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.

REFERENCES.

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.

teacher than to aid the comprehension of the student. Our pathological museums are chaotic, and therefore repellent to the student. Medicine is a science without a method, and until our leaders adopt one we shall not take the place that belongs to us.

THE RELATION BETWEEN CAUSE AND EFFECT IN DISEASE.*

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In considering the pathology of diseases we often have occasion to use the term "specific." It is well, then, to ask ourselves what this term means. A "specific disease" is literally a disease which has certain definite characters which distinguish it from all other diseases. There are two ways in which a disease may be specific.

1. It may be due to a specific causal agent.
2. It may be characterised by specific lesions.

It is obvious that our conception of any disease will differ according to which of these criteria we adopt as our basis of specificity.

For example, let us consider tuberculosis. If we adopt the anatomical basis—that is, if we consider tuberculosis to be a disease characterised by the presence of certain definite anatomical lesions called tubercles, we must include in this term, not only those cases in which the tubercle bacillus is the causal agent, but also other cases due to other organisms such as aspergillus, and we must exclude all diseases not characterised by these lesions. If, on the other hand, we adopt the etiological basis, we must exclude all cases which are due to organisms other than the tubercle bacillus, and we must include cases in which the anatomical tubercles are not present; for the tubercle bacillus occasionally gives rise to a diffuse instead of a nodular inflammation.

Thus the term "tuberculosis," which originally had a purely anatomical significance, has now come to have a meaning which is purely etiological, and in consequence those diseases which are characterised by anatomical tubercles, but which are not due to the tubercle bacillus, have been designated by the unsatisfactory term "pseudo-tuberculosis."

The same change is now, unfortunately, taking place in the meaning of the term "pneumonia." This originally meant a disease characterised by the anatomical condition of inflammatory consolidation of the lung. Now, however, there is a growing tendency to include pneumonia among the specific infective diseases, that is, among the diseases due to a specific micro-organism. If this is done we shall have to describe pneumonia, not as a disease characterised by an inflammation of the lung, but as a disease which is the result of infection by the diplococcus pneumoniae, and we shall have to include in it certain cases of broncho-pneumonia, pericarditis, meningitis, otitis, empyema, etc., any of which conditions may be caused by the diplococcus with or without the characteristic inflammation of the lung. Judging from the analogy of tuberculosis we may expect in the future to read about "pneumonia" of the meninges, ear, pericardium, etc., and we may also find the term "pseudo-pneumonia" introduced to describe those diseases characterised by inflammation of the lung, but not due to the diplococcus pneumoniae.

It is thus a great mistake to give an etiological significance to a term the significance of which is purely anatomical. It is sure to lead to great confusion in medical nomenclature. With regard to this question of specificity I should like to lay down the following two complementary laws.

1. No specific causal agent always gives rise to characteristic lesions.
2. No specific lesion is always produced by a single definite causal agent.

Besides the examples given above, I will give the following:

1. The diphtheria bacillus usually gives rise to a fibrinous inflammation of the fauces or larynx with the production of membrane. Sometimes, however, the membrane is wanting, and instead of a fibrinous there is a gangrenous or oedematous inflammation. On the other hand, fibrinous inflammation of

the fauces may be due to other organisms than the diphtheria bacillus.

2. Congenital syphilis, if it affects the liver, usually gives rise to a pericellular cirrhosis. It may, however, give rise to other forms of cirrhosis, or to gummata. On the other hand, pericellular cirrhosis of the liver is occasionally found in patients presenting no other evidences whatever of congenital syphilis.

3. The streptococcus pyogenes in the subcutaneous tissue may give rise either to a suppurative or a non-suppurative inflammation, and suppuration may be produced by organisms other than the streptococcus.

4. Chronic alcohol poisoning, if it affect the liver, usually gives rise to a multilobular cirrhosis. It may, however, give rise to other forms of cirrhosis, or to fatty degeneration without cirrhosis. On the other hand, multilobular cirrhosis may be due to other causes than alcohol poisoning.

These examples might be multiplied indefinitely, but I have given a sufficient number to demonstrate the general truth of the two laws given above.

So far I have considered only anatomical lesions. It remains to consider those lesions which may be termed functional, such as alterations in secretion or motor activity. At first sight it appears doubtful whether we can apply the laws to specific alterations in functional activity, but, on closer investigation, it will be seen that, although there are apparent exceptions, yet there are numerous cases in which the laws hold. At any rate, the second law would appear to hold good in all cases.

5. Morphine, while usually producing narcosis, sometimes produces excitement. On the other hand, narcosis is produced by many agents besides morphine.

6. Lead poisoning, if it affect the central nervous system, may give rise to either excitement or dulness.

As an apparent exception to the first law may be mentioned the action of tetanus toxin on the central nervous system. It is quite possible, however, that the tetanus toxin may sometimes reach the central nervous system without producing the characteristic convulsions, since an examination for the tetanus bacillus is only made when the characteristic convulsions are present.

We have now to consider the following question, Why does the effect produced by a specific causal agent vary in different cases? The answer is to be found in the fact that no disease is dependent on one cause alone. The factors concerned in the production of a disease can be divided into (1) the essential factor, and (2) the adjuvant factors; the essential factor being that without which the disease could not occur, and the adjuvant factors being those which assist in production of the disease, but which in themselves are not individually essential.

In those diseases which are due to a specific causal agent, this agent itself is the essential factor, the adjuvant factors being those usually known as the predisposing causes. It will be found that, granting the access of the causal agent to the body, the adjuvant factors can be reduced to two in number: (1) The locality to which the causal agent is applied; (2) the relative virulence (or dose) of the causal agent. By "relative virulence" I mean the virulence of the causal agent considered in relation to the susceptibility of the patient.

1.—The Locality to which the Causal Agent is applied.

- (a) An irritant applied to an epithelial surface usually gives rise to a catarrhal inflammation—for example, inflammations of the lung due to inhalation, or to the spreading of an inflammation along the air passages, usually have the characters of broncho-pneumonia.

- (b) An irritant applied to a surface covered by endothelium usually sets up a fibrinous inflammation, such as is seen in pericarditis, etc.

- (c) An irritant acting on connective tissue tends to produce a suppurative or an indurative inflammation, etc.

2.—The Relative Virulence (or Dose) of the Causal Agent.

The intensity of any disease varies with the virulence of the causal agent and with the susceptibility of the patient. Thus an irritant of low relative virulence acting on connective tissue gives rise to a productive inflammation, if the virulence is greater the result is suppuration, and if the relative viru-

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