly injected. In the lower portion of the duodenum there was an ulcer with red edges, 3 centimetres in diameter. In its centre was a fistulous opening, through which a probe could be passed into a cavity behind it, and on withdrawing the probe and applying pressure a whitish, viscid fluid, somewhat resembling cream in appearance, flowed out. There were two other smaller fistulous openings in the duodenum close to this large one.

The sound entered the common bile-duct for about 5 or 6 centimetres, but there it impinged upon a stricture, caused by the tumour pressing upon the walls of the duct. The gall-bladder contained about 50 c.c. of bile and three gall-stones. The liver was normal in size, but its ducts were all dilated and its tissues deeply stained by the pent-up bile. The kidneys and spleen were healthy. Immediately behind, and closely adhering to the duodenum and vena cava, was a tumour the size of the fist. It implicated the duodenal orifices of the bile and pancreatic ducts. The tumour was so adherent to, and so embodied in the head of the pancreas that it was impossible to distinguish between its limits and that of the proper pancreatic tissue. It was found to be of a different constitution and consistence in different parts. One portion was white, hard, and resisting to the knife; another, of a yellowish colour, was soft, and almost gelatinous, the head of the pancreas being thus completely occupied by the tumour. Although the remainder of the gland appeared from the outside normal, its interior, on section, was found to contain a number of differently-sized cavities, all of them being filled with the same yellowish-white, cream-like fluid as that already spoken of as having flowed from the fistulous opening in the centre of the ulcer in the duodenum. At about 8 centimetres distant from the duodenal orifice the pancreatic duct terminated in a cavity considerably larger than the others, which, like them, was also filled with the same kind of creamy fluid.

Although this case no doubt terminated as one of true pancreatic diabetes, from its clinical history it seems highly probable that it actually began as one of diabetes from "excessive sugar formation" and only changed into one of pancreatic diabetes after the cancerous degeneration of the pancreas became sufficiently advanced to entirely annihilate the functions of the organ. According to experimental evidence, pancreatic diabetes is caused by "diminished sugar consumption," or "defective sugar assimilation" as it may be equally appropriately called.

CASES OF NORMAL CHLOROFORM ANÆSTHESIA WITH A NOTE OF AN ACCIDENT FROM ABNORMAL ANÆSTHESIA.

By SURGEON-MAJOR EDWARD LAWRIE,
Hyderabad.

THE following cases of normal chloroform anæsthesia, which will relieve the monotony of the weekly records of death during abnormal anæsthesia, illustrate the method of administration and of recording cases of chloroformisation described in Part IX of the Report of the Hyderabad Chloroform Commission:—

CASE I.—October 10th, 1891. Temperature of room 78.5° F. Healthy Mahomedan male, M. S. A., aged 60. Disease: Wen. Operation: Excision; chloroformed at the Afzulung Hospital at 9h. 10m. 50s. by student Abdul Rahman. Full anæsthesia at 9h. 13m. 15s. Operation finished at 9h. 16m. 30s.

Observations.

	H. M. S.	
A.	9 10 50.	Chloroform administered on cap. Blowing rapidly 44 times a minute.
B.	9 11 15.	Cap brought close to the face.
C.	9 11 25.	Regular respirations, 20 a minute.

	H. M. S.	
D.	9 11 55.	Moving left arm.
E.	9 12 15.	Noisy moaning expirations.
F.	9 12 20.	More chloroform; while it was being poured into the cap patient took two inspirations of pure air.
G.	9 12 30.	Prolonged moaning expirations. The chloroformist was warned that this kind of expiration might lead to gasping, and not to allow the patient to gasp in chloroform.
H.	9 12 45.	Suddenly held breath; chloroform cap entirely removed. The students were told to watch the patient, as he was sure to gasp. He lay quite still, only winking when the eye was touched, until 9h. 13m. 15s.
I.	9 13 15.	Tremendous gasp of pure air, followed by regular quiet breathing. Chloroform cap reapplied.
J.	9 13 25.	Stertorous breathing. Stop chloroform.
K.	9 13 35.	Cornea insensitive.

If this patient had been allowed to gasp in chloroform at 9h. 13m. 15s. he would have been overdosed. He was very nearly narcotised when he held his breath, but the gasp of pure air averted all risk of asphyxia and of over-narcosis. It was followed by regular breathing and normal inhalation, and normal anæsthesia was produced after three or four more whiffs of chloroform.

CASE II.—November 7th, 1891. Temperature of room 76.4° F. Healthy Mahomedan male, I., aged 26. Temperature in the rectum 99.0° F., pulse 64. Disease: acute abscess; operation: incision. Chloroformed at the Afzulung Hospital, at 8h. 47m. 20s., by student Santhapillay. Full anæsthesia at 8h. 49m. 58s.

Observations.

	H. M. S.	
A.	8 47 20.	Chloroform on cap. Regular deep breathing, 32 a minute.
B.	8 47 35.	Cap close to face; regular respirations, 28 a minute.
C.	8 48 15.	Chloroform stopped while the table was moved round; patient took two breaths of pure air.
D.	8 48 23.	Chloroform again; regular respirations, 24 a minute.
E.	8 48 40.	Regular deep respirations, 20 a minute.
F.	8 49 5.	Cornea insensitive; stop chloroform.

In this case full anæsthesia was produced in 1m. 45s. The case is interesting, because it shows how rapidly and safely anæsthesia may be produced if the respiration is perfectly normal. The patient took two full breaths of pure air at 8h. 48m. 15s., when the table was moved round, and this did not delay the anæsthesia. The chloroformist made no effort to anæsthetise the patient rapidly. The rapidity was due to the perfect regularity of the respirations.

CASE III.—November 7th, 1891. Temperature of room 76.4° F. Very weak, nervous, native Christian, P., emaciated and exhausted from dysentery, aged 32. Temperature in the rectum 100.8°; pulse 136. Disease: Abscess of the liver. Operation: Aspiration of 28 ounces of pus. Chloroformed at the Afzulung Hospital at 9h. 5m. 35s. by Santhapillay. Anæsthesia maintained until the operation was finished at 9h. 19m. 15s. Vomited food at 9h. 15m. 10s.

Observations.

	H. M. S.	
A.	9 5 35.	Chloroform on cap. Blowing 36 times a minute.
B.	9 5 55.	Cap close to face.
C.	9 6 10.	Moaning.
D.	9 6 45.	Calling out loudly.
E.	9 6 55.	Struggling; one breath of pure air; stopped struggling.
F.	9 7 5.	More chloroform on cap; patient took three breaths of pure air.
G.	9 7 15.	Jerky expiration.
H.	9 7 30.	Cornea insensitive. Stop chloroform.
I.	9 7 40.	Snoring.
J.	9 7 55.	Stertorous breathing. Jaw pushed forward; respiration at once normal.
K.	9 15 10.	Vomited food. As always happens, with or without chloroform, the vomiting was preceded by pallor and weakness of the pulse.

This case forms a striking contrast to Case II. The anæsthesia was almost equally rapid, but in Case II the rapidity was due to regular breathing; in Case III to sheer weakness. In Case III it was extremely difficult to keep the breathing regular, and the slight and almost unavoidable irregularity in the inhalation caused overcharging of the air in the lungs to the extent that when the administration was discontinued at 9h. 7m. 30s. the anæsthesia went on deepening for twenty-five seconds afterwards. The explanation of what happens in these cases is to be found in Observation D of Experiment No. 64, on page 55 of the Hyderabad Chloroform Commission's Report.

CASE IV.—November 13th, 1891. Weakly pallid Hindu child, C., aged 18 months. Disease: Calculus in the urethra. Operation: Excision. Chloroformed at the Afzulung Hospital by student Ismail Khan at 8h. 54m. 45s. Full anæsthesia at 8h. 55m. 45s. The respiration became stertorous, then shallow, then stopped altogether, and no heart sounds could be heard for some time between 8h. 55m. 45s. and 9h. 15m. 25s. The counterpart of this case is to be found in Experiment No. 178, page 134 of the Report of the Hyderabad Chloroform Commission.

Observations.

	H. M. S.	
A.	8 54 45.	Chloroform on cap; crying.
B.	8 55 25.	Regular breathing—cap close.

FIG. I.—Case of pancreatic diabetes in a medical student aged 23. Fatal in eleven days. Pancreas closely adherent to duodenum, and converted into an irregular-shaped mass towards its head, while the body and tail portions were changed into a more flat and thin fibrous band.

FIG. III.—Microscopical section from head of pancreas in case of acute pancreatic diabetes. (A.) Atrophied lobules. (B.) Abscesses filled with leucocytes. (C.) Hypertrophied interlobular connective tissue.

FIG. II.—Section through the head of the pancreas, showing irregular-shaped abscess cavities filled with greenish-yellow purulent matter.

FIG. IV.—Microscopical section of head of pancreas in case of acute pancreatic diabetes, showing distribution of connective (A) tissue, and remnants (B) of glandular substance, after dissolving away all the fatty matters.