belladonna extract was given every hour; and, after nine grains had been taken, copious alvine evacuation ensued. In two attacks since, the latest being last month, eight grains and nine grains respectively were effectual.

No alcohol was prescribed in any of the above cases, but patient No. 1 took about two ounces of port wine before the belladonna treatment was begun. In the after-treatment, and as a preventive of obstruction, the most effectual remedy I have found has been pulvis glycyrrhizæ composita. The belladonna treatment was suggested to me by the perusal of a paper by Mr. Macrae of Gateshead-on-Tyne in January 1873.

ON LATENCY OF SYMPTOMS IN ACUTE ABDOMINAL AFFECTIONS.*

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IN a discussion on the diagnosis and treatment of the causes of intestinal obstruction, it has occurred to me that a paper will not be out of place which gives the results of an experience that perhaps has been somewhat exceptional: an experience in which it has been the rule in acute abdominal affections for the symptoms to be more or less obscure and latent, so that the condition present has not been marked by those features which are usually described as its characteristics. And, as in several of the most striking instances the chief clinical fact has been obstinate constipation, the importance of such cases has been impressed upon me as influencing the diagnosis, and therefore the treatment, of cases of intestinal obstruction.

A brief outline of several instances will best support my statement.

A woman aged 47 was admitted into the Bristol General Hospital, with a history of ten days' complete obstruction of the bowels. There had been three similar attacks during the preceding five years. There was a total absence of all acute symptoms, both local and general. There was no anxiety or pain; the pulse was good; the temperature normal; the skin warm and moist; the tongue perfectly clean; the abdomen was much distended, but entirely free from pain and tenderness; there was no vomiting. Two days later, without any previous change, symptoms of perforation set in suddenly, and the patient died. On *post mortem* examination, there was no mechanical obstruction; but intense peritonitis existed; over the cæcum and adjacent part of the colon, the peritoneum was mottled with dark ecchymoses and greyish-black gangrenous patches; and inside this portion of the gut were deep gangrenous ulcers with sloughy and blackly ecchymosed bases and edges.

In this case, the cause of the obstruction was thought to be probably fecal accumulation; it proved to be cessation of peristaltic action from intense enteritis. And yet there had been an entire absence of all acute symptoms up to the occurrence of perforation.

Again: last May, a young man aged 19 was admitted into the Bristol General Hospital. A week before, there had been vomiting and diarrhoea for two days, without any febrile symptoms; vomiting had continued, but diarrhoea had given place to complete constipation. The pulse was 80, large and soft; the temperature subnormal; the tongue almost clean; the abdomen moderately distended, almost free from pain, and but very slightly tender at the lower part on firm deep pressure. There was frequent bilious vomiting. Two days later, without the development of any further special symptoms, the patient sank rather rapidly and died. On *post mortem* examination, there were ulceration and perforation of the vermiform appendix, peritonitis, enteritis throughout the whole small intestine, and, from three inches above the valve, the ileum was plugged for four inches with a solid fibrinous cast. Here the only positive clinical evidence was the presence of constipation with persistent bilious vomiting. As the onset was acute, the absence of the causes of chronic obstruction was indicated; and, in the decision between those of acute obstruction, there was one point to which great importance was attached: the two days' diarrhoea which preceded the constipation; this was looked upon as strong evidence in favour of enteritis apart from any suddenly produced mechanical obstruction. This conclusion was adhered to in spite of the almost entire absence of those symptoms and signs usually described as characteristic of intense enteritis, for the reason that, in my experience, it had been the exception for such acute abdominal affections to resemble the descriptions ordinarily given of them.

Again, a patient was lately admitted suffering from a severe attack of acute dysentery. Up to the time of his death, there was but little pain, and the abdomen was soft and flaccid, and almost free from tenderness. And yet the *post mortem* examination showed that peritonitis existed; that perforation had taken place; and that the whole of the large intestine had been transformed into a mass of ragged sloughs, and was so rotten that, in removal from the body, it fell to pieces and became quite unrecognisable.

With regard to acute peritonitis, I cannot say that I have never seen a case typical according to the usual description of this condition; but I must say that such a case has been the exception; and that, in the majority of instances I have met with, there has been an absence, to a greater or less degree, of the so-called typical symptoms. In fact, my experience has compelled me to teach that, although the presence of the symptoms usually described as characteristic of acute peritonitis is most valuable, yet their absence, local or even general, does not by any means negative the existence of that disease.

The bearing of what I have said upon the question of diagnosis in intestinal obstruction is obvious, inasmuch as more or less complete cessation of the action of the bowels accompanies acute peritoneal inflammations and the graver forms of enteritis; and, if these conditions are not marked by their distinctive symptoms, there is great chance that the true nature of the condition may be overlooked, and the constipation put down to some one of the causes of chronic obstruction of the bowels.

And yet I think that this latency of symptoms is not without its positive value in diagnosis; for I believe that, where the obstruction is due to some suddenly induced mechanical cause relievable by timely operation, the acute symptoms are far more commonly pronounced; that in such a condition their absence is the exception; and that, therefore, an absence of urgent symptoms is so far evidence against the utility of surgical interference.

My experience has, therefore, led me to the following conclusions.

1. An entire absence of acute symptoms may coexist with an intense local inflammation; and therefore it is prudent, in all cases of intestinal obstruction, to adopt treatment consistent with this knowledge.

2. If, in a case of intestinal obstruction, urgent symptoms are in abeyance, this is so far in favour of the view that the obstruction is either non-acute, or, if acute, is not to be relieved by operation.

I have been induced to bring this paper before you, in order that I may learn how far my experience in acute abdominal affections has been consistent with that of members present who are far better qualified than myself to speak authoritatively upon this subject.

RESPIRATION OF ASCENDING AND DESCENDING RHYTHM.

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IN its typical form, this remarkable phenomenon consists of a respiratory cycle in which respiration, at first light and unfrequent, becomes, by an ascending scale, fuller, more forcible, frequent, and exaggerated, until marked dyspnoea is attained; and which gradually subsides, by a descending scale, to a condition of complete apnoea, that persists for a considerable period and completes the cycle; after which a fresh cycle begins by light and almost imperceptible respirations. Each cycle is naturally divided into the period of apnoea and the period of respiration or dyspnoea. First described by Cheyne, and nearly thirty years later by Stokes, it is often named after them; and to the Irish school we are indebted for much that is known on the subject. Yet it is possible, as believed by the late Dr. Warburton Begbie, that Hippocrates observed and recorded an instance of it in the case of "Philiscus, who dwelt near the wall", and died on the sixth day of the duration of an acute illness, in the course of which were marked nervous symptoms, and whose respiration throughout was "large and rare, like that of a person who required to be reminded to breathe".

The three recorded in this paper are the only cases in which I have detected typical respiration of ascending and descending rhythm (Cheyne-Stokes) among the insane. In the first, the general extreme atheroma of the arteries was the most striking feature observed at the necropsy; the atheromatous change, with the resulting rigidity, inelasticity, and tendency to dilatation, being most marked in the thoracic aorta, the left subclavian, and the coronary and cerebral arteries; whilst the principal cardiac alterations were dilatation and hypertrophy of the left ventricle. Associated with these changes was the chronic renal disease, and the Cheyne-Stokes respiration was not developed until

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after certain ocular, cerebral, and other symptoms had appeared, which are common in Bright's disease. The uræmic symptoms became aggravated during the time the Cheyne-Stokes respiration continued; and, during that time also, the vessels were relieved by hæmorrhage into the pulmonary tissue and into the bronchial tubes. In this case, the renal seemed to be but part of a general morbid change, and not the source and origin of the various alterations found. The impairment of the structural integrity of the heart and arteries appeared to be in advance of the impairment of renal excretion.

In the second case were pulmonary phthisis, a pale, dilated, flabby, and slightly enlarged heart, whose aortic and pulmonary valves were unduly thickened, coarse, and opaque, and whose mitral valve was atheromatous. Localised pleurisy and pneumonia affected the base of the right lung. The aorta was nearly healthy, and, though there was some fatty degeneration of the cardiac muscular tissue, it was far from extreme.

In the third case, that of a general paralytic, the peculiar respiration was subsequent to the first appearance of hypostatic inflammation of the lungs; and, moreover, occurred while the patient was prostrated by epileptiform convulsions, and was being supported by nutritive enemata; departed before the cessation of the fits, or of this mode of feeding; and did not return (or at least was not observed) during the remaining ten weeks of life. The necropsy showed gross organic changes in the brain and its membranes, and in the lungs; while the aorta was healthy, and the heart nearly so. Before detailing these cases, a few points will be briefly referred to.

1. *The Pathological Changes with which Cheyne-Stokes's Respiration is associated.*—Fatty degeneration of the heart, as well as other morbid change in it, was found in the earliest recorded cases of respiration of ascending and descending rhythm, and this symptom came to be regarded by some as pathognomonic of cardiac fatty degeneration. Cheyne simply recorded his case, in which, together with fatty degeneration, the heart was also very much enlarged, the left ventricle greatly dilated, and the aorta studded with steatomatous and earthy concretions; but Stokes, speaking of this peculiar respiration and of fatty heart, distinctly asserts that he has "never seen it except in examples of that disease". A number of cases, however, were soon brought forward (Drs. Seaton Reid, Head, and Little), in which there was no fatty degeneration of the heart, and attention was directed, in part, to the other cardiac and the valvular lesions, so frequently found when the peculiar respiration has existed; and certainly, upon examining a large number of *post mortem* records, the prominence of these lesions is obvious. The almost invariable rule is that some cardiac lesion coexists; it may be that the heart is greatly increased in size from dilatation, from hypertrophy, or from these conjoined; or that there is morbid alteration or incompetency of valves; or alterations in size of the openings; or impairment of the propelling power or effect of the heart, either from intrinsic changes in its tissue, or from extrinsic obstacles to the circulation. As a rule, several of these structural changes coexist. (Cheyne, Stokes, Reid, Head, Little, Laycock, Traube, Benson, Biot, Hayden, and recently (*Lancet*, 1877), Andrew, Broadbent, and Wharry).

Together with several of the cardiac changes just named, the aorta has most frequently been found more or less atheromatous, calcareous, inelastic, and perhaps dilated. So often has this been noticed, that Hayden has recently stated it as his opinion, that "the only lesion of structure with which rhythmical irregularity of breathing has been always found associated is atheromatous or calcareous change, with dilatation, of the arch of the aorta, involving loss of elasticity of its walls". But, in some cases of Cheyne-Stokes's respiration, no marked aortic atheroma or change is recorded in the necropsies, and two of the cases I detail below are examples of its absence. Yet is the combination of fatty heart and highly atheromatous aorta a frequent one in cases of Cheyne-Stokes's respiration; and, in the first case in this paper, there was very marked atheroma of the coronary and of the cerebral arteries. The same is recorded in other cases (Reid, Laycock), and in still others, the general description would lead one to infer the existence of this change in the cerebral or coronary arteries. In relation to this, it has occurred to me that possibly respiration of ascending and descending rhythm might depend, in Case 1, upon atheroma and inelasticity of the minute vessels of the medulla oblongata, and consequent disturbance of the rhythmical stimulation and the nutrition of that part; but the changes found by the microscope in the minute vessels of the medulla were somewhat similar in degree to those found in the cerebral convolutions generally.

The great variety of morbid states in which respiration of ascending and descending rhythm is said to have occurred, and its advent in cases, under the care of one observer, differing from each other so much as do the three given below, lead to the inference that neither atheroma, dila-

tion, and inelasticity of the aorta, nor yet of the cerebral and coronary arteries, is by any means invariably found with this rhythmical respiration. Besides occurring with the diseased conditions mentioned in the preceding paragraphs, this respiration of the ascending and descending rhythm is stated to have been observed in uræmic coma, cerebral diseases, tumour of the brain, cerebral hæmorrhage, extravasation around or pressure on the medulla oblongata, tubercular meningitis, and even in pericarditis, and in typhoid fever with enfeebled heart. (Traube, Th. von Dusch, Mader, Broadbent, and Andrew). In some of these, perhaps, the respiration was not of the true type, but, making all allowances for this, true respiration of ascending and descending rhythm is undoubtedly found in a considerable variety of pathological conditions.

In many cases, there is a *nervous complication*. Without detailing the numerous theories as to the pathology of this peculiar respiration, we may recall to mind how prominent a feature, in several of them, is the hypothetical "diminished excitability of the respiratory centre". (Walshe, Benson, Laycock, Traube, Filehne.) In fact, Laycock, denying that structural disease of the heart is necessarily present in this "recurrent brief apnoea", as he termed it, speaks of it as depending upon a neurosis of the vagus nerve—as a sentient palsy of the respiratory centre, or a paresis of the reflex sensibility of the mucous membrane of the lung; and he drew attention to the occurrence of the symptom at the advent of sleep in some cases, and to the insomnia it is liable to cause. Symptoms such as headache, mental obscurity, emotional facility, and delirium have been found with it. In several cases, it was associated with hemiplegia—in Dr. Benson's case, it followed hemiplegia immediately—and in others, various trophic lesions of nervous origin were observed. In not a few cases, the nervous system was suffering from the effects of uræmia when the peculiar respiration under consideration occurred. In some instances, indeed, it was but part of the process of dissolution dependent upon chronic Bright's disease; and was found linked with the morbid chain of diseased kidneys and heart, arterial atheroma, general tissue-degeneration, various hæmorrhages, dropsical effusion, albuminuria, uræmia, and uræmic coma. There is no doubt of this, notwithstanding that Hayden has denied that it is a symptom of cerebral disease or of the comatose state.

Pulmonary lesions are not infrequent coincidently with the rhythmical respiration, but probably are secondary affections, or occur as part of the general failure of vitality. Congestion, pneumonia, pulmonary apoplexy, pulmonary excavation, emphysema, and pleural effusions have been found.

2. *The State of the Pulse in Cheyne-Stokes's Respiration.*—Speaking generally, the pulse has often been found irregular, rapid, weak, or intermittent coincidently with Cheyne-Stokes's respiration. In certain cases at least, the tendency was for the pulse and heart's action to become more irregular in force and rhythm during the dyspnoeal period, and more steady during the pause. By some, the arterial tension is said to be less during the apnoeal period than during the dyspnoeal. (Traube, Biot.)

In Dr. Head's case, examined by Dr. Grimshaw, the period of apnoea seems to have produced an effect upon the sphygmographic tracing of aortic insufficiency, the opposite of that produced by it in M. C. Biot's case. In the former patient, "there existed permanent patency of the aortic valves, and he noticed that the tracing taken during the breathing period exhibited markedly the collapsing pulse of that lesion, while tracings made during the apnoeal pause showed no such character"; whereas, in the latter patient, the sphygmographic tracing of aortic insufficiency was more decided during the period of apnoea.

A slowness of the pulse during the period of respiration and dyspnoea, and an acceleration of it during the period of apnoea, was first recorded by Dr. Seaton Reid in 1859-60. His statement was confirmed in two of the cases published by Dr. James Little, and in the one by Dr. Benson. Recently, Biot joins himself to these observers, and cites Bernheim in support of that view. For example: in one case, Little found fifteen cardiac pulsations during an apnoeal period of ten seconds, and only six beats during ten seconds of the dyspnoeal period; and in another case were nineteen pulsations during an apnoeal period of ten seconds, and only ten pulsations during a portion of the dyspnoea of similar duration. In Biot's case, at different periods, were apnoea of eighteen seconds with thirty-six pulse-beats, and dyspnoea of forty-nine seconds, with only eighty-two pulsations (would be ninety-eight, if at same rate as during apnoea); again, in fifteen seconds of an apnoeal period were twenty-eight beats, and in fifteen seconds of the dyspnoeal period only twenty-four beats; again, during apnoea of eighteen seconds' duration were thirty-five beats, and during dyspnoea of fifty-five seconds' duration only ninety-three beats (would be one hundred and seven, if at the same rate); and again, during apnoea of thirty seconds' duration, there

were fifty-five beats, and during thirty seconds of the dyspnoea there were only forty-seven beats. In opposition to this experience, is that of Hayden. He distinctly states that he has not met with the *alternate rhythmic ascent* (and descent) of the pulse and respirations during the cycle, but, on the contrary, has observed "either an absolute uniformity of the pulse throughout the attack, or, as in one or two cases, a slight acceleration during the period of dyspnoea, and a gradual decline in that of descent till a minimum rate was reached on the accession of apnoea". Laycock's report is confused with reference to this point. In my own Case I, the slowing of the pulse during the period of dyspnoea, or its acceleration during that of apnoea, was noticed on some days, or at some hours on a given day; but, at other times, careful and repeated examination often failed to detect any change in its rate. Indeed, I now find that this was noticed to some degree by the first observer, Dr. Reid; for, in speaking of this peculiarity of the pulse, he says that "neither it nor the distress is to be found equally marked or intense on every day".

3. *Length and Relative Length of the Periods of Apnoea and of Respirations during the Cycle.*—These vary from day to day, and indeed, to a slight extent, almost from moment to moment, as may be seen by the excerpts from my notes in the cases at the end of this paper. The following examples of the relative length of the periods of apnoea and of respiration are selected from various writers.

	Period of Apnoea, in seconds.	Period of Respiration, in seconds.
I.	15	45
II.	24	26
III.	15	42
IV.	10-12	35
V.	10	25
VI.	17-18	42-3
VII.	18	49
VIII.	18	55
IX.	30	45
X.	40	35
XI.	15	15

4. *The Disappearance of the Respiration of Ascending and Descending Rhythm before Death in some cases.*—Lasting from a day or two to two months or more, in different cases, the respiration of ascending and descending rhythm is almost invariably found only during a short period preceding death; and, therefore, may well be deemed of evil omen. But in several cases, it has disappeared before death. In two of Little's cases, it ceased ten and fourteen days respectively before the fatal termination, and six days before it in Benson's case. In the third case reported at the end of this paper, it was but of temporary duration, and disappeared ten weeks before death; in the other cases, Nos. I and II, in which it was respectively of twelve and nine days' duration, it continued up to the time of death, becoming modified in the former on the last day of life. Dr. Little attempted to explain the cessation of the peculiar respiration before death, in his own cases, on the theory that pulmonary apoplexy in the one case, and pleural effusion in the other, had weighted the right heart, and thus restored in one way that adjustment between it and the left heart, upon the loss of which, he supposed the peculiar respiration to depend. But in Case I, below, there was no cessation of the Cheyne-Stokes's respiration when marked pulmonary apoplexy supervened. Hayden has not noticed the cessation of the peculiar respiration before death.

5. *Arrest of the Peculiar Respiratory Rhythm.*—In my first case, the peculiar respiratory rhythm was sometimes temporarily modified, or momentarily arrested, by a short fit of coughing; and again, by a change from the supine to the sitting position. In Benson's case, "it was only while the patient was allowed to remain in the semi-comatose state that this peculiar form of breathing was present. When she was roused up, the respiration became almost normal; but again it assumed the ascending and descending character when she was allowed to lapse into the semicomatose state." Rousing the patient, exciting his attention, or conversation with him, sometimes temporarily arrested or modified the peculiar rhythm in cases by Laycock and Biot; and in another case, by the former observer, the respiration was only irregular and interrupted during the waking hours, but assumed the Cheyne-Stokes's character when sleep came on.

Ziemssen and Biot noticed contraction of the pupils during the period of apnoea, relaxing during the period of respiration.

6. I think my first and third cases prove that the fully developed, typical, respiration of ascending and descending rhythm is reached by gradations through a respiration of increasingly abnormal rhythm, which, at first only rising and falling a little, becomes gradually more ascending and descending in its frequency, depth, and force, and this at first without any period of apnoea—the apnoeal pause appearing and increasing in duration as the (simple) rising and falling respiratory rhythm becomes more and more marked; also, that the typical respiration departs as it came—passing through a respiration of simply rising

and falling rhythm, without apnoeal pause, towards—and to—a respiration of normal character.

CASE I. *Monomania. Bright's Disease: Widely distributed Atheroma of the Arteries, including the Aorta: Cardiac Disease: Secondary Pulmonary Disease: Cheyne-Stokes's Respiration.*—J. F., of the 43rd Regiment, died April 1877, aged 52. He had been an asylum inmate for many years, and the subject of persistent delusions as to his possession of supernatural powers and the fulfilment of prophecy in his person. Formerly the subject also of paroxysmal excitement, he had latterly been quiet and industrious. For several years he suffered from general arterial atheroma, some cardiac hypertrophy, and Bright's disease. In March 1875, severe nocturnal dyspnoea was succeeded by pulmonary engorgement, with watery and mucous expectoration, and cedema of feet and legs. These passed away, and his health continued fair, under treatment, for nearly two years afterwards. The face was of a dingy pasty hue, of greasy appearance and feel, and on it were a few dilated cutaneous blood-vessels. He had hair tinged with grey; slight baldness; a dingy sclerotic, a pearly drop lying upon the lower eyelids; clear blue arcus senilis; tortuous and hard temporal and other arteries; a full hard pulse; and the heart's action strong, and thrusting against the chest-walls at the præcordia, with a pulsation towards the epigastrium also. At that time he passed plenty of urine. A year later (1876), the epigastric pulsation was marked: the apex-beat was felt below and to the left of its usual site; over the xiphoid cartilage was a systolic bellows-murmur; the second cardiac sound was accentuated over both the right third costal cartilage and the pulmonary valves; the arteries were rigid and tortuous, especially in the left upper extremity; the left subclavian also presenting some fusiform dilatation. Later on, with urine normal in amount, acid, of specific gravity 1017, there was albuminuria, which continued and increased. Dulness, drowsiness, and impairment of vision were obvious. In December 1876, there was dyspnoea with lividity, and at times orthopnoea. The heart's impulse was heaving, thudding, and its action occasionally intermittent, but without *bruit*. The arteries were as before. The urine was lessened in quantity. Bronchial *râles* and wheezing were heard over the right lung, and over its base behind were occasional crepitation, dulness on percussion, and increased vocal resonance. The same signs, less marked, were found over the left lung. But the pulmonary condition did not fully account for the distress in breathing: there was renal dyspnoea. The urine was, and for some time had been, highly albuminous. Temperature 98.7 deg. He improved under treatment, and was able to get out again. Later on, in March 1877, he again had similar pulmonary signs, with severe dyspnoea, and the sputum became slightly flecked with blood. The dyspnoea often increased to orthopnoea; the highly albuminous urine grew less in quantity; the expectoration became watery and mucous; and oftentimes he was heavy and drowsy. From this he again recovered sufficiently to leave his bed and move about on the finer days. The apex-beat of the heart was then indistinct, widely diffused, two inches or more below the left nipple and displaced towards the left side: there was still marked epigastric pulsation; the first sound was dull and heavy, the second loud and ringing, and there was no *bruit*. But on April 6th he again had to take to bed with a return of the lung signs, and for some days afterwards there were dyspnoea or orthopnoea, clammy sweats, cold and livid blue hands and feet, distended surface-veins, a greasy appearance and icteroid hue of the face, which was often covered with clammy sweats, and a muddy, bluish and yellow colour of the eyeballs. Only one pint of urine was passed in the twenty-four hours; the appetite was bad; the heart's action heaving and laboured; pulse 94; respiration 30 to 34. The urine continued of the same amount only, in spite of various treatment. On April 12th, Cheyne-Stokes's respiration was first noticed: the pulse was then softer and smaller than was usual to him; respiration 34; hands cold and purplish; eyeballs discoloured, as noted on the 6th; urine only one pint in twenty-four hours. He had had, and continued to take, aperients, salines, iodide of potassium; and also occasionally, and in small doses, spirit of ether, spirit of chloroform, brandy, carbonate of ammonia, and later, bromide of ammonium. Diet: milk, bread, farinaceous puddings, onions, and a little fresh meat.

On the 14th, the voice was low and husky; Cheyne-Stokes's respiration continued. Once for all, it may be said that on various occasions during the rest of the patient's life the respiration was carefully counted and timed, watch in hand, throughout a very large number of cycles. On this day, the (1) period of respiration consisted of from 30 to 32 respiratory acts, lasting in different cycles from 55 to 65 seconds, the respirations commencing very lightly and easily, and being, in fact, scarcely perceptible at first, increasing in loudness, depth, and rapidity, and again sinking gradually but somewhat more rapidly than they had risen. (2) The period of apnoea lasted in different cycles from 25 to

30 seconds. During the breathing period, the deeper respiratory acts were liable to bring on coughing, which interrupted the peculiar ascending respiratory rhythm. For a few moments, the assumption of the sitting instead of the supine position lessened the peculiarity of respiration; but the latter was soon re-established. There was a difference in the pulse-frequency during the two periods on this day. It was a little slower during the period of respiration, sinking, in different cycles, from the rate of 100 to 104 per minute during apnoea, to the rate of 90 to 96 per minute during the part of the cycle when respiration became dyspnoeal. The pulse was now softer and more compressible than was usual in his case. Moist *râles* were heard posteriorly, more marked, and with dulness, at the right base. The urine amounted to one pint. The bowels were freely moved.

April 16th. Cheyne-Stokes's respiration had continued since. First there were, in different cycles, respirations varying in number from 42 to 44 and lasting for 65 to 70 seconds, which alternated with periods of apnoea of 30 to 35 seconds' duration in different cycles. The urine amounted to a pint and a half. There were crepitation and increased dulness at the base of the right lung; the expectoration, which for some days had been thick, viscid, adherent, and mucous, was now intimately intermixed with a large amount of blood. With this hæmorrhage the arterial fulness diminished, and it remained comparatively low thereafter.

April 18th. The urine was a pint to a pint and a quarter in quantity, and pulse 84 to 93; Cheyne-Stokes's respiration had been persistent ever since the 12th. The bowels were open. The expectoration was as on the 16th. His voice was low and indistinct; he had cold bluish and livid hands, feet, ears, and tip of nose. His features were livid and of a muddy yellowish hue. Pulse, to-day, 84, rather small and soft, and of about the same frequency during the periods of apnoea and of dyspnoea.

April 21st. The same thick, viscid, bloody expectoration was brought up. The pulse continued to be rather small and irregular in frequency, 80-88. He took food better. There was a greenish yellow and muddy hue of the sclerotic, and a pearly drop in the conjunctiva and lying upon the lower eyelid. The urine was considerably increased in quantity, even to three pints in the twenty-four hours; and the bowels were freely moved. The hands and feet were livid and cold, and the latter cedematous. The restlessness had become intense, and the patient had been slightly delirious at times; nor had the bromide of ammonium had much effect upon these nervous symptoms. His voice was low, indistinct, and husky. The lung-signs were as on the 16th, with Cheyne-Stokes's respiration.

April 22nd. There was puffiness about the eyes; cedema of the feet and legs; very slight ascites; Cheyne-Stokes's respiration still continued. The pulse was 96 to 108, running up for fractions of a minute to the rate of two beats per second. He had thirst; there was purpuric ecchymosis on the arm and chest. He was excessively restless since, with much muscular jerking and subsultus. Three successive cycles showed the following increasing duration:

	Apnoea.		Dyspnoea.
1.	15 seconds.	.	50 seconds.
2.	20 "	.	53 "
3.	23 "	.	55 "

At midnight, with a soft and feeble pulse of 96, the typical Cheyne-Stokes's respiration was suspended for a time, and the respiration merely rose and fell in depth, loudness, and frequency; quiet respiration alternating with dyspnoea. Then the Cheyne-Stokes's characteristics were gradually resumed.

April 23rd. He had muttering delirium and moaning; his utterance was low and almost unintelligible; he was excessively restless. Respiration was as at last midnight, periods of more quiet respiration alternating with others of more noisy, frequent, and difficult respiration. Later on in the day, the breathing assumed more of the typical Cheyne-Stokes's character. Pulse varied from 72 to 78; temperature 100 deg.; urine still free. The hands were cold and blue, the tongue red and dryish. He had had great thirst for several days.

April 24th. The respiration varied in frequency and rhythm; it was laboured and noisy, but less so than during the height of the former dyspnoea; in frequency, it varied from 32 to 36 and 38 per minute; now and then there was a double inspiration, each component inspiratory act of which was short, quick, and shallow. Pulse 108 to 110. There was diarrhoea, and four pints and a half of urine had been passed in twenty-four hours; these had reduced the lividity of the hands and the cedema, including that of the conjunctiva. The expectoration was less, was thinner, but still bloody. Temperature 98.8 deg. The patient was restless, moaning, tossing about, and muttering unintelligibly; but he swallowed food. Fluid had collected in the right pleural cavity.

Until near the close of life, the patient had greater ease in the sitting than in any other position. He also complained of heat rather than of cold. He died on the evening of April 24th, the Cheyne-Stokes's respiration having persisted in a typical form from the 12th to the 23rd of April inclusive.

Abstract of Necropsy.—There were yellowish muddy discoloration of the body; purpuric stigmata and petechiæ on the upper extremities, and purpuric ecchymoses on the backs of the hands and on the left arm and chest. The calvarium was thick and heavy; there was but little blood in the diploë, or clot in the sinuses. All the arteries at the base of the brain were highly atheromatous, most of them standing widely open, with dilated, thick, and rigid walls, presenting on their inner surface opaque yellowish material, possessing the microscopical characters of atheroma. The vertebral arteries showed this change to a high degree; so also did the terminations of the internal carotids and the middle cerebral arteries, especially on the left side. In the remaining arteries, at or about the base, the change was less extreme; but it was found more or less in all, and slightly more in the arteries of the left than in those of the right side of the encephalon. The *right* inferior cerebellar artery was but slightly atheromatous; but in the *left* inferior cerebellar artery the change was well marked. After the vessels left the base, the atheroma occurred only in closely set or more scattered points and plates, which gradually became smaller and less numerous, but were found on the lateral surface of the brain almost to the summit, and again were met with on the internal surfaces. The dura mater was somewhat thickened; the soft membranes were pale, moderately opaque, rather thick and tough, and considerably cedematous; they stripped off readily from the convolutions, which were slightly wasted in the frontal and parietal regions. The lateral ventricles contained two drachms of fluid; their lining membrane was rather thickened and opaque. The lining membrane of the fourth ventricle was thick, semi-opaque, and rugous. The brain-substance was pale. The right hemisphere weighed 20½ ounces; the left, 20¼ ounces; the cerebellum, 4¼ ounces; the pons Varolii and medulla oblongata, ¾ ounce; the bloody serum from the brain, 3 fluid-ounces.

Heart.—The apex of the heart lay two inches and a quarter below and one inch external to the left nipple. The pericardial fluid was three ounces and a half in quantity. There was a milky patch on the front of the right ventricle, and slight fibroid thickening of the pericardium on the posterior surface, and points of subpericardial ecchymosis. The coronary arteries were both extremely atheromatous, standing widely open, feeling here and there like pieces of whipcord, and elsewhere feeling beady from atheromatous patches of yellowish colour, which at points were calcareous. The left ventricle alone was somewhat dilated and hypertrophied; the muscular tissue of its walls, like that of the rest of the heart, was very pale; and, under the microscope, the striæ were less distinct than usual, and there was very slight granulo-fatty degeneration. The mitral aperture was wide. All the four sets of valves, with the exception of the tricuspid, were somewhat thickened and more opaque than usual, and were of large size. The aorta and pulmonary artery seemed to be rather wide; and in the arch of the aorta there was considerable atheroma, seen in slightly raised, dull-yellowish, closely set patches; which atheroma also extended downwards and affected the entire thoracic aorta in scattered spots. The left subclavian was highly atheromatous, and was wider than the right. The heart, with the thoracic aorta, subclavian, etc., attached, weighed 25 ounces. The thickness of the wall of the left ventricle at its middle was five-sixths of an inch; of the wall of the right at the same part, one-fifth of an inch. The external length of the left ventricle was four inches and seven-eighths; of the right ventricle, three inches and seven-eighths. The internal width of the aortic arch and of the pulmonary artery was one inch and nine-sixteenths.

Lungs.—The right lung weighed 42 ounces; it was congested and cedematous; and there were large masses of pulmonary apoplexy, surrounded by irregular zones of inflamed pulmonary tissue, and found especially in the lower lobe. In front, there was some emphysema. The surface of the lung was studded with subpleural firm whitish plates of semicartilaginous consistence, each surrounded by a dark pigmented zone. At the apex were a few cretaceous subpleural nodules; elsewhere were a few soft subpleural granulations. The bronchi were congested and loaded with secretion. The left lung weighed 21¾ ounces. It was in a condition similar to that of the right lung, but with scarcely any pulmonary apoplexy, and with far less congestion. There were thirty ounces of fluid in the right pleural cavity, and ten in the left.

Abdomen.—There was some turbid serum in the cavity. The kidneys weighed 4¼ and 4½ ounces; they were very pale, of a yellowish and dull-whitish, somewhat homogeneous appearance, with a few

minute cysts on the surface. The cortices were slightly atrophied, and the convexities of both kidneys were coarsely granular. The right one had some irregular fibrous thickening of the capsule; the capsules separated in fragments, but were only a little adherent. There was no lardaceous change. The spleen was small and firm; it weighed 4 ounces. The liver weighed $5\frac{3}{4}$ ounces; it was slightly hobnailed on the upper surface, and very slightly cirrhotic on the lower surface. The prevailing hue of the sections was yellow. The liver was not lardaceous; and although, under the microscope, some of its cells were rather too fatty, there was no well-marked fatty disease of it.

It may be added that, under microscopical examination, both the cells and the minute vessels of several of the cerebral convolutions exhibited morbid changes. Of the minute vessels, some showed hyperplasia of the nuclei of their walls, others had irregular molecular deposits here and there in their walls, and others had a hyaline appearance. In the medulla oblongata, beneath the floor of the fourth ventricle, the walls of the minute vessels were irregularly thickened at points by finely molecular deposits in or upon them. The nerve-cells also had undergone some granular degeneration.

CASE II. Chronic Insanity: Phthisis Pulmonalis with Bronchitis: Cardiac Change: Cheyne-Stokes Respiration.—T. D., of the 8th Regiment, died in September 1876, at the age of 44. He was an old inmate, the subject of chronic mania with periods of mental depression, but latterly without active mental symptoms. At the age of 43, he became affected with latent pulmonary phthisis, and died about a year afterwards. During the last few months of life, he had a soft and rapid pulse, usually a little above 100 in the minute, with dyspnoea, and a bluish colour and coldness of the face and hands. It was only when bronchitis supervened that any cough or expectoration was noticeable. The heart-sounds were comparatively feeble, the impulse was only felt distinctly at the epigastrium, and the first sound was heard more clearly there than elsewhere. The patient rested on the right side. Generalised bronchitis came on four weeks before death. Towards the close of life, slight pneumonia and pleurisy attacked the base of the right lung; tuberculosis spread in other parts; emaciation progressed; the dyspnoea was considerable; the purplish lividity and chilliness of the face and hands increased; perspiration was free; the pulse continued slightly above 100; and the respiration varied from 32 to 37 per minute. Nine days before death, and thenceforward until that took place, the Cheyne-Stokes respiration was noticed. Respiration was laboured, mainly thoracic, rather loud, increasing in depth, loudness, and frequency in a crescendo movement; then gradually diminishing in these respects, until it ceased altogether for a period, after which the cycle was renewed. The only coincident abnormality made note of in the characters of the heart-sounds was their weakness.

Abstract of Necropsy.—The consistence of the brain was slightly diminished; the fornix was soft; the membranes were slightly oedematous. There was full vascularity of the brain-substance. The cerebrum weighed $42\frac{1}{4}$ ounces; the cerebellum, $5\frac{1}{4}$; the pons Varolii and medulla oblongata, 1 ounce. The *Heart* weighed $12\frac{3}{4}$ ounces; it was flabby, dilated; its apex was situated somewhat below and to the right of the usual site. The muscular substance of the heart, examined with the microscope, showed some granulo-fatty degeneration, and the transverse striæ had not their normal distinctness. All the cavities contained black clot, and by it the right cavities of the heart and the large veins emptying themselves into both auricles were gorged. The external fat covering the right side of the heart was only in moderate amount. The pulmonary semilunar valves were thickened and unduly opaque, and the corpus Arantii of one was greatly hypertrophied. The aortic semilunar valves were thickened and unduly opaque; the mitral valves were large and atheromatous; there was incipient atheroma in the arch of the aorta. *Lungs.* The right weighed $45\frac{1}{2}$ ounces; the left 33 ounces. On both lungs were old close pleuritic adhesions and some leathery bands. The caseous changes were widely spread in the left lung, and at points had led to small excavations; while there was some cirrhosis in the upper part. In the right lung were scattered tubercles, some of which were in process of softening; and at the base were recent red hepatisation and some flakes of recently effused lymph on the pleura. In the central part of this lung, a pink and white soft material with fibrous stroma showed the characters of ordinary tubercle under the microscope. In both lungs, the bronchi were markedly hyperæmic, were slightly dilated, and contained secretion. Of the kidneys, each weighed six ounces and was slightly granular at one extremity, the capsule being also slightly adherent. The spleen was small, firm; and the liver nutmeggy. There were seven gall-stones in the gall-bladder.

CASE III.—General Paralysis of the Insane: Successive Attacks of Left Hemiplegia: Apoplectic and Epileptiform Seizures, followed by temporary Right Hemiplegia and by Cheyne-Stokes's Respiration:

Hypostatic Pneumonia.—H. B., of the 4th Battalion, Rifle Brigade, died at the (stated) age of twenty-eight. This case of general paresis was of four years' total duration, and the physical signs were well marked. At first, the patient was elated and optimistic; later on, he was morose, querulous, foul-mouthed, and at times confused, void of expression, and stupid. Without entering into details, it may be said that, fourteen months before death, he had an attack of acute herpes zoster over the front of the left thigh, groin, and upper gluteal region; and that he had temporary attacks of left hemiplegia, without convulsions, on several occasions—eleven, eight, six, and four months before death. Ten weeks before death, he was flushed, feverish, and there was slight pneumonic mischief. Several days afterwards, an apoplectic seizure occurred, and in a few hours was followed by three severe epileptiform convulsions, beginning in the right upper extremity. Right hemiplegia succeeded these fits, which were treated by enemata of chloral-hydrate. On the next day, the right hemiplegia was much less; the face and ears were flushed; the right conjunctiva almost insensitive. There were also muco-purulent expectoration, scattered pneumonic consolidation, and numerous *râles*, especially over the right lung. Temperature 101.2 deg.; pulse 114 , full, soft; respiration 25 per minute, possessing the Cheyne-Stokes characters, though not quite in a perfect typical form. The period of apnoea occupied one-third of the cycle; then two-thirds of it were occupied by respirations which increased in depth, loudness, and frequency, and then diminished; but each period of respiration included, on the average, six respirations only. After a severe paroxysm of coughing, the rhythm was temporarily modified, and there was for the time a regular ascending and descending rhythm of greater and less frequency, depth, fulness, and loudness of respiration, without a period of true apnoea. He was now, and for five days in all, supported entirely by nutritive enemata, epileptiform seizures recurring on several occasions. The right hemiplegia cleared up; but the left hemiplegia returned, and was persistent and increasing from this time until death; while the left limbs became extended and rigid, and the right limbs had sudden and irregular increments of their weakness. Biebs also appeared on the right hand, and slight sores from pressure on the right foot.

After death, were found adhesions and other changes in the membranes and substance of the brain, common in general paralysis, but which, in this case, affected the right much more than the left hemisphere, and were differently distributed on the two sides. The brain and meninges were pale. Some induration of the white matter, and much slighter induration of grey matter, existed, neither at all universal, and both most marked in the right frontal lobe. The basal ganglia were much alike on the two sides, but were not plump. The right hemisphere weighed $23\frac{3}{4}$ ounces less than the left; the right weighed $15\frac{1}{2}$ ounces; the left, $17\frac{3}{4}$; the cerebellum, $5\frac{3}{4}$; the pons Varolii and medulla oblongata, $1\frac{1}{2}$ ounces. The spinal cord was rather soft and pale; its membranes were thickened, and beneath them were hæmorrhagic masses. Scattered hypostatic pneumonia was found in both lungs, the pulmonary tissue being also broken down here and there, especially at the right apex, which was excavated, but there was no tubercle. The heart weighed $9\frac{1}{4}$ ounces; it was healthy, save that its muscular tissue was rather dull in hue and slightly friable, and that there were a few small points of atheroma in the left coronary artery. The aorta seemed to be perfectly healthy, there being in it only a few small spots of atheroma just above the sinuses of Valsalva.

ON A CASE OF TYMPANIC ABSCESS HAVING AN UNUSUAL TERMINATION.

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THE case of tympanic abscess which I propose to narrate will, I trust, be deemed worthy of notice by the members of the Association, especially those in general practice, who, being familiar with the usual mode of termination of such cases—namely, perforation of the membrana tympani and otorrhœa—are likely to overlook such a rare contingency as presented itself in this instance.

On April 24th, 1878, a young gentleman aged 15 came under my care suffering from acute pain in the left ear. There was considerable constitutional disturbance, the temperature being 102 deg. and the pulse 120. The pain, which extended over the temple and left side of the head, was greatly aggravated during the night, and was attended

* This modification of the respiratory rhythm was also seen in another general paralytic under my care two days before death. He was then partially comatose, and had dextral hemiplegia, following right unilateral convulsions. Besides this, modified Cheyne-Stokes' respiration was seen in the fifth case, also a general paralytic.