whether the series given has been a specially favoured one, and whether cases may occur of a still more severe type, upon which no impression can be made by medical treatment. Upon all these points, as well as upon the question as to the method and details of the treatment, this paper aims at eliciting valuable information which may be available, though not yet recorded. It may also be of some use in pointing out that it is desirable, in every case proposed for operation, to inquire closely, not only whether medical treatment has been applied, but in what form.

REPORT OF SOME CASES OF STOPPAGE OF RESPIRATION DURING CHLOROFORM ANÆSTHESIA TREATED BY TRACHEOTOMY.

WITH THE REPORT OF ONE FATAL CASE OF CHLOROFORM ANÆSTHESIA.

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CASES OF ARRESTED RESPIRATION: RECOVERY.

THE frequent occurrence of recorded cases of death during chloroform anæsthesia has led me to submit these short notes of the following cases, which occurred in the Victoria Infirmary, Glasgow. They may be of interest as indicating a method of treatment on which greater stress might be laid by those who are responsible for the instruction of students of surgery. In the cases here recorded there is no need of discussing whether the respiration or the heart failed first, as in all it was noted that the heart continued to beat during the stage of apnoea.

None of the patients had undergone the usual preparation for chloroform, with the exception of Case i.

CASE I.—J. H., aged 44, was admitted in September, 1895, with necrosis of the femur. The heart and lungs were normal. Chloroform was administered by Mr. W. E. Macharg, M.B., C.M., House Surgeon. Violent struggling occurred during the first stage. This subsided as anæsthesia progressed. The pupils were well contracted, and anæsthesia otherwise seemed complete. Without warning sudden stoppage of the respiration took place shortly after the operation was begun. Artificial respiration, which was at once started, did not cause either air to leave or enter the chest. Tracheotomy was immediately performed by Dr. Parry, Visiting Surgeon, and then—but not before—did artificial respiration have the desired effect of initiating normal breathing. The administration of chloroform was resumed, and anæsthesia continued without a hitch till the end of the operation.

CASE II.—N. T., aged 32, was admitted in November, 1895, with mangled fingers—the result of an accident. The heart and lungs were normal. Chloroform was administered by Dr. Primrose, House Physician. Violent struggling took place, accompanied by voluntary apnoea. The struggling and straining subsided shortly, but the breathing remained laboured and obstructed, though the patient was well under the anæsthetic. One finger had been removed, and while another was being amputated respiration suddenly ceased. This took place about ten minutes after the inhalation of chloroform had begun. Three or four heaving movements of the chest and abdomen, simulating the ordinary rhythmical movements of respiration, were noticed, but no air either entered or left the chest. The jaw was raised well up, the tongue pulled out, and the epiglottis hooked forward with the point of the finger, but still breathing could not be made out. Artificial respiration was then begun, but proved useless, as in Case i. The patient meantime had become profoundly cyanosed. The pupils did not dilate. I hastily performed tracheotomy, and was greatly relieved to hear air whistle into the lungs through the opening in the trachea, which was kept patent, at first by the blade of the knife placed transversely. Chloroform was continued through the tube till the conclusion of the operation without further trouble.

CASE III.—R. T., aged 17, was admitted in November, 1895, with crushed fingers. The heart and lungs were normal. Chloroform was administered by Mr. A. R. Anderson, M.B.,

C.M., House Surgeon. Violent struggling occurred during the initial stage, soon followed by quiet and regular respiration with complete anæsthesia and well-contracted pupils. Twenty minutes later, towards the end of the operation, some heaving movements of the chest and abdomen were observed, and were considered to indicate the onset of sickness. Nothing, however, was vomited, and at the same time it was noted that the respiration was not quite so satisfactory. The throat was cleared, and respiration again became free and regular for some little time. Then the same violent, jerky movements of the chest and abdomen were again observed. These may have been due to sickness, but probably they were movements of inspiration. The tongue was drawn out, and the head allowed to hang over the end of the operating table. Artificial respiration was started. Breathing apparently was resumed, but only momentarily. It then ceased completely, and—as in the former cases—artificial respiration proved useless. Tracheotomy was immediately performed by Dr. Parry. This was at once followed by a long inspiration, and by the continuance of natural respiration till the end of the operation. On returning consciousness the patient vomited a few lumps of semidigested apple, and if the spasmodic movements referred to were due to retching, then a piece of apple blocking the trachea might have given rise to the symptoms. There was, however, no cough either then or later on.

THE CAUSES OF ARREST OF RESPIRATION.

In all the foregoing cases the heart sounds were normal. None of the patients were anæmic or debilitated, and indeed in Cases ii and iii they were quite the reverse. They were all males. Duncan, Flockhart and Co.'s pure chloroform was employed in all cases. Struggling during the first stage was a marked feature in all, and was accompanied by voluntary apnoea. The failure of respiration in all took place within the first twenty minutes of anæsthetising. It was noted in all that the thoracic and abdominal muscles continued to act after respiration had ceased, thus demonstrating the well-known fact that movement *per se* of chest or abdomen, or both, is no reliable or positive indication of true respiration. In none of the cases could the failure of respiration be due to a central cause, as the appearance of the patient and the course of the symptoms were undoubtedly due to peripheral obstruction, probably laryngeal, as traction on the tongue and epiglottis had no beneficial effect whatever. The dangerous condition is due to the straining, and begins before anæsthesia is complete.

The late Sir G. H. B. MacLeod used always to teach "that to push the administration of chloroform during this phase of its action was a very dangerous procedure."

This obstruction to breathing may become complete after the lapse of a shorter or longer time, but long before the respiratory centre could possibly be depressed. The obstruction may be due to one or more of the following causes:

1. Tenacious secretion in the larynx or trachea.
2. Spasm of the chords.
3. Oedema of the larynx.
4. Vomited matter.
5. A combination of two or more of the above conditions.

1. *Secretion*.—It is the rule, rather than the exception, for the chloroformist to be greatly annoyed by the increased secretion, but this *per se* would not cause such obstinate obstruction, as in the cases referred to, pressure on the chest, as a preliminary act in artificial respiration, did not cause air to be expired.

2. *Spasm*.—This might be due to (1) a reflex cause at a stage when the patient is insufficiently anæsthetised, a condition which did not exist in the above cases; or (2) to a spasm at first voluntarily maintained, but soon passing into a species of involuntary cramp. This is a possible explanation.

3. *Oedema of the Larynx*.—As is well known, oedema of this organ is readily induced, and often suddenly becomes extreme, thereby producing complete laryngeal stenosis. Is it not possible that the prolonged straining may give rise to this condition, a condition which might be called traumatic oedema? This oedema would explain the persistency of the obstruction, which persistency was present notably in Case ii, and was almost demonstrated by the fact that when the tracheotomy tube was withdrawn during the operation,

asphyxia at once recurred. The reinsertion of the tube immediately relieved the condition.

4. *Vomited Matter*.—Food particles are often made responsible for some cases of obstructed respiration, especially when the respiratory difficulty follows movements which have been considered to indicate sickness. Such movements are not, however, due to sickness in many instances, but to the obstruction in the respiratory passage.

5. *The Obstruction may be due to Two or More of the above Causes combined*.—This is probably the true explanation. For example, tenacious secretion in the larynx or trachea, accompanied by spasm or oedema of the chords, would effectually block the air passages for some time. These different causes would enter into the condition in varying degrees in the different cases.

A CASE OF FATAL ARREST OF RESPIRATION.

The report of the following fatal case of chloroform anaesthesia, which has not, to my knowledge, been published hitherto, seems to suggest that more prominence should be given to the treatment of accidents of this kind, by tracheotomy, than has been done formerly.

J.H., male in middle life, was admitted to the Western Infirmary, Glasgow, with a fractured upper arm. Being then a house physician in the Western Infirmary, it happened to be my lot to administer chloroform. Violent struggling of an extreme type took place during the initial stage, accompanied by such voluntary apnoea as to be alarming. The chloroform mask was removed till breathing became freer, though it still continued laboured. As the patient was "coming out" the inhalation was proceeded with. The pupils became somewhat contracted, and breathing regular though still embarrassed.

Suddenly stoppage of the respiration took place. The pupils remained as before and did not dilate at that stage, showing that tonic contraction was still present.

Artificial respiration did not answer, and air neither entered nor left the chest till death had undoubtedly taken place. Had tracheotomy been performed when it was manifest that artificial respiration was useless instead of persisting with it, in all probability a totally different result would have been secured. This case was that of a middle-aged, very robust man, apparently in perfect health. His heart was not sound, and at the *post-mortem* examination old pericarditis with adhesions and hypertrophy of the heart, with mitral disease, were found. The heart condition might account for the comparative suddenness of the death.

The efforts to breathe, which were noted in this, as in the former cases, with an obstructed windpipe, would put extra strain on the circulatory system within the chest. The positive or negative Valsalva's experiment gives rise to most alarming symptoms and signs, as any one who has tried the experiment knows. So in this case the extra strain thrown on a damaged heart would cause death to be more rapid than it would otherwise have been.

In the cases in which tracheotomy was performed no obvious harm accrued, and I believe the procedure should be used in desperate cases of this sort without hesitation.

CASE OF SNAKE BITE TREATED BY CALMETTE'S ANTIVENENE SERUM: RECOVERY.

BY SURGEON-MAJOR S. J. RENNIE, A.M.S.,
Meerut, India.

THE following is of interest in being one of the first cases of snake bite treated in India with Professor Calmette's antivenene serum.

About 6.30 P.M., on September 21st, a Hindu boy, aged 11, son of a groom, was drawing water from a well, and in returning accidentally stepped on a snake, which bit him on the right foot, the foot being bare at the time. Two men were with him who both saw the snake, but were unable to kill it before it disappeared in the grass. They promptly bound the end of a pugaree tightly round the boy's leg, and, picking him up, ran with him to my quarters. Not more than three minutes elapsed from the time he was bitten until I saw him.

The typical imprint of a snake bite, with its two deep fang

punctures and the crescentic row of small teeth marks between, was clearly seen on the inner side of the right foot. It being "the hour at which men most do congregate at the club," no fewer than five medical officers were on the spot in a few moments. I at once injected 8 c.cm. of Calmette's antivenene serum into the subcutaneous cellular tissue of his abdomen. At the same time Surgeon-Major Birt, A.M.S., treated the wounds and their immediate neighbourhood with a hypodermic solution of permanganate of potash, after which they were carefully washed and dressed. The case was then placed under observation and seen from time to time during the evening, but the patient never had a bad symptom, and is now running about as well as ever he was.

REMARKS.—There is no doubt one weak point in the above case—namely, that the snake was not killed, and that, therefore, there might be an element of doubt as to the nature of its species. The reptile, however, was clearly seen by both men who were with the boy, who gave an accurate description of it, and recognised it as a krait (*Bungarus caeruleus*), that most deadly and dangerous Indian snake. The characteristics also of the wounds were clearly those of a bite from a snake with fangs. My own personal observation led me at once unhesitatingly to conclude that the injuries were caused by a poisonous snake, and in this I was borne out by the unanimous opinion of the five medical officers by whom the case was seen, several of them of long and varied experience in India. Taking all these points into consideration there can, I think, be little doubt that the boy was bitten, and bitten savagely and deeply, by a krait, a bite from which under ordinary circumstances is necessarily fatal.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

MALARIA AT ADEN.

WITH reference to the memorandum of Surgeon-Captain Jones, I.M.S., in the BRITISH MEDICAL JOURNAL of October 31st, giving a table which shows the extraordinary and progressive increase in the number of cases of malarial fevers amongst the civil population of Aden since the year 1885, I wish to point out that a corresponding increase took place at the same time in the number of admissions for these diseases amongst the European troops quartered there.¹

I was in charge of the Station Hospital, Aden, from October, 1892, to October, 1894, and from previous reports and inquiries I found the prevalence of malaria attributed to 4 causes:

1. Increased rainfall.
2. Disturbance of ground in making forts at Steamer Point.
3. Dredging of the harbour.
4. The establishment of salt works on the isthmus.

1. As regards rainfall, on reference to Surgeon-Captain Jones's table it will be seen that in 1885 the fall was 12 inches, in 1886 it was 3 inches, while in 1890 it was 12 inches and in 1891 4 inches. Therefore the years 1890-91 were as regards rainfall counterparts of the years 1885-86, but the last-named years were not characterised by any increase in the amount of malarial fever, and this was the case also as regards European troops.² Why, therefore, should a certain rainfall cause malaria in 1890-91, when the same rainfall did not do so in 1885-86?

2. The disturbance of soil took place at Steamer Point. The station of Aden consists of three separate "positions":

- (a) Steamer Point, situated on the harbour.
- (b) The Isthmus Position, separated from Steamer Point by a large plain and by a range of precipitous hills, which also separate it from the harbour.
- (c) The Crater Position, separated from both Steamer Point and the isthmus by hills.

Now, of these three positions, the isthmus was by far the most unhealthy, and had to be abandoned by troops in 1892 on this account; the latter came next as regards unhealthiness; and Steamer Point, where all the fortifications were made, was the least unhealthy.

¹ *Army Medical Reports*, 1892, p. 143.

² *Ibid.*