and a half of alcohol daily was the limit of safety, but no such rule can be applied, for this amount would be quickly dangerous to some while others would be unaffected by much larger quantities. We are all pretty well agreed that alcohol in reasonable amounts and at proper times is not only harmless but actually productive of good, and it often seems as if those who needed it most are the bitterest opponents of its use. Indeed the case of a total abstainer should be searched into as diligently as the case of the habitual drinker, since abstinence may be due to a knowledge of hereditary taint, to personal intemperance in early life, or to some physical condition which does not go with robust health and long life. But there is a class of risks which all companies agree to consider bad. This is the class of liquor dealers and manufacturers who are brought into daily and intimate connection with alcohol. It is here a little and there a little unconsciously, but without intermission. The tissues are never wholly free from its effect and this is a condition favourable to the slow, chronic change which ends in permanent impairment by the substitution of lower tissue for the higher, connective tissue for the proper vital cells of the organ. There is besides a moral hazard associated with this business, which no one can estimate exactly, and for that reason the after-history of cases rejected for this cause is conflicting. There were twelve rejections for this alone and three deaths.

Many cases are rejected on account of pregnancy, and at first this would seem unjustifiable, seeing that the mortality from childbirth is extremely low, not more than one-half of 1 per cent. This is to prevent women from insuring against that particular contingency and rules are made accordingly. If a woman insures her life under the usual conditions the company lays no restrictions upon her child-bearing, but they will not accept her within two years of marriage nor until she has borne her first child, or until it is pretty clear that she will never have any. This condition led to five rejections.

A large number of rejections arise from causes personal to the medical examiner himself. He puts upon the records remarks that should never go there. He may answer "Yes" to the question of spitting of blood, and then explain that it occurred during pneumonia or from laryngitis, or from "hard work" or "bad weather." One examiner answers "Yes" to gravel, and then explains that it was probably "dyspepsia."

A wrong impression is made upon the director's mind, or if he understands the true situation he cannot issue a policy on any ambiguous application, for at any time it is liable to become a death claim and go before the Claims Committee, who see only the fact and not the explanation which negatives it. The medical director wants no observations, however acute they may be; no reasoning, however subtle, no opinions, however judicial; he wants a nay to be a nay and a yea, yea, without qualification, and above all he does not want a good risk vitiated by "remarks."

REFERENCE.

1 BRITISH MEDICAL JOURNAL, February 5th, 1887.

A CASE OF VARICOSE ANEURYSM OF THE AORTA COMMUNICATING WITH THE PULMONARY ARTERY.

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CASES of aneurysm of the aorta opening into the pulmonary artery are rare, and as the one described below was under observation for some weeks, and the symptoms pathognomonic of this condition are not absolutely settled, I have thought it worthy of record.

A brief summary of the clinical history is that the patient, without obvious cause, began to suffer from shortness of breath, palpitation, and oedema beginning at the feet. There was a marked thrill over the left side of the chest, and a loud, continuous murmur, with systolic and diastolic exacerbations. Later there were marked anasarca, scantiness of urine (varying in quantity from 9 to 16 ounces per diem), orthopnoea, and gradual failure of pulse.

History.—A B., aged 36, was a hotel porter, and had never had any previous illness. He had had gonorrhoea, possibly syphilis, and drank about four pints of beer daily. Three weeks before admission on going up a steep

hill one evening he experienced great shortness of breath; this progressively increased. A week later his feet began to swell, and in another week his abdomen swelled and the urine became very scanty in amount. There was no pain, and no other symptom besides palpitation.

Condition on Admission.—He was well nourished, rather puffy looking, and the skin rather dusky in hue. The pulse 110, not collapsing or water-hammer; there was no capillary pulsation; arteries slightly thickened; respiration 24. The chest wall was cedematous, especially on the left side. There was marked pulsation in the carotid and subclavian arteries; the veins of the neck also showed pulsation, and they filled up from below. The heart's apex was in the fifth space, 2 inches outside the nipple line; the area of dulness extended $\frac{1}{2}$ inch to the right of the sternum and to left to the apex beat; it was enlarged upwards and to the left, there being dulness over the sternum as high as the lower border of the first ribs, and outwards to left in first and second intercostal spaces. There was an area of deficient resonance extending about $\frac{1}{2}$ inch from left border of sternum. A very marked thrill could be felt over the whole of the dull area and slightly beyond it, and also over the subclavian arteries and first piece of sternum; this thrill continued almost throughout the cardiac cycle. The precordial area was bulged, and pulsated everywhere. In addition there was deep-seated pulsation to be felt to the left of the sternum in the second and third intercostal spaces. On auscultation a low-pitched roaring murmur was heard, continuous but varying in intensity forming as it were a background to loud blowing systolic and diastolic murmurs. These murmurs were loudest at the sternal end of the third left intercostal space and fourth left costal cartilage, next loudest in the second intercostal space. During his first week in the hospital the murmur was audible without a stethoscope some inches away from the patient's chest. Below the fourth rib the heart sounds had a galloping rhythm, and the systolic and diastolic murmurs were heard. A loud double murmur was also audible in the back down the left vertebral groove. No tracheal tugging and no laryngeal paralysis were present. There was some fluid effusion into the left pleura. The liver was much enlarged, and showed true pulsations following immediately after the heart beats.

..... Area of Cardiac & Liver Dulness.
 ■■■■ Area of Deficient Resonance, Thrill,
 and Pulsation.
 ○ Area where Thrill & Murmur Plainest.
 XX Sites where Murmur Audible.

Fig. 1.

After-History.—The murmur remained much as above described until his death, the systolic portion being the loudest as a rule; but both systolic and diastolic murmurs varied in this respect, one day one being the louder and another day the other. Both thrill and murmurs became less distinct towards the end as the heart's action grew weaker, and the cardiac dulness to the right of the sternum became more extensive owing to progressive dilatation of the right ventricle. The murmurs could be heard over the great vessels and faintly over the carotids, but not over the brachial or radial arteries. The pulse was never of the water-hammer character, it remained regular, and the rate varied from 100 to 116 until a few days before death, when it reached 140. Respiration 26 to 32; it was easy so long as he was quiet, and there was no orthopnoea until just before the end. Temperature subnormal. There was no cyanosis. A dusky hue being only noticed for a day or two after admission and for three days before death, and even this was not marked. There was no marked anaemia. The urine was very scanty, the quantity passed being from 9 to 14 ounces in the twenty-four hours, once 16 ounces; specific gravity 1022 to 1026; it contained urea 3.5 to 5 per cent., a trace of albumen, a moderate deposit of urates on most days, no sugar, occasionally a hyaline cast. The dropsy steadily increased, and fluid accumulated in the pleural and abdominal cavities; the abdomen was drained, and a large quantity of fluid drained from the legs by means of Southey's trocars,

The anasarca was more marked on the left side of the trunk and limbs on admission, but later was much greater on the right—probably merely due to the position in which he mostly lay in bed.

Post-mortem Examination.—The veins of the neck and upper part of the trunk were very full of blood. Both pleural cavities contained fluid, and the lower lobes of the lungs showed more or less collapse. Just above the anterior cusp of the aortic valve opened out a small sacculated aneurysm, rather oval in shape, and about the size of a bantam's egg; it bulged into the pulmonary artery, and formed a rounded swelling on the right side of the root of the aorta. The wall of the aneurysm was very thin, and it bulged into the pulmonary artery over the position of the right posterior cusp of the pulmonary valve, which was stretched over it and closely adherent to it, so that as a valve it was non-existent; the other two cusps of the valve were somewhat thickened. In the thinnest part of the wall of the aneurysm a perforation had taken place into the origin of the pulmonary artery just below the cusps of the valve; this perforation was irregularly oval, and about the size of a sixpence. The pulmonary artery appeared small, the conus arteriosus was dilated and its wall hypertrophied, being nearly $\frac{1}{2}$ inch in diameter. The aortic valve segments were somewhat thickened and opaque, but came together. Both pulmonary and aortic valves were incompetent to the water test, the former most so. The heart was enlarged, weighing 15 ounces; both ventricles were dilated and hypertrophied, the right the most. Its wall was $\frac{3}{4}$ inch in thickness, and the tricuspid valve admitted six fingers. Above the aneurysm the aorta was somewhat dilated for a short distance, and its posterior wall very thin and slightly bulging. The lining membrane showed patches, mostly smooth, of commencing atheroma. The other organs simply showed the changes of chronic congestion from heart failure.

Treatment chiefly consisted in endeavours to promote diuresis and to

Fig. 2.—About one-third natural size. Taken from the anterior surface of the heart, the right ventricle having been laid open and the incision continued through the pulmonary artery. *a*, cut edge of pulmonary artery; *b* is placed on the perforation in the wall of the aneurysm, the arrow points to the remains of the pulmonary valve segment above it; *c* placed on the aneurysm bulging into conus arteriosus, the rod close to *b* has been passed through the perforation; *c*, cavity of right ventricle; *d*, of left ventricle.

maintain the strength of the heart; all attempts, however, to produce a greater flow of urine were unsuccessful, no drug of the many tried having any effect; amongst these was diuretin, which had a remarkably good effect in Sir Wm. T. Gairdner's case to be referred to.

For the last two or three weeks of his life the aspect of the patient, with the general anasarca and scanty flow of urine, recalled that of a patient in a late stage of renal disease. The diagnosis of a varicose aneurysm was made from the first, chiefly from the very peculiar character of the murmur at the base of the heart, and of the thrill felt over so wide an area; both murmur and thrill recalled these features as seen in two cases of varicose aneurysm formerly under my care. The aneurysm was also obviously at the commencement of the aorta; the difficulty was to decide into what it had perforated. Against the communication being with the superior vena cava were that in these cases the onset is generally

marked and sudden; there is great cyanosis, together with oedema and distension of the veins of the upper part of the body; whether the chest wall is oedematous depends upon the position of the obstruction, whether above or below the point of entrance of the azygos vein into the vena cava; an abnormal area of dulness, if present, would be behind or to the right of the sternum (first piece), and thrill and murmur would either be over the sternum or to the right of it, or at any rate not so distinctly to the left of the sternum as in the above case. In the absence of these signs it seemed tolerably clear that the varicose aneurysm was not one of the aorta and superior vena cava, and during life it remained uncertain into what vessel it would be found to have opened, although the physical signs and the known relative frequency of the direction of such perforations made it probable that the aneurysm had opened into the pulmonary artery or into the right ventricle or its conus arteriosus. The site of the murmur and thrill, and position of extension of area of dulness upwards and to the left of the sternum, were certainly suggestive of communication with the pulmonary artery, but at this time I was not aware that such a communication could take place without causing marked cyanosis, and this was entirely absent.

Fig. 3.—Slightly larger than Fig. 2. *e*, cavity of left ventricle just below aortic valve; *f* placed on the centre of the opening of the aneurysm from the aorta, the opening is just above the aortic valve, the two rods keep it open; *g g* indicate the margins of the aneurysm; *h* placed next the perforation (faintly indicated) at the bottom of the aneurysm; *i*, cut edge of aorta; *m*, surface of left ventricle.

Walshe,¹ after describing the symptoms in the diagnosis of a sudden communication between the aorta and the pulmonary artery or right ventricle (amongst which he mentions a powerful systolic thrill limited to the second and third interspaces close to the sternum), goes on later to say that "habitually sudden, actually instantaneous in its occurrence, the communication may, on the contrary, be slowly effected..... If the communication arise in this gradual fashion, the attendant symptoms may be wanting in the elements of sudden cardiac shock and respiratory distress." This statement was certainly borne out by my case.

Sir Wm. Gairdner² publishes a full report of a case which corresponds almost exactly with the one above described, and judging from the excellent plates, the aneurysm was of about the same size, and opened into the pulmonary artery in the same position. Sir W. Gairdner notices especially the re-

markable euphoria or complacent optimism of his patient even when the subject of advanced general dropsy, and I should have added that my patient was always cheerful and contented, and did not appreciate the grave nature of his illness.

In Sir William Gairdner's case the patient, aged 33, had suffered from shortness of breath for twelve months; the pulse "was fairly characteristic of aortic regurgitation"; there was a remarkable tendency to muttering delirium, especially at night, with more or less albumen in the urine, and no definite dull area over the chest apart from the extension of the cardiac dulness. His case corresponds with mine in the appearance of dropsy one week before admission to hospital, and in the absence of other symptoms than shortness of breath and palpitation; in the absence of hæmoptysis, marked cyanosis, and of orthopnoea until shortly before death; in well-marked increase of the transverse dulness of the heart, especially of that due to dilatation of the right ventricle, in high degree of general dropsy, enlargement of the liver, and moderate pulse-rate (98 to 116) until near the end. He describes the murmur as being extremely loud and all but continuous over the right ventricle. "Over the aortic area V.S. and V.D. murmurs very much of the usual characters in aortic disease can still be separately distinguished; but..... the loud, almost roaring, hollow murmurs heard midway between the left nipple and the middle line are such as to confuse the ear as to the rhythm."³

It will thus be seen that with some minor points of difference the correspondence between the symptoms in the two cases is remarkably close, the chief points to be borne in mind for the diagnosis of future cases of gradually-established communication between the aorta and pulmonary artery being the gradual onset, with shortness of breath, palpitation, and dropsy, the peculiar murmur and thrill heard and felt at the base to the left of the sternum (with or without deep-seated pulsation and a small area of dulness in the second and third left intercostal spaces), and possibly also over the right ventricle; enlargement of the heart, and especially dilatation of the right ventricle; rapid development of general dropsy, with scanty excretion of urine; absence of marked cyanosis and orthopnoea; moderate increase in the rate of the pulse which may be, but is not always, of the regurgitant type; great engorgement of the liver, and possibly a sense of well-being relatively to the serious condition present.

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¹ *Diseases of the Heart*, Fourth Edition, p. 518. ² *Glasgow Hospital Reports*, 1899, p. 1. ³ *Loc. cit.*, p. 6.

WHAT IS A DISEASE?

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THE meaning attached to the word "disease" has undergone a process of evolution during many centuries, while the science of medicine has emerged from darkness into light. The current conception of "disease" is indeed, and has always been, the central thought in the medical world. The time has come to give the word a definite value and meaning in accordance with the position the science of medicine now occupies. Not probably till her sister science bacteriology

came to the help of medicine was it possible to do this; the task is now, however, simple.

The most direct method will be to take an example from the group of germ diseases. Under certain conditions the typhoid microbe becomes parasitic in man; pathological changes occur in consequence, which we include under the name of typhoid fever. In the absence of this particular microbe this disease, according to present knowledge, cannot occur. Here is a self-defined disease, and from it we deduce a general definition:

The sum total of the pathological consequences resulting in a patient from the interference with his physiological state by a disease cause.

When the disease cause is of extrinsic origin, as in typhoid, the resulting disease is a self-defined entity. Lead poisoning and heat stroke may be cited as examples representing other classes of diseases due to causes of extrinsic origin. There is some apparent, but no real, difficulty in defining the cause when this is of intrinsic origin, provided always that we make up our minds to confess ignorance where it exists instead of concealing it in a cloud of words. Lithæmia, gout, and goutiness form at present a very vague symptom group. Define the cause as a sluggish metabolism resulting in ill-prepared waste products, and they are bound into a definable and treatable "disease." Some forms of insanity, hysteria, chorea, and asthma are also caused by function failures of other kinds, and the failure of a function is just as definable a disease cause as the invasion by a microbe. The unphysiological stress that fractures a patella, ruptures an aortic valve, or that produces "athlete's heart," emphysema, or the bone changes of laborious occupations is no less real or definable as a cause of disease than the ingestion of alcohol or lead. Incomplete development and prolonged wear and tear are accountable for other groups of diseases, properly so-called, that are familiar to us all. It is only necessary to cultivate a definite habit of mind to make the method that would result from the adoption of the above definition of disease perfectly plain.

At the risk of being thought presumptuous, I would say that it is on this foundation—the adoption of a recognised method—that the teaching and practice of medicine must in the immediate future be remodelled. Lengthy explanation and a multiplication of examples are equally unnecessary in addressing those whose lives are devoted to the study of medicine. The table below gives at a glance a rough idea of the shape a scientific classification of diseases would take.

There are clearly some diseases, though not a great many, the causation of which is still in doubt. Carcinoma is an example of one such class. Cirrhosis of the kidney—when not part of gout, lead poisoning, or other already-defined disease—cirrhosis of the thyroid leading to the symptom group, myxœdema, the changes in the cord resulting in idiopathic spastic paraplegia, and many other similar conditions in various organs, are probably all parts of toxæmias, whose true causation will, let us hope, soon be cleared up. It is in such cases that the "frank confession of ignorance which takes us a long way towards enlightenment" would be such an advantage and such a relief.

At present, the lack of one uniform method in medicine leads to an utter want of uniformity in textbooks and all other means of instruction. It is little exaggeration to say that "diseases"—the current coin of our science—are at present arranged and valued rather to suit the speciality of the

Diseases are due to Causes of Intrinsic, Extrinsic, and Undetermined Origin.

Intrinsic.				Extrinsic.			Undetermined Origin.	
Wear and Tear.	Incomplete Development.	Function Failure.	Unphysiological Stress.	Parasitic.	Non-parasitic Matters introduced into the Economy.	Deleterious Physical Interference.	Tissue Overgrowth.	Toxæmias of Unknown Nature.
Atheroma, chronic senile general paralysis	Idiocy, cleft palate, congenital heart defects	Gout, insanity, hysteria, lateral curvature	Writer's cramp, athlete's heart, transverse fracture of patella	Tuberculosis, malaria, hydatid disease	Alcoholism, plumbism	Wounds, heat-stroke, frost-bite	Carcinoma, lipoma	Spastic paraplegia, anterior poliomyelitis, cirrhosis of kidney.