ordinary ague, from which this disease differs, otherwise than in the suggested sequence of symptoms, *in toto*. The inquiries I have made do not support such a sequence, and certainly not a relationship to malarial ague. The symptoms are just such as would be caused by the ingestion of a quantity of an irritant metal, sufficiently large to cause vomiting and its attendant depression. Such, indeed, is my opinion of the causation of the symptoms, and therefore the name "ague" should not be continued, as being wrongly suggestive and misleading.

It will be remembered that it is only when fresh to the work that brassworkers suffer from "ague;" but, though they do not suffer from acute metallic poisoning, they do suffer from its chronic effects, and it is extremely probable that, as with arsenic and opium eaters, they may become inured to the use of the metals. It is not very common for brassworkers to use tooth-brushes, and the accumulating tartar will be found coloured green. If every effort be made to cleanse the teeth, there will yet be generally green discoloration of them. This has been proved to be due to the presence of copper. White hair is often coloured green among these workmen, and the underclothing is often stained green by the perspiration. The gums may be slightly blackened at the edges, but there is nothing distinctive as in the case of the blue line of lead poisoning; nor, indeed, beyond the green colouring of the hair and teeth, do brassworkers present any unequivocal evidence of their calling.

Ague is not a disorder for which brassworkers consult a medical man; they know how to treat it themselves, and also that it is transitory in its effects; but they come to hospitals in large numbers to be treated for bronchitis. About this there is nothing special. They suffer from it in common with all workers in dusty trades; and, so far as I can learn from the secretary of the Brassworkers' Organisation, they usually die from chronic bronchitis or fibroid phthisis, unless they succumb to some acute malady.

The existence of nervous disorders, especially paralysis agitans, has been said to be common amongst them, but I cannot find that a larger percentage of brassworkers than of the rest of the community suffer from diseases of the nervous system. It is common, however, amongst them to meet with complaints of disturbance of the digestive function. They suffer from dyspepsia, loss of appetite, gastro-intestinal catarrh, nausea, vomiting, metallic taste, thirst, colic, constipation, and diarrhoea. They are often nervous and hypochondriacal, complaining of headache and muscular pains. There is nothing distinctive about any of these disorders, except the obstinacy with which they resist ordinary methods of treatment, and the readiness with which they yield to the administration of iodide of potassium in combination with the other drugs indicated by the various conditions of ill-health. All the symptoms bear a remarkable resemblance to those produced by chronic copper poisoning, and in Guy and Ferrier's *Forensic Medicine* an outbreak of copper poisoning from the use of copper vessels in cooking is recorded, in which the symptoms were almost identical with those here mentioned.

The inmates of a convent suffered severely from obstinate and severe colic, retching, and bilious vomiting, costiveness and flatulence, burning pain in the pit of the stomach and extremities, and paralytic weakness in the arm. According to Stevenson, it is impossible to distinguish between the symptoms produced by zinc and copper poisoning; these are just such as brassworkers suffer from, and it is therefore impossible to say which metal—copper or zinc—is most concerned in the production of these symptoms.

Dr. Greenhow attributes them all to the inhalation of the de-flagrating zinc; this forms zinc oxide, which is only sparingly if at all soluble, and therefore is not likely to be freely absorbed in the stomach; but he pays little or no attention to the common and chronic gastric and intestinal troubles to which brassworkers are liable. These affect all those engaged in the various processes by which either in vapour or in minute particles copper and zinc—that is, brass—are distributed in the atmosphere.

Dr. Hogben, on the other hand, considers copper alone to be the efficient cause of the symptoms, and advances the following arguments:

1. We have no evidence that the internal administration of zinc ever produces the symptoms of brass-ague. Enormous doses of the oxide have been administered without apparently producing the characteristic febrile reaction of brass-ague.
2. The malady is observed in individuals whose work is other than casting.

3. The malady is not observed in operatives such as galvanised ironworkers, who work with zinc and are exposed to its fumes.

4. Zinc is rapidly excreted, and does not, like lead, mercury, or copper, become fixed in the body, and produce chronic affections.

It seems more probable that, accepting Stevenson's statement of the impossibility of distinguishing between the effects of acute copper or zinc poisoning, the symptoms of ague are due to an admixture of the two metals, whereas for the chronic complaints the copper is responsible.

How the practice of taking milk during an attack of so-called ague has arisen is not clear, but its wisdom is proved by the fact that in cases both of copper and zinc poisoning, milk is one of the best antidotes, since it precipitates both these metals into insoluble albuminates.

It is abundantly evident that brassworkers are specially liable to diseases from the use of the metals employed in its manufacture, but they are not new disorders; they are either proofs of chronic poisoning by zinc or copper, or as in the so-called ague, are due to intoxication by them.

For much of my information as to the effects of this work I am indebted to Mr. Lilly, the local secretary of the Brassworkers Association.

ON TESTING THE COMPETENCY OF THE VALVES OF THE HEART BY MEANS OF AIR.

By D. J. HAMILTON, M.B., F.R.C.S.E., F.R.S.E.,
Professor of Pathology, University of Aberdeen.

THE ordinary method of testing the competency of the valves of the heart with water is admittedly faulty in many respects. It can be applied only to the aortic and pulmonary artery orifices, and even with them the pressure of $1\frac{1}{2}$ to 2 inches of water, which alone can be retained in the attached portions of the vessels, does not represent anything like the pressure to which they are naturally subjected. Hence a valve which during life might have been sufficiently incompetent to give rise to a murmur, may be found after death to be perfectly competent. The water method is also defective from the fact that the cusps of the valve are not seen in motion, so that the mechanism by which a murmur may have been elicited is incapable of being demonstrated in the *post-mortem* room. We possess, moreover, no exact method of gauging the amount of incompetency of the mitral and tricuspid, nor of ascertaining when they are competent.

For some time past I have been in the habit of demonstrating to my class the natural action of the valves by driving air into them instead of water, and also of reproducing the murmurs heard in disease of the valves by several simple modifications of the same method.

The ascending aorta with the attached valve is cut off from the heart of an ox. A brass tube attached to a bellows is introduced into the distal end of the aorta. The aorta is now fixed in as nearly as possible its natural position, and air is driven in from the bellows with a sharp click, so as to imitate as closely as may be the effect of the arterial recoil. With each inflation of the bellows the cusps are retracted, to be closed again as soon as the air is expelled; and so perfect is their adaptation, even after death, that not only do they prove perfectly competent when the heart is healthy, but the sharp sound elicited bears a remarkable resemblance to the second sound of the heart. The valve can be turned up in any position, so as to be made readily visible to a whole class.

The same means may be employed to display the action of the pulmonary artery valve. In the case of the mitral and tricuspid, the respective auricles should be cut away close down to the orifices. The nozzle is pushed down through the aorta or pulmonary artery, as the case may be, past the valve, so that it enters well into the cavity of the ventricle. It is tied into the vessel so as to prevent regurgitation of the air.

It struck me that the same means might be utilised for testing the competency of the valves in the *post-mortem* room; and, having employed the method lately in a number of cases, I find that it works admirably. It can be applied almost as quickly as the water test, and has the great advantage of being available for all the orifices.

The procedure I adopt is the following. An opening is made into the left auricle, so as to expose the auricular aspect of the

mitral. Any *post-mortem* clots which may be adhering to the edge of the valve are removed with care, so as not to disturb the position of the cusps. An aperture sufficiently large to admit the brass nozzle is next made into the apex of the left ventricle in the line of incision which will afterwards be required to lay the ventricle open. The nozzle is then introduced, and air driven in by the bellows in the manner before described. The heart should meanwhile be suspended by the cut edges of the auricle. A series of hooks fixed on supports is useful for this purpose, the supports being movable so as to allow of the proper apposition of the cusps.

The slightest incompetency can be detected quite as much by the irregular manner in which the cusps mutually adjust themselves, as by the escape of air between them.

The same means of demonstration is to be followed in the case of the tricuspid, and, after its condition has been ascertained, the incision in the left ventricle is carried up as close as possible to the root of the aorta, without, of course, injuring the aortic ring. The heart is now suspended, as before, by the cut edges of the ventricle, and the nozzle is tied into the distal end of the aorta. The aorta should either be cut off at the termination of the ascending part of the arch, or be laid open up to this point. The nozzle both in the case of the mitral and in that of the aortic may be simply held in with the hand, or it may be tied in; or, what is even preferable, a simple clamp apparatus may be attached to its extremity, which seizes the cut edge of the vessel or cavity into which the nozzle is introduced, and holds it air-tight or nearly so. In watching the action of the valve, the ventricular aspect should be turned upwards, the air being driven in from below.

The pulmonary artery orifice is next tested in the same manner. In a perfectly healthy heart it will be found that the aortic, mitral, and pulmonary artery valves are quite competent, but, so far as my present experience goes, I have never met with a tricuspid in man which will stand the test. There is always a slight leakage, even when the cusps are perfectly free to move.

THE PROCESS OF COMPENSATION AND SOME OF ITS BEARINGS ON PROGNOSIS AND TREATMENT.

By BYROM BRAMWELL, M.D., F.R.C.P. EDIN.,

Assistant Physician to the Edinburgh Royal Infirmary; Lecturer on the Principles and Practice of Medicine in the Extra-Academical School of Medicine, Edinburgh, etc.

(Concluded from page 840.)

Compensation in Cardiac Lesions.—One of the most common and obvious methods of compensation is the increase (hypertrophy) of muscular tissue which takes place in muscular tubes and muscular organs when an increased effort is frequently and repeatedly demanded of them. The hypertrophy of the bladder, œsophagus, and intestine in cases of organic stricture are familiar examples; but there is, perhaps, no organ in which compensatory hypertrophy is more beautifully developed than in the heart. But, although hypertrophy of its muscular walls is by far the most important manner in which valvular lesions are compensated, it must also be remembered that in some forms of valvular disease a certain limited amount of dilatation and alterations in the frequency of the cardiac contractions also take part in the compensation, and assist the hypertrophy to restore and maintain the balance of the circulation. It may be stated broadly that in obstructive valvular lesions compensation is effected by muscular hypertrophy, aided, in some cases, by slowing of the cardiac action, while regurgitant lesions are compensated by hypertrophy, aided by a certain but strictly limited amount of dilatation, and it may be by increased frequency of the cardiac contractions. The manner in which the different valvular lesions are compensated is briefly as follows:—

In stenosis of the aortic orifice, compensation is chiefly effected by hypertrophy of the left ventricle, by means of which an increased quantity of blood is forced through the narrowed orifice at each ventricular contraction. The duration of the ventricular systole is at the same time lengthened, while the frequency of the cardiac contractions is diminished rather than increased, with the result that more time is afforded for the hypertrophied left ventricle to discharge its contents. In aortic stenosis, dilatation of the left ventricle affords no aid as a means of compensation, and when it does occur, it is a secondary and injurious result.

In stenosis of the mitral orifice, compensation is partly due to hypertrophy of the left auricle, but chiefly to hypertrophy of the right ventricle. Prolongation of the ventricular diastole, increased duration of the auricular systole, and slow action of the heart are also, when present, important factors in the compensation, since they facilitate the flow of an increased quantity of blood through the stenosed orifice during each cycle of the heart. At the best, however, the compensation in severe mitral stenosis is imperfect, much less perfect than it is in many cases of aortic stenosis, for even when fully developed the pulmonary circulation is subject to some strain. The most that the compensation can do in mitral stenosis is to enable the left ventricle to supply an adequate quantity of blood to the peripheral tissues, and to protect the organs and tissues which are situated behind the right heart from the effects of venous engorgement. In mitral stenosis, the pulmonary circulation is exposed to engorgement and all its secondary consequences, for the obstruction in front (that is, at the mitral orifice) is not suddenly relieved, as it is in mitral regurgitation, by the bursting open of the mitral valve and the suction action of the left ventricle. Even in those cases, then, in which the compensation is most perfect, the blood-vessels of the lung are exposed (in consequence of the obstruction in front and the powerful action of the right ventricle behind) to increased pressure and the injurious consequences which may result therefrom.

In stenosis of the pulmonary orifice, compensation is partly due to hypertrophy of the wall of the right ventricle, but chiefly in congenital cases (which comprise the great majority of cases of pulmonary stenosis) to the fact that the foramen ovale remains patent and allows a portion of the blood to avoid the lungs and to make a short cut, as it were, from the right to the left heart. The imperfect aëration of the blood which results from this arrangement is, of course, a disadvantage, and the compensation is, consequently, always imperfect; but if the foramen ovale did not remain open, the circulation could not be carried on with the extreme contraction of the pulmonary orifice which is present in some cases of this description, for the blood which could get through the contracted pulmonary orifice and lungs to the left heart would be inadequate to distend the arterial system, while the engorgement of the venous circulation behind the lesion would be too great to allow of the long continuance of life.

In incompetence of the aortic orifice, compensation is effected chiefly by hypertrophy of the left ventricle, partly by a certain but limited amount of dilatation of the left ventricle, and partly by shortening of the ventricular diastole, and, it may be, by increased frequency of the cardiac contractions. When the dilatation of the left ventricle is just sufficient to accommodate the blood which regurgitates through the aortic orifice, and, at the same time, to receive the normal quantity of blood from the left auricle, and, when the hypertrophy is sufficiently great to enable the cavity to empty itself at each systole, compensation is as perfect as circumstances will allow. The circulation in the parts behind the mitral valve is little, if at all, interfered with; and, provided that the arterial anæmia, due to regurgitation from the aorta during the ventricular diastole, is not great, there are few, if any, symptoms. But in free aortic regurgitation, compensation can never be perfect for any length of time, for a greater amount of dilatation than the theoretical minimum required for the perfect restoration of the balance of the circulation is sooner or later established; this necessitates a proportionate excess of hypertrophy if the cavity is to be completely and satisfactorily emptied, and this dilatation and hypertrophy tend to go on increasing. Again, the increased frequency of the cardiac contractions and the diminished duration of the ventricular diastole prevent the normal amount of blood flowing into the left ventricle from the left auricle, and consequently tend to cause some obstruction to the blood-flow behind the mitral valve and left heart. The sudden and excessive distension of the arterial system is also apt to produce dilatation in the aorta and the vessels arising from it.

In incompetence of the mitral orifice, compensation is effected in a still more complicated manner—namely, partly by dilatation and hypertrophy of the left auricle, partly by hypertrophy of the right ventricle, partly by dilatation and hypertrophy of the left ventricle, and perhaps to some slight extent by diminished duration of the ventricular diastole and increased frequency of the heart beats.

When the dilatation of the left auricle is just sufficient to accommodate the blood which regurgitates through the mitral orifice, and at the same time to receive the normal amount of