OBSERVATIONS

ON

SEA-SICKNESS, AND ON SOME OF THE MEANS OF RELIEVING IT.

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THE cause of sea-sickness and its possible amelioration is a subject particularly appropriate at the present time. It is more agreeable to offer suggestions for relief than to comment on the sybaritic weakness which is generally displayed in impatience of the evils usually to be encountered in a passage across the Channel. When we remember that not half a century ago, people, after waiting days or perhaps weeks for fair wind and weather, were glad to consider a voyage of six hours of tossing and sickness as a very favourable one, one can hardly understand how the present generation feel it unendurable to submit to a single hour and a half after having started at a given time. Though, however, we must admit the fact that we are less brave in meeting discomforts than our predecessors, there is still satisfaction in diminishing those discomforts to the utmost in our power.

We have seen the futility of various childish devices, such as stimulants, globules, ice-bags, etc., all of which, not being based on any true knowledge of the evils to be met, are merely empirical. Rejecting all of these, therefore, it will be well to invite attention to a scientific explanation of the cause of sea-sickness, and to base on that explanation a proposal for a remedy.

A suggestion was made sixty years ago by Dr. Wollaston, that sea-sickness proceeds from pressure of blood upon the brain; and this view is supported by pathological observations, since injury or pressure on the brain is almost invariably attended by vomiting, which is its earliest symptom. Dr. Wollaston explains the way in which pressure is induced upon the brain during the motion of a ship at sea, by reference to the action of mercury in the tube of a barometer. He says "that if a barometer be carried out to sea in a calm, the mercury will rest at the same height as when on shore; but when the ship falls by the subsidence of the waves, the mercury is seen apparently to rise in the tube which contains it". I may add to this, that anyone who has carried a mountain-barometer, and has happened to let it descend suddenly, must have been sensible of a concussion of the mercury against the top of the tube, and must have both felt and heard the blow. In fact, the mere action of walking is sufficient, by the alternate rise and fall of the hand, to produce this effect. Dr. Wollaston considers that the action of the blood on the brain, at the moment of the descent of the ship, is identical with that of the mercury on the top of the barometer, and that there is an actual pressure, and even a blow, which, by frequent repetition, produces nausea and vomiting. Now it is to be regretted that Dr. Wollaston does not state the true scientific explanation of the apparent rise of the mercury in the barometrical tube, to which inaccuracy may possibly be referred the reason why so little notice has been taken of this valuable suggestion. It is not necessary here to analyse his statement with a view to refute it. The fact is undeniable, that contact does take place between the mercury and the upper part of the tube with more or less violence, and the proper explanation of the apparent rise of the mercury when the tube descends is this: that when the rigid tube falls, the mercury, having its own inertia, and not being attached to, or a part of, the tube, remains stationary—at least for a time; thus the tube is pushed down upon or over the mercury, and the concussion takes place. Exactly the same occurs between the brain and vessels on the one part and the blood on the other. The approximately rigid brain and vessels are carried downwards, the blood remains by its own inertia, and the consequence is to crowd blood into the vessels of the brain, and so press with increased force, producing a certain shock; this shock and the attendant pressure produce sickness and vomiting. The vomiting thus induced is of a peculiar character—very different from that proceeding from a common disordered stomach; it occurs in a spasmodic manner, and violent retching remains after the contents of the stomach have been ejected. This continuous retching seems to indicate the repeated action of the increased pressure.

Referring to the experience of sufferers from sea-sickness, it is admitted by all that they are most sensible of the miserable feeling at the moment of the descent of the ship. They are also conscious at that particular time of an instinctive effort to sigh or take a deep inspiration, the meaning of which is manifest. During deep inspiration the chest is dilated for the reception of air, and its vessels become more

open to admit blood, so that a return of blood from the head is then more free than at any other period of complete respiration; whilst on the contrary, by the act of expelling air from the lungs, the ingress of blood is obstructed. This obstruction is proved by observation when the surface of the brain is exposed by the operation of trephining; a successive turgescence and subsidence of the brain is then seen in alternate motion with different states of the chest. A deep inspiration, therefore, at the time of the descent of the ship tends to counteract the turgescence of the brain.

Sickness is sometimes produced by waltzing. In this case the same theory of pressure on the brain holds good; but during rapid gyration in waltzing the blood is acted on differently: it is centrifugal force which causes the blood to rise in the vessels supplied to the brain. There is an additional cause of cerebral disturbance from the confusion of objects rapidly presented to the eye; from this comes giddiness.

In reference to sickness brought on by swinging, I cannot do better than quote Dr. Wollaston: "Sickness, by swinging, is evidently from the same cause as sea-sickness, and that direction of the motion, which occasions the most piercing sensation of uneasiness, is conformable to the same explanation already given. It is in descending forwards that this sensation is perceived, for then the blood has the greatest tendency to move from the feet towards the head, since the line joining them is in the direction of the motion; but when, in the descent backwards, the motion is transverse to the line of the body, it occasions but little inconvenience, because the tendency to propel the blood towards the head is then inconsiderable."

This last observation of Dr. Wollaston, quite accurate as to the result, plainly suggests the practical bearing of the subject. Knowing the mode in which the ship's movement acts on the brain, we are at once furnished with the only rational way of averting sea-sickness.

The first point is wholly to avoid the upright posture. Every one knows that it is a common practice to lie down, and this is done almost instinctively, but it is also known that to do so, though frequently successful, it is not invariably so. The way in which the motion in a swing affects the brain affords the proper explanation why lying down is not invariably successful; and shews that it is necessary, not only to take a recumbent position, but to lie in the right direction. A person lying down with the feet towards the bows of a ship is, whilst it descends in pitching, in the same position as a person in a swing descending forwards, in which case we have seen that sickness is produced by blood being forced upon the brain. On the contrary, a person lying down with his head towards the bows is, during the descent of the ship, in the position of one descending backwards in a swing, in which case the pressure by the blood will be towards the feet, and, therefore, relief rather than an inconvenience will be experienced, the tendency being to reduce the natural supply of blood to the brain. It is necessary, therefore, not only to lie down, but to do so with the head to the bows; and it is highly desirable that this position should be assumed before the ship begins to move. There is a secondary advantage to be gained by closing the eyes, and so shutting out the confusion arising from the movement of surrounding objects.

If the philosophical explanation here given be the correct one, which there is no reason to doubt, it adds one more to many unanswerable objections to the device of taking passengers in railway carriages on board gigantic vessels. No relief would be afforded by that plan to the miseries of sea-sickness, since, except in a perfect calm, nothing can prevent the rising and falling of the ship, and the consequent action of the blood upon the brain. The sitting posture would be equally unfavourable with the upright, and there would be, in addition, the common motion of a carriage, which alone, with some persons, produces sickness.

It is well to forbear imagining the miseries of sitting, shut up, face to face, with fellow-sufferers, and, at the end of the voyage, of missing the one consolation of leaving the scene of your sickness, with all its disgusting evidences behind, and getting into a clean and pure conveyance free from late contaminations.

It is beside the subject of this paper to discuss the danger of unstable equilibrium from deck-loading, which must raise the centre of gravity and depress the metacentre; also to hint that engineers may promise immunity from danger, whilst the melancholy fate of the *Captain* forbids us to trust in their scientific calculations. One thing is more than probable, that, in rough weather, deck-loading will produce heavy rolling motion.

Short of the wild idea which goes by the name of the "Ferry scheme," with all its doubtful experiments and certain tremendous outlay, much might be done to alleviate the discomforts of the passage across the Channel. There might be larger vessels, with increased steam-power; better means for embarkation; shorter passages might be ensured, and comfortable arrangements might be made, so that every passenger could recline with the head to the bows of the vessel, whether on the deck or

in the cabin. All these points might be ensured without any dangerous experiment, and there can be little doubt that the testimony of passengers would soon prove the efficacy of the plan.

CLINICAL LECTURE

ON

A CASE OF CHRONIC BRIGHT'S DISEASE, WITH RAPIDLY FATAL SANGUINEOUS APOPLEXY.

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Rapidly fatal Apoplexy in a Woman of Intemperate Habits.—Copious Hæmorrhage into the Brain.—Small Granular Kidney.—The Left Ventricle of the Heart hypertrophied.—Hypertrophy of the Muscular Walls of the Minute Arteries in the Kidneys, the Pia Mater, the Skin, and the Intestines.

GENTLEMEN,—A few days since there was admitted a typical case of rapidly fatal sanguineous apoplexy in an intemperate woman, who had also been the subject of chronic Bright's disease. I propose now to give you a short account of this case, and to explain to you its pathology.

Mary T., aged 46, was brought to the Hospital at 11.30 P.M. on February 10th. She was perfectly unconscious, and the people who brought her knew but little about her. They were with her in a public-house, where they saw her drink a glass of beer; and they believed that she had previously had some spirits. She was seen to stagger suddenly, and then to fall and to become unconscious. They lifted her up, and brought her to the hospital. She was said to be a single woman, about forty-six years of age. She is believed to have suffered from occasional attacks of giddiness, but no distinct history could be obtained. When seen by the house-physician, Mr. Warner, to whom I am indebted for the notes of the case, she was being supported in a chair, in a state of complete unconsciousness. There was no dropsical swelling. The heart's sounds were normal. There were no marks of external injury. The pupils were of natural and of equal size. When the right eyeball was touched, there was reflex action of the lids; none when the left was touched. The left arm and leg appeared to be perfectly relaxed and powerless, but there was slight resistance when the right arm and leg were flexed or extended. Two slight epileptic convulsions occurred, during which the whole body became rigid. The stomach was emptied by the pump, and a liquid smelling of brandy was withdrawn. She was then sent up to the ward, and placed in bed. She remained unconscious; the breathing gradually became more feeble; and she died quietly at 7 A.M., in rather less than eight hours after the first seizure in the public-house.

On examination after death, we found a very copious hæmorrhage into the brain. A large portion of the right hemisphere, on a level with the lateral ventricle, was excavated by effused blood; the outer margins of the corpus striatum and optic thalamus were partly broken down by the clot; and the blood had passed through the torn septum into the left ventricle. The arteries at the base of the brain had many atheromatous patches in their walls. The kidneys were small and granular; the right weighed $3\frac{1}{2}$ ounces; the left $2\frac{1}{2}$ ounces. The liver was rather pale and fat. The heart weighed $14\frac{1}{2}$ ounces. The walls of the left ventricle were hypertrophied. The valves were all healthy; and the coats of the aorta were not diseased. The lungs presented the engorged appearance which is always found when death has taken place by coma and consequent slow apnoea.

I have here under the microscope a section of the kidney. An examination of this specimen will show you the whole series of changes which occur during the progress of that form of chronic Bright's disease which results in the small red granular kidney. You will see that many of the tubes are opaque, in consequence of being filled with the granular debris of disintegrated epithelium. From other tubes, the

Fig. 1.—Normal artery from the kidney, showing the thickness of the walls in proportion to the canal. In the muscular wall there is an outer circular layer and a thin inner longitudinal layer.—X200.

epithelial particles have been removed; and these tubes, composed only of denuded basement-membrane, have an abnormally transparent appearance. Again, you will find that some of the denuded tubes are smaller than natural, having undergone various stages of atrophy. The minute renal arteries have their muscular walls much hypertro-

phied, presenting the appearance—so constantly found in the hypertrophied renal arteries—of an inner longitudinal layer of fibres equal in thickness to the outer circular layer. You must compare the appearance of a normal artery (Fig. 1) with that of an artery which has undergone this remarkable hypertrophy of its muscular walls (Fig. 2). You will

Fig. 2.—Artery with hypertrophied walls from the kidney. The inner longitudinal layer of fibres is equal in thickness to the outer circular layer. The canal of the vessel is filled with injection.—X200.

see in the section of the kidney no indication of a so-called proliferation of connective tissue between the tubes; but, where there has been much wasting of the gland, the atrophied remains of tubes and capillaries have a fibrous appearance, and the matrix around the wasted tubes seems to be thicker in proportion as the area of its meshes is diminished. The essential changes in this, as in all the forms of chronic Bright's disease, are found in the tubes and in the blood-vessels. The hyperplasia of connective tissue, as the essential anatomical condition of the small granular kidney, is a theoretical fiction, not an anatomical fact, as you may see from a careful inspection of this specimen. You see, too, the very striking changes which the tubes and the arteries have undergone—changes of which the theory in question takes no note, and which it is utterly inadequate to explain.

I have here also, for your inspection, specimens which have been kindly prepared for me by Dr. Kelly, of minute arteries from the pia mater, the skin, and the intestines, of the same subject. You will find that, in all the specimens, the muscular walls of the arteries are conspicuously hypertrophied (Fig. 3).

Fig. 3.—Artery with hypertrophied walls from the subcutaneous tissue. The canal of the artery is injected.—X200.

Now, having given you the morbid anatomy of this case, the pathology, or, as I prefer to say, the physiological interpretation of the facts, is sufficiently simple and intelligible. The patient was a woman of intemperate habits, and a large proportion of the renal gland-cells had been destroyed in the discharge of their functions as eliminators of alcohol. The minute renal arteries, as the regulators of the blood-supply to the gland, resist the entrance of blood in proportion to the destruction of the secreting cells; and this continued excessive action of their muscular fibres results in hypertrophy of their walls. The contraction of the minute renal arteries, in opposition to the blood-stream, when the secreting tissue of the kidney is partially destroyed, is exactly analogous to the arrest of the circulation by the contraction of the minute pulmonary arteries when, from any cause, the respiratory changes are suspended or greatly diminished. As the renal degeneration made progress, the blood became impure, and the physically altered blood excited unusual contraction of the minute arteries throughout the system. Again, this resistance in the minute systemic arteries was met and compensated by more forcible contraction of the left ventricle of the heart; and the result of this continual over-action was hypertrophy of the arterial walls and of the walls of the left ventricle. The amount of hypertrophy is an index of the degree and duration of the excess of muscular contraction during life. One result of this antagonism between the propelling and the regulating forces is to cause increased pressure upon the walls of all the arterial tubes be-