

sent treatment of pneumonia is, as a general rule, perfectly satisfactory—the treatment, I mean, first formulised by the late Dr. Hughes Bennett, and founded on the principle that the patient is to be fortified and sustained in the trial that awaits him by means of such nourishing food as he can best take. That by this method an acute disease of such apparent, nay, of such real gravity, should be practically recovered from in a little over a week, is, it will be admitted, remarkable. There is nothing that I know of which drugs can achieve half so striking as is this result achieved by discarding them. There can be no greater mistake, however, than that of supposing that the treatment just indicated amounts only to a treatment of waiting and expecting. On the contrary, it implies a very urgent need for support, and a very present danger when such support is withheld. It is the spirit and not the precise letter of the treatment which has to be kept in view. It may happen in some cases that the need is so pressing that mere feeding will not suffice, or the danger may be so imminent that there is not time to wait for the good of it. It is not always that “nutrients” can be taken in sufficient quantity; sometimes they can hardly be taken at all. These are not instances where the treatment fails, they are instances where it requires special modification; where we have to substitute for the while some means of support which shall be more prompt and immediate than ordinary food.

It is here that the question of alcohol occurs, and the great difficulty is to know betimes exactly where and when to apply it. If we measure pneumonia by the amount of lung that is solid, we shall never, or only by occasional accident, get a correct estimate of it. On the other hand, if we consider the actual present condition and aspect of the patient as well as his immediate antecedents and surroundings; if we remember that the pneumonia of destitution and of drunkenness; the pneumonia that is fought against and for a while disregarded; the pneumonia that appears, be it ever so small as to its site, after severe nervous shock or prolonged exposure, that all these have a special need of support, and as a rule an absolute need for alcohol, then I think we shall be taking such a view of the disease as experience teaches, and applying legitimately the great principle upon which its successful treatment is based.

It was from this chair, not long before his death, that my friend and colleague, the late Dr. Anstie, in a clinical lecture upon pneumonia, spoke of the large quantity of evidence that he had collected and was preparing to publish in proof of the proposition that high temperature combined with large urea discharge furnished the strongest *primâ facie* reason for the administration of alcohol. I will not assume so much as this. I will take rather the admitted service of alcohol as defined accurately enough for our purpose in the well-known investigations of Professor Parkes. We can hardly contemplate the condition of these pneumonic patients, their low vitality, and the physical change which has to be accomplished within them before relief comes, without being reminded that here are precisely the circumstances where alcohol claims to be of use. Just at the pinch of crisis, when a little access of strength, a little more ability to assimilate food, is so urgently called for, when, moreover, as the nature of the disease teaches, a few hours will bring us to the time when we shall be able to pay the penalty incurred by resorting to such a succour, here, if anywhere, is the occasion and opportunity for alcohol.

Such a method of employing alcohol in pneumonia restricts its use to a particular period and a particular phase of the disease. When the food that the patient is able to take is obviously insufficient, when with a small lung implication his aspect is like that of typhoid fever, when he is past middle age, or his habits of living have been dissipated, or a period of mental or bodily distress¹ has preceded, and perhaps caused, the pneumonia—in all such cases, I think, we may expect great service from alcohol, and often find necessity for it. If I were called upon to express an opinion in few words as to the use of alcohol in this disease, I could (apart from the question of age) put the result of my observation into no more definite or scientific shape than this—that the pneumonia of mystery, that which comes from some obscure

or conjectural cause not commonly productive of such result, overwork or anxiety, or physical injury, or what not, and which nevertheless, upon interrogation of the other organs, appears to be a primary disease, is the kind that commonly needs alcohol; while the frank open pneumonia which is the result of some definite chill, or short exposure, commonly does without it.

And, finally, let it always be remembered that alcohol in pneumonia must be given in anticipation of danger rather than in the immediate presence of it. We find the patient blue and gasping, and hardly conscious, and, as by an instinct, in accordance with universal practice, we pour in brandy. But if we consider the matter, and especially the known action of alcohol as a powerful narcotic, it is rather harm than good that we ought to expect from such an agent at such a time. The opportunity for alcohol has passed. Whatever may be the hope in such a condition (a question I do not attempt to discuss now), it can hardly be this.

ON THE MYDRIATIC PROPERTIES OF HOMATROPIN, OR OXYTOLUYLTROPEIN.

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MOORFIELDS.

WITH AN ACCOUNT OF ITS

GENERAL PHYSIOLOGICAL ACTION,

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THE discovery of a new mydriatic must always be a matter of practical interest to the ophthalmic surgeon. For many years his *materia medica* contained only one pupil-dilator—namely, belladonna or its alkaloid, atropin—and even now one or other of these is almost always and solely employed. There are, however, many objections, some trivial, some serious, to the routine use of atropin. In some instances solutions of this alkaloid prove so irritating that they cannot be endured; and in all cases when applied for the purpose of temporarily suspending the power of accommodation in order to make manifest the real state of the refraction of the eye, its effects continue an inconveniently long time. But much more serious than either of these is the proneness of atropin to precipitate an attack of acute glaucoma in certain conditions of the eye which otherwise call for the use of a mydriatic. The discoveries of late years have added largely to the number of pupil-dilators, and some of these may, perchance, be more or less free from these ill-effects of atropin. Daturin, duboisin,¹ ethyl-atropium,² gelsemin,³ hemanthin,⁴ hyoscyamin,⁵ muscarin,⁶ narcissin,⁷ and pituria⁸ have all been found to be more or less powerful dilators of the pupil when locally applied, and impairers of accommodation. To this growing list we have now to add homatropin, which promises to be of very great value and utility in ophthalmic practice. It is a bland but efficient mydriatic, rapid in its action, and of comparatively short duration. It has, besides, many of the better characteristics of atropin. The specimen which I have used was supplied a few weeks ago by Mr. Martindale of 10, New Cavendish-street, and was a solution of hydrobromate of homatropin, four grains to an ounce. Dr. Ringer kindly undertook at the same time to test its general physiological action. The following is his report:—

“As regards hydrobromate of homatropin, I shall speak first of its action on frogs, and then of its effects on man. Three-fifths of a grain administered to a moderate-sized frog

¹ THE LANCET, March 2nd, 1878.

² Ringer's Therapeutics, 8th edition, p. 534.

³ THE LANCET, June 9th, 1877.

⁴ See forthcoming number of Seguin's Archives of Medicine (New York) for an article by Dr. Ringer.

⁵ Pharmaceutical Journal, Dec. 9th, 1876.

⁶ Ringer's Therapeutics, p. 493.

⁷ Foster's Journal of Physiology, 1878-9, p. 437.

⁸ Journal of Physiology, 1878-9, pp. 377-381, and THE LANCET March 1, 1879.

¹ Many examples might be quoted showing the greatly increased danger of pneumonia when happening (*ceteris paribus*) to a community exposed at the time to severe strain and excitement, as, for example, an army in the field, even although there should be no actual hardship or privation. The high mortality of pneumonia amongst our troops in Afghanistan may be perhaps accounted for in this way.

produces complete paralysis in about an hour, and the movements just preceding complete paralysis are tetanic. The paralysis soon grows less, and but little remains in the course of six or eight hours, and as voluntary movement returns the tetanus becomes more marked. Homatropin then, like atropin, paralyzes and tetanizes. The paralysis from both is transitory, and the animal recovers even when the paralysis is complete; but atropin paralysis more rapidly, and its effects are more persistent than homatropin. With atropin, tetanus only comes on after an interval of several days, but with homatropin tetanus appears just before paralysis is complete, and becomes well marked in a few hours, as the paralysis disappears. Whilst homatropin is a more speedy tetaniser than atropin, it does not tetanise more powerfully than atropin, and the tetanus, I think, lasts a shorter time.

"As after complete paralysis a very weak current of electricity (as, for instance, Du Bois Reymond's induction apparatus at 12) applied to the sciatic nerve through the skin causes energetic contractions of the lower leg and foot muscles, I conclude that the paralysis is not chiefly, if at all, due to the action of the poison on the motor nerves, and as the muscles contract readily and energetically with a very weak electrical stimulus, I conclude that the paralysis is not due to the action of the poison on the muscles, and as the paralysis is not due to the condition of the muscles or motor nerves, it must be due to the action of the poison on the nervous centres, and I conclude that, like atropin, its chief action is on the spinal cord.

"Homatropin, like atropin, paralyzes the intra-cardiac inhibitory apparatus. I exposed the heart of a brainless frog, and on applying the electrodes to the junction of the sinus and the auricles, the heart was strongly inhibited and stopped beating for some time. I then poured some of the homatropin solution into the abdominal cavity, and a few minutes later again tested the heart with the same strength of electricity; but the heart was not stopped—on the contrary, its contractions were accelerated. I repeated this experiment on two frogs.

"Like atropin, I find that homatropin antagonises the action of extract of muscaria on the frog's heart.

"Both substances, when topically applied to the heart, slow its action. In a series of comparative experiments I find homatropin, on an average, slows the heart thirteen beats, and atropin twenty-three beats, in the minute.

"I am indebted to my resident assistant at University College, Mr. W. H. Copley, for the experiments on man, the greater number of which were made on my clinical clerk, Mr. Charles Stonham, who received on separate days a hypodermic injection of one-fiftieth, one-fortieth, and one-thirtieth of a grain respectively. On each occasion his pulse was slowed on an average of 21 beats, and became irregular in force and rhythm. No other effect occurred. His pupils were not dilated, nor did his mouth become dry, nor was his face flushed, nor his breathing hurried. In two observations on other people, the pulse fell 18 and 12 beats per minute respectively. In the first the pulse became irregular. The first received one-seventieth of a grain; the second, one-sixtieth.

"On a subsequent day Mr. Stonham received hypodermically one-sixtieth of a grain of sulphate of atropin, but as some of this escaped in thirty-five minutes, the dose was repeated. The atropin raised his pulse from 72 to 100 per minute, dried his mouth and throat, and dilated his pupils.

"Mr. Copley next tested on two occasions the antagonism between pilocarpin and homatropin. He administered hypodermically one-third of a grain of nitrate of pilocarpin, and immediately sweating became profuse he injected one-sixtieth of a grain of hydrobromate of homatropin, and on both occasions this dose stopped the sweating in about ten minutes. On another day he tried a comparative experiment with sulphate of atropin, using the same dose of atropin as of homatropin. Mr. Copley found atropin a more powerful arrester of sweating than homatropin.

"Homatropin, then, appears to possess many of the properties of atropin, but in a weaker degree. On the heart, however, their effect is very different, for atropin accelerates and strengthens the heart's contractions in man, whereas homatropin slows the beats, and renders them irregular in force and rhythm."

The first sentence of the last paragraph of Dr. Ringer's report exactly describes my opinion of the action of homatropin on the eye when locally applied. "It appears to

possess many of the properties of atropin, but in a weaker degree." It must, however, be distinctly understood that though relatively weaker than atropin, its action on the iris and ciliary muscle is really very powerful while it lasts. It is not merely weak atropin in the sense that it only does what a weak solution of atropin would do. A solution of the homatropin of the strength above mentioned widely and fixedly dilates the pupil in from fifteen to twenty minutes, and affects the accommodation in an equally rapid manner. The chief peculiarity, and in some respects the great advantage, of homatropin, is that its effects soon pass off, and certainly do not last anything so long as those of atropin. Within four-and-twenty hours after an application the accommodation, after complete apparent suspension, recovers its pristine vigour, the pupil is less dilated, and reacts to light, though it has not quite regained its original size. As regards its action on the ciliary muscle, though decidedly more prompt, it is not quite so intense as that of atropin. Homatropin is, moreover, singularly unirritating. I have applied it to about fifty cases, and in not one was there the smallest sign of irritation or discomfort.

One of the most remarkable facts respecting homatropin has yet to be stated. According to Herr Ladenburg, this body may readily be obtained by treating amygdalate of tropin with dilute hydrochloric acid in a water-bath, and precipitating the solution with potassium carbonate.⁹ Now amygdalate of tropin is itself quite inert, at least it does not possess any of the properties of atropin. Locally applied to the eye it does not affect either iris or ciliary muscle, nor does it, according to the observations of Dr. Ringer, like atropin, antagonise the action of muscarin on the frog's heart. Injected hypodermically by Mr. W. H. Copley, it did not in any appreciable degree check sweating artificially produced in man by pilocarpin.

A few final explanatory words may assist the reader to understand chemical the relations of this body.

Some time ago Kraut and Lossen almost simultaneously found that atropin may be split up into *tropin* and *tropic acid*, and last year Ladenburg succeeded in reforming atropin from these two constituents.¹⁰ By treating the different salts of tropin with dilute hydrochloric acid, a class of alkaloids may be artificially produced to which Ladenburg has given the generic name of *tropeins*; and homatropin is one of these alkaloids obtained from the amygdalate of tropin—that is, tropin combined with amygdalic acid.¹¹

OBSERVATIONS ON THE UPPER AIR-PASSAGES IN THE ANÆSTHETIC STATE.

BY BENJAMIN HOWARD, A.M., M.D., F.R.C.S.E.

I.—THE SUPPOSED ELEVATION OF THE INSENSITIVE EPI- GLOTTIS BY PULLING FORWARD THE TONGUE.

UPON one point at least, and one not altogether unimportant, the entire profession, both in theory and practice, may fairly be admitted to be everywhere unanimous. To this unity, the jaw-priser and the tongue-forceps in every operating room bear silent witness. The livid face, pulseless wrist, motionless thorax, and locked-jaw supervening in anæsthesia, make it by common consent imperative, that instantly, at whatever amount of violence, the tongue be forcibly pulled forward; because thus, and thus alone, can the paralysed and fallen epiglottis be raised and the air-way be made free. With others, in this belief and practice I have fully shared, and I suppose for the same reasons: firstly, of authority; secondly, of supposed experience.

While recently pursuing some cognate investigations, however, I thought it well to take the opportunity to observe, not whether, but precisely how, the pulling forward of the tongue, as practised, raises the epiglottis as alleged. The result compelled me on this point to multiply my observations. In the course of them I endeavoured to see precisely what traction upon the tongue does, and what it does not.

⁹ Berichte d. deutsche chemische Gesellschaft, xiii., 166; also Pharmaceutical Journal, March 20th, 1880, p. 751.

¹⁰ Comptes Rendus de l'Académie des Sciences, tom. xc., No. 13, p. 874 (April 12th, 1880); also Pharmaceutical Journal, May 1st, p. 571.

¹¹ Pharmaceutical Journal March 27th, p. 771.