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.

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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.

H. M. S. Cornea insensitive. Stop chloroform; continued afterwards until 9h. 15m. 25s. The notebook was now inadvertently handed over to a student who was not accustomed to take notes, the regular note-taker being absent, and no further entry was made till the conclusion of the operation.

After the operation was commenced, and more chloroform had been given, the child struggled. The chloroformist was told peremptorily to keep the patient properly under until the operation was finished. He said the patient was "under," but the cornea was sensitive, and continued the administration after the respiration had become markedly stertorous. He was warned when the breathing was becoming stertorous, but unfortunately I did not interfere further. An attempt was first made to dislodge the stone from the urethra without cutting. This was troublesome, and my attention was wholly occupied for a few seconds, until suddenly I became intuitively aware that the child's breathing was shallow. The jaw having been pushed forward without benefit, the tongue was instantly pulled out with catch forceps. This had no effect whatever, and I then realised that the child had taken in a dangerous overdose of chloroform. Gradually, in spite of vigorous efforts to produce artificial respiration, the breathing ceased. Up to the cessation of the chloroform the assistant house-surgeon could feel the pulse at the groin, but not at the wrist. Regular systematic attempts to carry on artificial respiration, with the tongue well drawn out, entirely failed to get any air into or out of the chest, and death appeared to be inevitable. Seeing that artificial respiration was ineffectual, I applied my mouth to the child's and inflated the lungs two or three times, the air passing out through the nostrils. After the last inflation I could not detect the faintest heart sounds; no pulse could be felt, and the child was considered by everybody to be quite dead. I had turned away, and was washing my face, when the child began to gasp feebly. Artificial respiration was forthwith resumed, and, to our intense relief, was now effectual. At first I could hardly believe that the gasps were not caused by *post-mortem* contractions of the diaphragm, like those reported in the Commission's Experiments Nos. 80, 92, and 103; but artificial respiration was kept up, and natural respiration was eventually restored. The operation was afterwards concluded under chloroform administered by the same student, and the child was put to bed at 9h. 15m. 25s.

No notes were kept of the times at which the above events occurred. There was embarrassment but no irregularity of the breathing before it stopped, and the case was one of uncomplicated overdosing. The surgeon became impatient with the chloroformist, and the chloroformist weakly broke the rule and pushed the chloroform knowing that the breathing was already stertorous. The rule is to be found on page 287 of the Chloroform Report, and is as follows: "(5) Full anæsthesia is estimated by insensibility of the cornea. It is also indicated by stertorous breathing, or by complete relaxation of the muscles. Directly the cornea becomes insensitive or the breathing becomes stertorous the inhalation should be stopped. The breathing may become stertorous while the cornea is still sensitive; the rule to stop the inhalation should notwithstanding be rigidly enforced."²

The most important question with regard to the above case is, What saved the child's life? The heart can only stop under chloroform from arrest of its nutrition or from reflex inhibition. If it stops from the former cause recovery is impossible. If it is stopped by reflex inhibition it is a safeguard against poisoning. This point is proved again and again in the Hyderabad Commission's Experiments, especially well in No. 178, Pick reading (8 and 13). In manometer experiment No. 178, chloroform was administered at 2h. 29m. 45s. Respiration became very slow and shallow at 2h. 32m. 10s., and stopped at 2h. 34m. 30s. This was immediately followed by cessation of the action of the heart from 2h. 34m. 30s. to 2h. 35m. 30s. Ample pulsations returned at 2h. 35m. 30s. (*vide* Fig. 1, 8). Artificial respiration was not resorted to until 2h. 36m. 25s., and there is no doubt the animal would have revived without it. This is almost precisely what occurred in the clinical case under reference. The child was overdosed, and its breathing slowly stopped, and there was then a long cessation of the action of the heart. Inflation of the lungs was resorted to, but it was not continued long enough to warrant the belief that it helped much to revive the patient. In point of fact the inflation was abandoned, and the child was thought by all present, including two visitors, to be dead. But the pulse returned, and the respiration recommenced, spontaneously, before any further treatment was adopted. It is fair to infer, therefore, that one of the natural safeguards³ which surround the abnormal administra-

tion of chloroform, namely, reflex stoppage of the heart, was the principal factor in the recovery of the patient.

Anyone who is interested in this case will find it worth while to study the tracings of the experiments of the Hyderabad Commission which bear upon it.⁴ In Experiment No. 186 (Ludwig tracing III) there is a stoppage of the heart

Fig. 1.—Part of Pick tracing referred to in text (No. 178). The upper tracing shows respiratory, the lower cardiac, movements. 8. Heart and respiration stopped. 9. Heart going again. 10. Respiration also. at 2h. 56m. 10s., which is identical with the stoppage of the heart, above referred to, in Experiment No. 178 (Ludwig tracing I), at 2h. 34m. 30s. In No. 186 the arrest was caused

Fig. 2.—Part of Ludwig tracing (No. 178). a Chloroform. b Cornea insensitive. c More chloroform. d Respiration shallow, irregular. e Respiration quick. f Respiration and heart stopped. g Chloroform stopped. First heart-beat. Between 8 and 9 Heart touched with needle. 9. Near first heart-beats. h Artificial respiration commenced. i Artificial respiration stopped.

by electrical stimulation of the vagus, and it saved the animal's life. In No. 178 the stoppage was due to stimulation of the vagus, probably by asphyxial blood when the respiration stopped from overdosing, and a careful examination of the tracing shows that the stoppage of the heart likewise saved this animal's life. I have no doubt that exactly what happened in Experiment No. 178 as a natural safeguard, and in Experiment No. 186 as a deliberate remedial measure against chloroform poisoning, took place as a natural safeguard in the child and saved its life also.⁵

interfered with; (b) prevents chloroform from getting into the lungs—example, holding the breath; (c) prevents chloroform from getting to the nerve centres, due to vagus stimulation.

⁴ Photographs of all or any of the tracings of the experiments, at the cost of one shilling each, can be obtained from Lala Deen Dayal, photographer, Secunderabad (Deccan).

⁵ The actual value of the inflation of the chest was probably to blow out or eliminate some of the residual chloroform from the lungs through the nose while the heart was stopped, and so prevent further poisoning when the heart began to act again.

¹ The majority of the clinical class stated next day that I inflated the lungs altogether five times.

² I need hardly state that if the child had died I should have taken the whole blame on myself.—E. L.

³ The three natural safeguards are (a) lowering of the blood pressure. (b) stoppage of the respiration, (c) stoppage of the heart. (a) Saves and protects the heart, it may proceed to excess if the heart's nutrition is