On recent researches upon nitrous oxide narcosis : and their bearing upon the practical question, when and how should laughing gas be administered? : read at the Annual General Meeting of the British Dental Association at Brighton, August, 1889 / by Dudley Wilmot Buxton.

With the writer's compts

(4)

ON RECENT

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And their bearing upon the Practical Question— When and how should Laughing Gas be administered?

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On Recent Researches upon Nitrous Oxide Narcosis,

And their bearing upon the Practical Question—When and how should Laughing Gas be Administered?

The questions which practical men ask themselves in considering the value of an anæsthetic, are:

1. Is it efficient?
2. Does its use entail danger to the patient?

I take it we have severally been asked to read papers upon the various anæsthetic substances in order that some comparison may be made between these anæsthetics, and some conclusions formulated upon their individual uses and dangers.

I propose to investigate how far Nitrous Oxide Gas is an efficient anæsthetic, what the limits of its application are; and, secondly, to indicate that it is within these limits a safe anæsthetic.

The audience I address to-day will probably be prepared to accept my last contention, as their experience will have proved to them the extreme rarity of any untoward symptoms occurring under "Gas," but I venture to think that it has only recently been competent for us to explain in a satisfactory manner why nitrous oxide is so safe, and why it is so effectual. Among my medical colleagues, with the exception of those who have gained a special knowledge of the matter, there is still a curious dread of nitrous oxide, and time after time have I been told that Dr. So-and-So considered Miss Blank "not strong enough to take gas." This arises from the fact that the average medical mind regards nitrous oxide narcosis as a modified form of asphyxia, and is prone to communicate this idea to the patient, who very properly translates asphyxia as

being smothered or choked. A text book which we find in every student's hand describes, with charming assurance, the action of nitrous oxide as that of an asphyxiant, and recently a venerable and deeply-respected member of our profession, Dr. George Johnson, has promulgated similar views with the weight which his name must always lend to any statement. But nitrous oxide is no asphyxiant, it possesses a specific action upon the organism which differs widely from what obtains when an indifferent gas is respired, while oxygen is withheld.

In the course of a prolonged research, the results of which I had the honour of communicating to the Odontological Society of Great Britain, the following facts were pretty clearly made out—

Nitrous oxide does not, to any appreciable extent, split up in the organism; hence its action, if any, must depend upon itself, or upon deprivation of oxygen.

We find by actual experiments that nitrous oxide produces gross changes in the organism.

Thus if the skull of a mammal be trephined, and a sufficiently large window be made in the bone to expose an area of an inch or so across, one is able to watch the pulsation of the brain beneath the dura mater, to observe the colour of the membrane, &c. Under ordinary circumstances there exists, after trephining, a very distinct space between the bony calvarium and the dura mater, and rhythmic pulsations occur bearing a direct relation to the systole, or general arterial dilatation.

It is a simple matter, the muscular system being paralysed by curare, to establish artificial respiration by means of a bellows worked by a water power engine, to ensure a respiration of any rate and depth. Of course the normal rate of respiration during sleep is selected, and by the use of a three-way valve, air can be excluded, nitrous-oxide or other gas admitted, and at expiration the tidal air removed. With this apparatus the following results were arrived at:— The brain, as soon as nitrous oxide was admitted, began to swell up, and although preserving its normal colour for a time, the hemispheres assumed a most remarkable appearance. Simulating a hernia cerebri they protruded into the trephine hole. The colour of the brain now changed from

a bright vermilion to a lakey purple. The brain undulations changed in character, becoming less in frequency and amplitude. The volume of the brain increased and at length the movements ceased. The dura mater now was pearly and glistening with a bluish lustre, and upon examination of the vessels of the pia mater, the well-known appearance of commencing stasis was seen. Upon resumption of air and shutting off of nitrous oxide, the phenomena above described were repeated, but in a reverse order. The brain undulations increased in frequency and amplitude, the fulness of the hemispheres gradually lessened, and the membranes resumed their ordinary colour and appearance.

Although we are not concerned especially in this connection with the *modus operandi* of the anæsthesia production by nitrous oxide, I will here answer a suggestion which was made some time since, viz., that the brain substance—which as I had then shown swelled considerably—might press upon the calvaria or bony walls, and so a species of compression be established. I, at the time, pointed out that whatever pressure existed was probably interstitial and not found without, but I further tested the question by removing the bony coverings of the hemispheres, and in spite of this obtained the full physiological effects of nitrous oxide.

We will now pass on to consider the spinal cord. Pursuing a similar method, several laminæ were removed from the vertebral column and the spinal cord exposed; in some cases the theca being incised, in others being left in its entirety. Nitrous oxide was then administered and the following phenomena observed:—The spinal cord, just as occurred with the brain, underwent an increase in size, which increase was very prettily evidenced by the overflow of cerebro-spinal fluid.

The difference anatomically between the brain and cord made the former an easier organ upon which to study the changes in the vascular membranes, but no more striking proof of the enlargement of the whole cord could be obtained than that afforded by the outflow of cerebro-spinal fluid which took place as soon as the animal came under the influence of the nitrous oxide.

But to establish the truth of the results thus arrived at it is necessary to obtain them under other and test conditions.

Accordingly curare was not used in one series of experiments, while parallel series of experiments were devised in which mouth respiration in some, and tracheal inhalation in others, were employed. It also appeared necessary to grapple with two questions, viz. :—

1. Are the phenomena detailed above really due to nitrous oxide ? (2) Or to de-oxygenation of the tissues—apnœa—a term, as Dr. George Johnson suggests, more satisfactory than asphyxia ?

To deal with the second question, control apnœal experiments were made.

In one series the trachea was tied, while in the other a curarised animal was after a time left without artificially performed respiration, this being done to avoid the dyspnœal struggles, which by their very violence produce a rise in blood pressure and so give an illusory resemblance between the states of apnœa and nitrous oxide narcosis. For particulars of these experiments I would refer to my original papers, and will content myself here with stating conclusions.

Apnœa produces changes far more slowly than those occurring in nitrous oxide narcosis.

The brain and cord, although when much muscular struggling is permitted, undergoing some engorgement and becoming purple and almost black, do not swell up, but actually lessen in volume in apnœa.

The lessening in volume may, provided the apnœa has not been carried too far, be checked and even changed to a state of enlargement if nitrous oxide be administered.

Again, experiments (for details see papers quoted) show, that besides causing anatomical alteration in the spinal cord and encephalic centres, nitrous oxide produces physiological phenomena with which we are familiar in certain conditions of the cerebro-spinal axis, viz. : a loss of certain reflexes, namely, the superficial, such as the skin, and conjunctival, persistence of the patella reflex, sometimes its exaltation, development of ankle-clonus, the occasional development of clonic and tonic contractions, opisthotonos, emprosthotonos, pleurosthotonos, and occasional transient paresis and hemiparesis—all symptoms of extreme significance and interest. About the cardiac and vaso-motorial systems we have very important facts to consider.

Those regarding nitrous oxide as an asphyxiant have been accustomed to caution persons with weak hearts against its use, and indeed, if the term asphyxiant could be correctly applied to it, it would be most detrimental in nearly every form of heart and pulmonary disease.

But having shown that nitrous oxide acts *per se*, and *not* as an asphyxiant, we will give the effects it has upon the heart force and rhythm, and blood pressure.

There are several *ways of showing the heart's action:* placing the *hand upon the chest, removing the chest wall and watching the viscus* in its pericardium, taking *cardiographic tracings*, and the less satisfactory method of recording the pulse at the wrist or elsewhere, either by the use of a sphygmograph or simply trusting to the finger. The first methods are preferable, but any, if carried out carefully, show that the heart is but little affected by nitrous oxide.

If an animal is made to inhale until the respirations grow slower and slower and finally cease, the heart will be found to beat steadily on in marked contrast to its laboured tumultuous action during the condition of apnœa.

The sphygmograms which are shown and which are passed round show the following changes in the pulse.

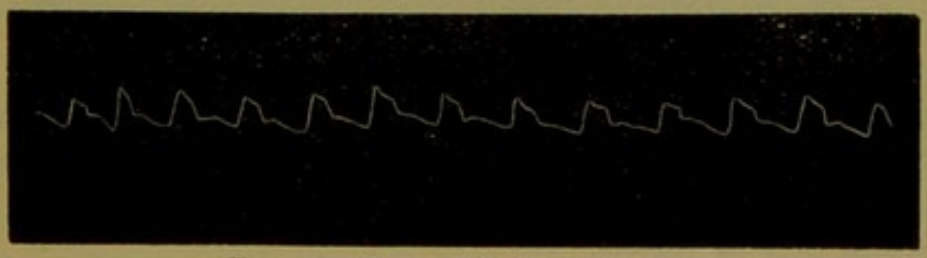


FIG. 1.—Normal Pulse. Case 1.

The normal pulse trace consists of the initial rise, as the tidal wave distends the artery, and the gradual descent as

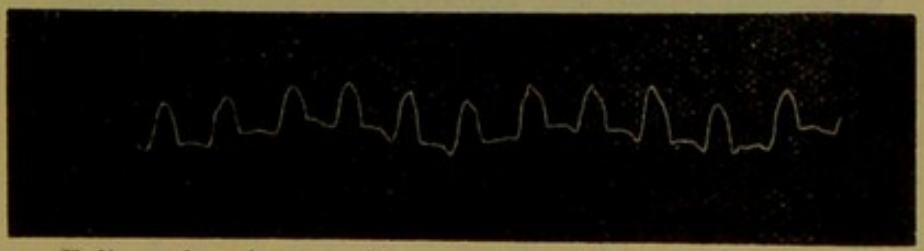


FIG. 2.—Fully under nitrous oxide ; shows no tidal wave ; marked dicrotism, evidencing lowering of arterial tension.

the tidal wave passes onwards, which descent is marked by secondary waves, these being due, partly to oscillation and

partly to reflux of blood driven back by the obstruction caused by the capillaries.

Under gas there appears to be a lessened tension evidencing a lessened tidal wave, this lessened tension being shown

FIG. 3.—Shows gradual resumption of normal characters during "coming to."

by the greater acuteness of the initial curve, the dicrotic wave being placed lower down the curve, and the dicrotism increased, while to the finger the lift is perceptibly diminished. These results are most important, and are at variance with

FIG. 4.—Continuation of 3; normal tidal wave re-appearing.

much that has been published elsewhere—material based upon the very unreliable foundation of experiments conducted upon hospital patients, all more or less under the tyranny of fear or terror.

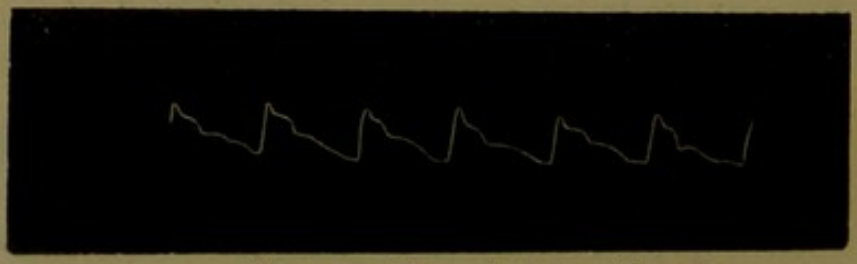


FIG. 5.—Normal trace. Case 2.

The rhythm of the heart, at first accelerated, usually returns to its normal rate during narcosis, or drops a few beats.

The blood pressure under nitrous oxide must next be considered.

For the first period but little change occurs; later on there is a gradual fall in blood pressure, a fall which, although occurring throughout the whole body, is most marked in the

FIG. 6.—Under—shows loss of tidal wave; pointing of apex; respiratory curves exaggerated; shows lowered tension.

splanchnic areas, as is evidenced by the kidney curve shown in the diagram (p. 11.)

Upon the animals breathing air again, a short gradual recovery of blood pressure takes place.

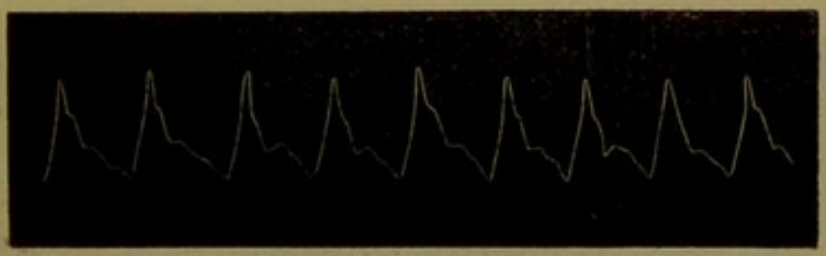


FIG. 7.—Still under, beginning "to come to."

Control experiments made upon curarised animals showed that when they were rendered apnœic, blood pressure at once rose and became extremely high, while the heart's beats became weaker and weaker *pari passu* with increased blood pressure.

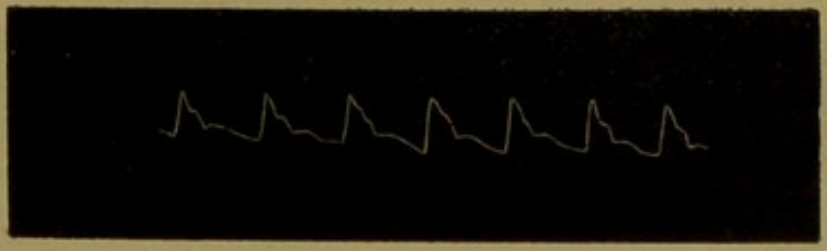


FIG. 8.—Resumption of normal beat.

Upon respiration nitrous oxide acts as follows:—

The respirations grow slower and at first fuller; as narcosis progresses they become still more retarded, but less full, and

at length they cease. At this point the heart still beats, and if a gentle pressure be made upon the thoracic parietes respirations are resumed, and provided access of air be allowed to continue, consciousness is regained.

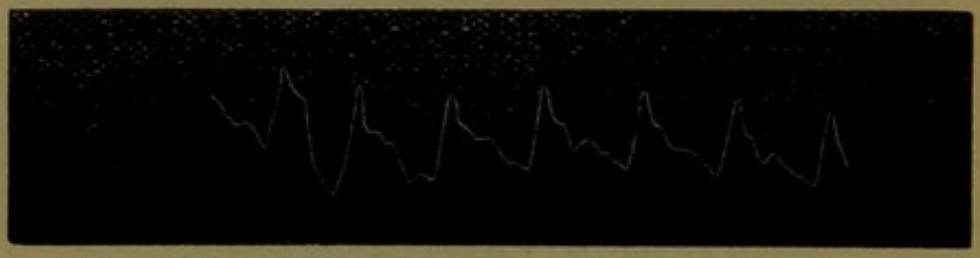


FIG. 9.—Normal beat, shows tidal wave, aortic notch, dicrotic wave. Case 3.

This is wholly different from the wild convulsions incident upon a corresponding period of apnœa and strongly suggests to the mind that the cessation of respiration under nitrous oxide is due to a sedative action exerted upon the medullary

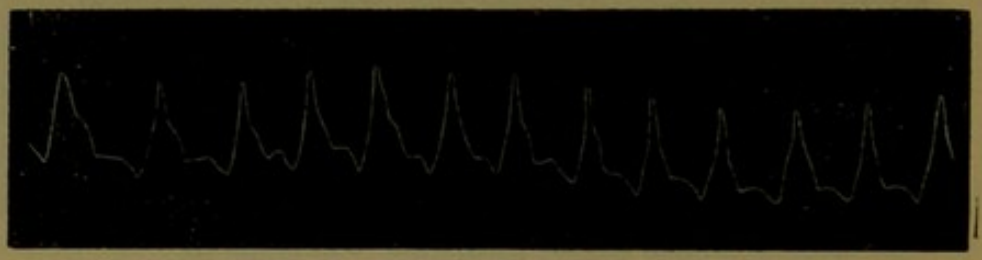


FIG. 10.—Under nitrous oxide. Shows acceleration of heart beats; increased acuteness of curve; diminution of tidal wave; dicrotic wave occurs later, this is shown by being farther from apex.

centres. Having now reviewed at what, I trust, you will not regard as an undue length the subject of the action of nitrous oxide, I will, with your permission, point out the practical

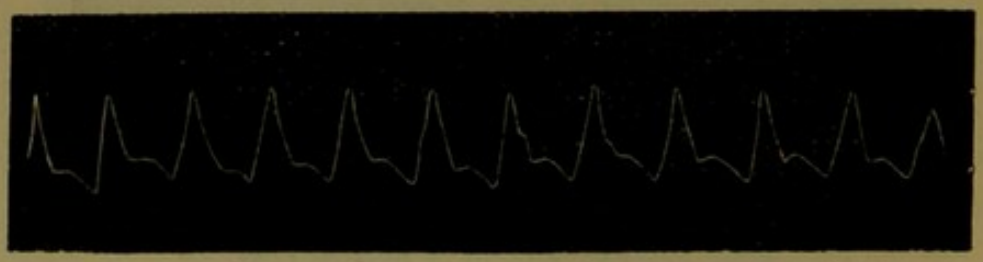
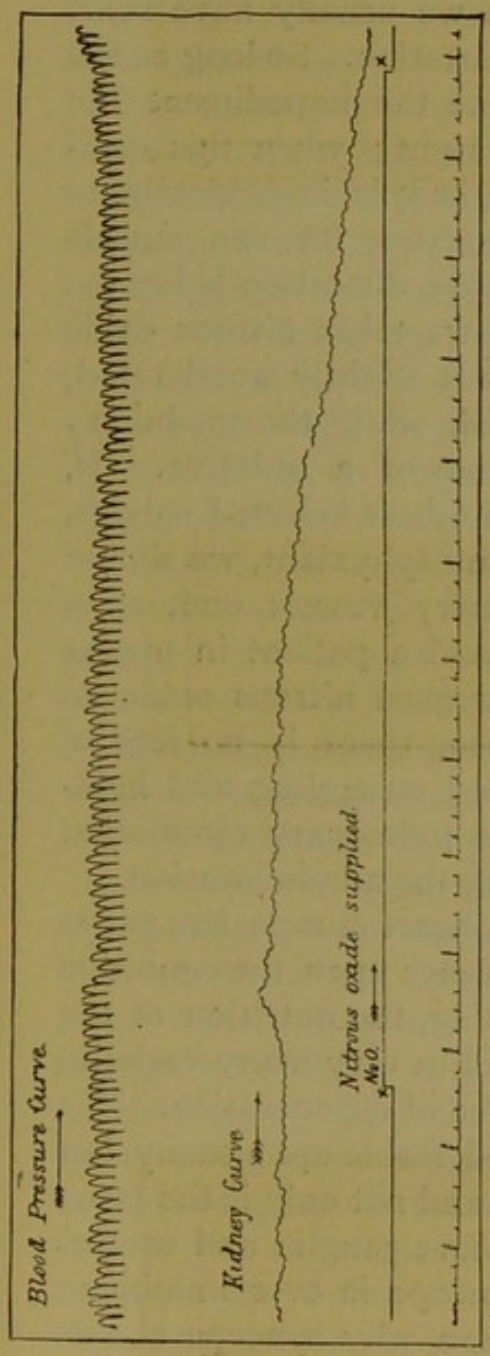


FIG. 11.—Nearly same as 2, but features more marked.

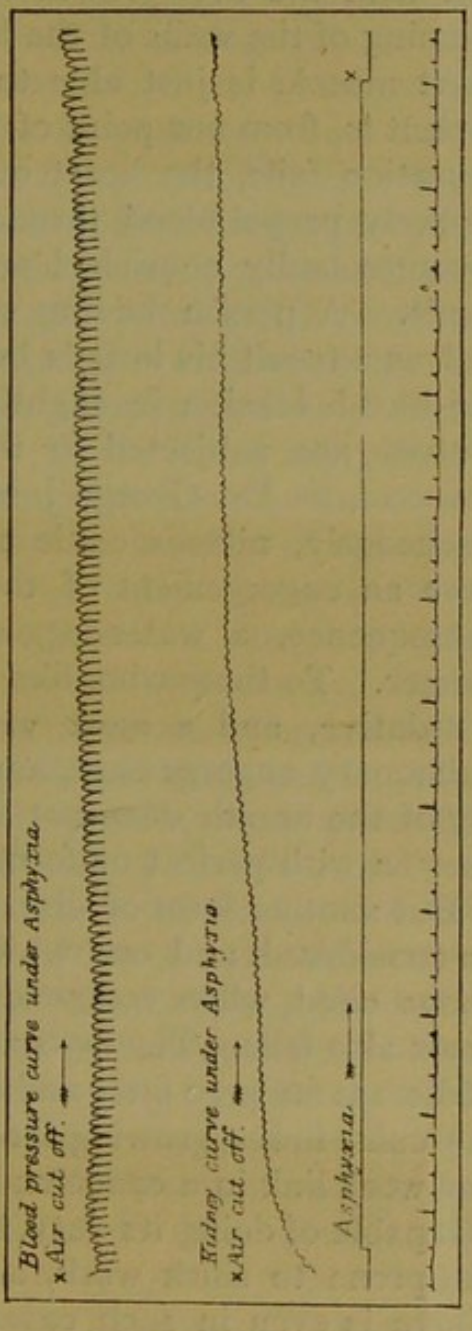
deductions we are justified in making, firstly, concerning heart and vascular diseases.

The heart may be hampered by organic disease; by functional disease; by mechanical displacement. Let us ask ourselves how far nitrous oxide is contra-indicated in organic heart disease. Broadly, we may consider the matter under two heads.



The upper line shows the blood pressure trace taken from carotid.
The second line is taken from the left kidney enclosed in an oncometer and shows the variations of blood pressure in the kidney.
The third line shows the duration of the experiment. N^2O being inhaled from the mark x, to the other mark x.
The bottom line is the time marker. The tracings are to be read from left to right—⅓ size.

FIG. 12.



Upper line shows carotid blood pressure during a period of asphyxia.
The second line shows blood pressure, &c., in the kidney enclosed in an oncometer.
The third line marks (between x, x, x), the duration of the experiment, and the bottom line is the time marking record.
Tracings to be read from left to right,—⅓ size.

FIG. 13.

(1) When the heart muscle is diseased.

(2) When the valves are diseased, although as we are all fully aware, the conditions are only too often coincident in the one individual.

The heart muscle may be enlarged—hypertrophied—with vigorous heaving impulse and displacement of the apex beat, and we know that such a heart is doing hard work against an obstruction existent somewhere in the circulation. Such a heart might exist in granular disease of the kidneys, in which the arteries impose a severe impediment to circulation. But with the over-growth of muscle we usually have some thinning of the walls of the heart—dilatation. So long as the heart muscle is just able to overcome the impediment the patient is, from our point of view, all right; when this compensation fails, the heart overworks, or it is incompetent to properly propel blood through the arteries, its own muscle becomes badly nourished and so yields, dilatation being the result. A person having such a heart takes nitrous oxide and as a result his heart's beats are but slightly accelerated, the blood tension is slightly lowered, while the medullary centres are subjected to the influence of a sedative. If, however, as Dr. George Johnson and others assert, I submit, erroneously, nitrous oxide acts as an asphyxiant, we should have an engorgement of the pulmonary vessels and, as a consequence, a water-logged heart and a patient in urgent danger. To those who, like myself, regard nitrous oxide as a sedative, and a most valuable one, there is no fear of pulmonary engorgement, and, provided struggling and holding of the breath does not occur, the pulmonary circulation goes on with perfect uniformity during the administration.

But another form of disease of the heart muscle has yet to be considered, and one which supervenes upon the condition above cited, when compensation failing, the nutrition of the heart also fails. The myocardium will in very many varieties of disease undergo fatty and other forms of degeneration. The muscular fibres growing fat-laden and feeble are usually but one weak link in a condemned chain, and not only is the heart incapable of doing its duty, but the nerve ganglia and centres are prone to shirk work, and so syncope is ever imminent. There is even in such cases no reason why syncope should be determined by nitrous oxide, and there is certainly less fear of it than from its incidence from shock. I am here speaking broadly and from the point of view of the peculiar action of nitrous oxide. In any very enfeebled individuals I should say clinical experience would decide one in estimating

the danger of the anæsthetic. My own experience has been that the cases of syncope one meets with are usually not associated with old fatty hearts, but occur in persons in whom anæmia is present; the tissues, while demanding much, obtain little; and in those whose nervous system is more or less enfeebled.

Of valvular heart lesions we must consider mitral and aortic obstruction, and mitral and aortic incompetence.

Here again were nitrous oxide an asphyxiant we might and ought to refuse the solace of gas to patients whose valves were sufficiently diseased to cause symptoms, but as it is not an asphyxiant I have in ordinary cases seen no symptoms the least suggestive of heart failure consequent upon an additional strain. We know that syncope is a frequent and a dangerous symptom of advanced valvular trouble, and upon the frequency or infrequency of these attacks I should decide whether or not to administer gas. There is not any property of gas which would determine syncope in valvular disease.

The group of cases falling under the heading of functional heart disease are far more dangerous. Such cases are those in which the heart's action is weak, feeble, irregular, and of poor tone, the anæmic persons worn out by prolonged disease or debauch, persons of ill-nourished nervous system, who for want of better names are called hysterics—male and female,—hystero-epileptics, the gouty and dyspeptic; those whose failing heart has ever been "assisted" along the downward path by nips of alcohol, *et hoc genus omne*. These persons have nervous systems which will take large quantities of sedatives without much effect; they are timorous, peculiarly liable to fear reflexes, and nitrous oxide has but a transitory effect on their cerebro-spinal axis, and not infrequently baulks us by its apparent failure. Another reason why nitrous oxide is often so unsatisfactory in these cases is that there is commonly a lack of due oxygenation of the tissues in these persons, and so when the full dose of nitrous oxide gas is administered there is a concurrent impoverishment of oxygen which induces an amount of apnœa such as should be absent when gas is properly given.

In our third class of cases we must group displacement of the heart by *pericardial adhesions*, by *pleuritic effusions*, *ascites*, *abdominal or other tumours*. Nitrous oxide gas, from the fixation

of the thorax, which the tonic spasm of the muscles may bring about, if that agent is pushed far, would be contra-indicated, and it would be wise, while commencing with nitrous oxide, to continue with ether.

The slight influence which nitrous oxide exerts upon the respiratory system renders detailed notice unnecessary. I have never, in my experience, found nitrous oxide, when pure, act as an irritant, and as this bland gas, as I have endeavoured to show, does not impede the pulmonary circulation, there is no ordinary intra-pleural or intra-pulmonary condition which should, in my experience, contra-indicate its use.

How far do conditions due to disease of the central nervous system contra-indicate nitrous oxide?

I can find no record, either in the experience of others or of myself, to show that any organic or functional lesion of the cerebral hemispheres or spinal cord, interfere with administration of nitrous oxide. One would expect from the very marked changes in the vaso-motor areas of the brain and cord which occur under that anæsthetic that whatever condition were brought about by vaso-motor perturbations would be initiated by nitrous oxide, and I believe that this is so in some degree. *Epileptics* and *hysterical* persons are often *deeply impressed* by it, and I have many times been struck by the curious condition which supervenes upon its administration to persons afflicted with *mania*. You are also aware that Dr. Savage who, during his control of Bethlehem Hospital enjoyed an almost unique opportunity of studying the effect of anæsthetics upon the insane, found that in persons who had once been mad, nitrous oxide, ether, or chloroform, would, in a small percentage of cases, give rise to an outbreak—usually transitory.

In the following case, for the notes of which I am indebted to the kindness of Dr. Colman, Registrar of the Queen Square Hospital for Paralysis, there is a record of an instance in which a temporary attack of insanity followed frequent dosage with nitrous oxide.

Lydia Askew, twenty-six. Married. No neurotic heredity could be traced. No previous illnesses. Admitted under Dr. Bastian suffering from diphtheritic paralysis. When convalescent, nitrous oxide was administered to facilitate the extraction of some carious teeth. The extraction proved very

difficult, and she was kept, more or less, under the influence of the gas for *about twenty minutes*. Although the apparatus was in good order, and she took a large quantity readily, it was found most difficult to get her completely under, and she afterwards proved that she had heard remarks made, although she felt but little pain. During the rest of the day she was merely rather stupid.

Next morning she thought she was in a "hospital ship," and complained of the motion. Made incoherent remarks when asked a definite question. In the afternoon suddenly became more rational, conversed intelligently, but had a wild expression.

During the next *four days*, in the beginning of the morning and late in the evening she relapsed into the previous condition, not knowing where she was and talking nonsense. During the rest of the time she was sensible, but much depressed. On the evening of the *fifth day*, after the administration, she suddenly jumped out of bed, saying she was going to drown herself, and had delusions that her children were dead.

She was discharged on the *sixth* day, in the care of her friends, and taken to the country. About a *fortnight* later she was reported to be as well as ever.

That a small minority of persons suffer from severe headache and temporary nervous paresis after gas, I can well believe, although one is apt to accept the accounts of such cases with a grain of salt. If they exist, they are very rare.

The question has arisen how far are persons with diseased arteries in danger from the increased intra-cranial pressure of nitrous oxide narcosis? Would, for example, a patient who suffered from miliary aneurism of the basal vessels incur a grave risk? The risk certainly exists, but is certainly less than that entailed by the shock of operation without gas, and may, I believe, be incurred in the interests of the patient. Mr. Browne-Mason's interesting case which appeared in the "Transactions of the Odontological Society," is especially noticeable in this connection. I have given gas to hemi- and paraplegics, to cases in which cerebral or cerebellar tumours were present, but in none of these cases have untoward, or even peculiar symptoms arisen.

In conclusion, what practical lessons have these researches taught us as to methods of administration?

The one lesson of all others is: To administer nitrous oxide pure and simple. If you adopt any means which causes your patient to re-breathe exhaled air, you are doing him harm and yourself discredit. All my preceding remarks refer to nitrous oxide narcosis, and not the mixed narcosis of that agent and suffocation. Those who prefer "suffocative narcosis" must please not to lay the flattering unction to their souls that its employment is free from danger. The one great mistake that most observers have made, and which Dr. George Johnson has recently rehabilitated, is to confound nitrous oxide narcosis with asphyxia, arguing from the mixed narcosis of suffocation and nitrous oxide to the safe anæsthesia of pure gas. The experiments upon which Dr. Johnson's arguments are based are suffocation experiments, and so far as nitrous oxide narcosis is concerned his arguments are invalidated.

I need not detain you long by dwelling upon the merits of nitrous oxide as an anæsthetic; its range of utility will be sufficiently indicated in the remarks I have made when referring to the diseased conditions of the body in which it can, with comparative safety, be employed. Chloroform, whatever be its merits—and they are many—cannot be used as freely as gas, nor can precautions as to posture, after effects, and so on, be neglected, as we rightly neglect them in giving nitrous oxide. Ether from its offensive flavour, its after effects, and its too violent action upon the blood pressure, cannot be taken in the hundred and one cases in which nitrous oxide proves itself our friend.

Its one drawback is, so I am told, the briefness of the anæsthesia it ensures. But I am not at all sure whether—and I speak under correction—this is not one of its main merits, as it prevents extensive laceration of the fifth pair of nerves in multiple extraction, which in very many cases is, I am sure, not free from danger. But this is not my department, not is it my business to enquire by what means, by mixed narcosis, we are enabled to prolong anæsthesia to a sufficient length of time for lengthy dental operations. I may say, however, that as a rule nitrous oxide alone can be manipulated to ensure a most satisfactory period of anæsthesia, and that I fear there is often too great a tendency to distrust gas alone and to have resort to some form of mixed narcosis, when such is really unnecessary.