of rheumatic fever, came to the hospital for pain and swelling of the left breast. The swelling had lasted a week, and there was tenderness and some hardness on examination. This symptom rapidly disappeared with the aid of mild aperients and warm applications.

CASE II.

Anne M., aged 10 years, was under me at 7 suffering from rheumatism, with arthritis, chorea, and endocarditis. She made a good recovery, and had kept apparently free from any relapse. Her mother now brought her for pain, tenderness, swelling, and hardness of the left breast. There was definite evidence of her previous attack of rheumatism in an enlarged heart and a double mitral murmur. Her temperature was not raised, and the condition rapidly subsided.

CASE III.

Florence M., aged 13 years, had been under my observation through two attacks of chorea, one at the age of 9 and another at the age of 10. There had been dilatation of the heart and arthritis, but a long rest in the hospital followed by a convalescent home had brought about complete recovery. Although over age, she was brought to me in 1909 with pain, tenderness, and swelling of the left breast. She had been free of chorea since her second attack, and had no symptoms of rheumatism.

CASE IV.

Alice P., aged 10 years, had been under me at intervals for some years suffering from chorea, endocarditis, and arthritis. In October, 1910, she again came back suffering from rheumatic pains, with a well-compensated mitral lesion, and complaining of pain and tenderness of both breasts, which were harder than normal.

Three other cases gave almost precisely the same history. I am not, perhaps, justified in quoting these cases under the title of my lecture; but they have interested me because later in life I think dysmenorrhoea of a painful kind is not infrequent in the rheumatic.

EPISTAXIS.

Lastly, I should like to direct your attention to epistaxis in rheumatism. My attention was first called to this by a paper of Dr. Sydney Phillips upon the subject, and since that date I have learnt to appreciate its reality. Epistaxis may occur at the commencement of an attack, during the illness, or as incident in the more quiescent phases of a long rheumatic history. Exceptionally it may be very severe, but as a rule it is moderate in degree and not of any moment in itself. I would add that it is not by any means necessarily associated with obvious nasal disease, although I hardly think we lay sufficient emphasis upon diseased conditions of the nose in rheumatic children as a possible source of infection, in spite of the writings of Dr. C. R. Stratton.

In this lecture I have dwelt a good deal upon haemorrhages in rheumatism, and I would add further to them the occasional occurrence of haematemesis. This I have only seen at present in grave heart disease, and it is possibly only a mechanical event, although in some examples it has also been associated with profound toxæmia and extreme anaemia.

It will need another lecture at least to complete the remarks that I have to make upon other of the less common occurrences in the rheumatism of childhood, and I shall hope to continue with them when the next opportunity arises.

DR. ROBERT WILLIAM COE, of Clifton, consulting surgeon to the Bristol General Hospital, who died on August 11th, 1910, aged 89, left estate valued at £28,612 gross, and £27,485 net.

THE Secretary of the Medical Missions Department of the Society for the Propagation of the Gospel in Foreign Parts asks medical men and women of Great Britain, who may not wish to preserve medical journals after reading, to consider the needs of the medical missionaries in the foreign field. He states that any journals sent to Miss B. Gambier Parry, Bacton, Wokingham, Berks, will be gratefully received, and forwarded without delay to a missionary doctor.

Clinical Lectures ON KIDNEY PAIN.

BY
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[With Special Plates.]

LECTURE I.

GENTLEMEN,—Although expert radiography has conclusively proved that calculous disease of the kidney is comparatively rare, the profession cling to the belief that any severe pain experienced in the region of that organ is due to stone. I suggest, therefore, that you consider with me this session the varieties of kidney pain and their approximate causes, and for the first step we will discuss that form most frequently encountered—the pain of a distended renal pelvis. It is probable, on physiological and on clinical grounds, that the kidney is only able to feel in two areas—the pelvis and the neighbourhood of the true capsule. Parenthetically, I may remind you that patients are very occasionally met with who do not have sensation in either area, and then crippling and progressive disease of the kidney may be painless from start to finish. As examples you may call to mind the hydronephrosis the size of a small football I operated upon a short time ago, the patient having no inkling that he was a sick man until

a week before he was admitted here, when he noticed the increase in the size of his belly. Then, again, the largest stone I have removed from the kidney was fifty years in the organ without causing any pain.¹ Generally, however, there is pain in surgical kidney disease characteristic both in degree and place.

I start with two aphorisms which I have taught here in the operating theatre since 1900. The

commonest structural change accompanying kidney pain is a distended pelvis; the commonest cause of kidney pain is dilatation of the renal pelvis.

To prove my contention, I must refer to certain salient symptoms which afford a valuable clue to the existence of surgical disease of the kidney pelvis or cortex.

1. EXCESSIVE THIRST.

If you eliminate the thirst produced by certain poisons, such as sugar, alcohol, opium, chloroform, you will find that a consuming thirst is a danger signal in urology, because it is the first decided and noticeable symptom of uraemia, in that it appears before any alteration in the urine. Sir A. Clark,² one of our most revered teachers here, read a paper in 1884 on "Catheter Fever." He noticed that death occasionally occurred after using the catheter in cases of enlarged prostate, and he contended that this untoward result generally occurred in persons who appeared in perfect health, but whose urine was low in density. He considered such patients to have "renal inadequacy." Of course we had known for long that "renal inadequacy" was merely another term for a low form of interstitial nephritis, induced by prolonged back pressure exerted by an unrelieved bladder, and marked by filtration of water rather than the excretion of urinary salts; but we did not then know bacterial invasion was the fire which fell on the prepared "tow"—the weakened kidney. Even now the profession at large does not realize that this renal inadequacy is marked by *excessive thirst as an onset symptom*. This thirst is mostly due to diuresis—noticed or unnoticed. The symptom commences when the specific gravity of the urine is 1010, and it becomes very pronounced when it has sunk to 1004. It is then accompanied by a graver symptom of depreciated kidney

DESCRIPTION OF SPECIAL PLATES.

Fig. 1.—To show upper and lower polar aberrant arteries, and the area they affect.

Fig. 2.—Aberrant vessels to the lower pole underrunning the ureter and dilating the pelvis (Newton Pitt).

Fig. 3.—A one finger-tip pelvis (normal).

Fig. 4.—Kidney in section. A one finger-tip pelvis in which one or even more papillae are touched, showing the commencement of the pelvic dilatation.

Fig. 5.—A two finger-tip pelvis, showing elongation of kidney and some dimpling of cortex.

TO ILLUSTRATE MR. HURRY FENWICK'S LECTURE ON KIDNEY PAIN.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

TO ILLUSTRATE MR. HURRY FENWICK'S LECTURE ON KIDNEY PAIN.

Fig. 6.

Fig. 8.

Fig. 7.

Fig. 9.

Fig. 10.

function—anorexia and sickness. It must also be remembered that the same symptom may be induced by inflammatory processes, acute or chronic, affecting the interstitial areas of the kidney. The thirst in this instance is due to the absorption of toxins arising from destruction of the inflamed area, and it is not, then, necessarily marked by diuresis or fever. If now we find excessive thirst with low specific gravity urine, we look with suspicion on the kidney function, and expect to find some interstitial pressure of that organ, or some recent inflammatory focus in its structure. Both these conditions may exist as the result of a distended pelvis.

2. THE POSTURE ADOPTED BY THE PATIENT IN SLEEP.

Very few sleep, as Confucius says, "on their backs like dead men"—most have a favourite side. I do not enter into the fascinating and far-reaching study of the predisposition to unilateral disease promoted by this habitual lateral attitude, I merely want to draw marked attention to the clue which a sleep posture may afford us in locating disease to the kidney.

In 1896⁸ I raised this point and suggested that when a patient was forced to lie on a painful kidney in order to relieve pain, this attitude indicated a stone in the cortex of that kidney. I was wrong; I had only operated on 80 kidneys. I now know that a large percentage of patients suffering from pain in one kidney can only get ease at night by lying on that kidney—that this posture is adopted in order to keep the kidney at rest and thus prevent the cause of pain, whether it be a stone or a dilated pelvis, from moving with the kidney in respiration; also that this posture denotes an absence of cortical inflammation. I know, moreover, now that if the cortex be much inflamed and therefore adherent to the fatty capsule (corticitis), and in proportion to the acuteness of that inflammation, the patient *cannot* sleep on that kidney, but must turn to the opposite side.

Hence the axiom: If the patient is forced to sleep on the aching side, the kidney is still movable, and the pelvis is locally irritated. If the patient *must* rest on the unoffending side, the aching kidney is fixed and its surface is inflamed (corticitis). As a corollary, one of the first symptoms of return to health after removal of a stone from the kidney is that the patient can sleep easily on the unoffending side, and the marked sign of convalescence after a sharp attack of coli-bacillary nephritis of one kidney, in which, of course, the cortex has been involved, is that the power returns of sleeping on that kidney.

3. INTERMITTENT DIURESIS.

Aphorism: Intermittent diuresis marks some dilatation of the renal pelvis. If we are able to eliminate a dietetic and a cerebral influence on the urinary secretion, we possess an important clinical clue to disturbance of the renal vasomotor action in the filtration of urine, when marked intermittent diuresis occurs. A fair proportion of patients with renal pain complain of bouts of passing large quantities of water. I do not here concern myself with how this comes about. I press the remark home upon you that these rushes of urine have a distinct effect upon the capacity of the renal pelvis. Read carefully the pathological findings of cases of so-called diabetes insipidus in which large quantities of urine are secreted: you will notice how often the renal pelvis and ureters and even bladder are dilated and their walls thinned. Judging by analogy, the large amount of work they are called upon to do has first hypertrophied and then thinned these cavities. Examine the pathological findings of a series of nephrectomized patients who have died, say, five years after their operation, you will find the pelvis and ureter of the working kidney are dilated. Given, then, a kidney which has bursts of secretory activity, and you will find operatively that its pelvis is always perceptibly dilated; and this I state on fifteen years' careful scrutiny of the renal pelvis in all cases I have operated upon.

From this subject we pass naturally to the next: the vasomotor irritation of the arterial supply of the kidney, producing intermittent diuresis.

We know that the amount of urine secreted rises or falls with increasing or diminishing fullness of the renal artery; even moderate compression of the artery in animals is followed by distinct reduction. Now withdraw your mind for a minute from the main source of arterial blood—the renal artery. Fix it on a small aberrant artery. We meet with these running either to the upper pole or to the lower pole (Fig. 1). Now mark this: They are not uncommon. The surgeon who works with a head lamp—and I have used artificial light in this way in every case for fifteen years—at once detects the aberrant artery and notes the condition of the cortex to which it runs. There is, I believe, a distinctive group of symptoms induced by the tug of an aberrant vessel upon the upper pole. I may say, if a kidney which has an aberrant vessel to its upper lobe becomes loose and intermittently drags upon this vessel, alternately stretching it and slackening it, that the secreting tubules of the area of the kidney which it supplies (Fig. 1)—and luckily it is a small area—become greatly altered in health, and the arterial anaemia may even be so marked as to induce corticitis, and thereupon pain, which is, according to the site involved, referred roughly to the liver or splenic area. I have also noticed attacks of polyuria and even thirst traced apparently to the influence of tension, or torsion, of these aberrant polar arteries.

We need not deal with the aberrant artery running to the lower pole of a movable kidney; it produces similar changes in the pole, and a specific form of renal pain. If

we ligate these vessels and divide them, we render a small area anaemic and softened; but it finally scars, the pole becomes adherent, and the patient loses the characteristic pain. But, should the aberrant artery pass over or under the ureter near its pelvo-ureteric junction, and any sudden or gradual drop of the kidney strain the line of the artery taut, it not only starts inflammatory changes in the cortex, but—as we shall come to it

immediately—it also induces dilatation of the pelvis, and this will be found a cause in 16 per cent. of all cases of primary pelvic dilatation.

To sum up now these prefatory points. Thirst, with or without diuresis, rouses the suspicion that renal changes, generally bilateral, are present, and that there may be back-pressure upon the parenchyma of the kidney. Sleep posture may indicate a movable kidney with an aching pelvis if the painful side is slept on, or an inflamed fixed cortex if the painful side is avoided. Intermittent diuresis points to a dilated renal pelvis, and some interference by tug or torsion with the vasomotor influence of the renal vessels. These clinical findings, taken in conjunction, are of real inferential value. Now for our operative points.

THE OPERATIVE POINTS OF DILATED RENAL PELVIS.

I was much impressed in 1894 by a communication which Dr. Newton Pitt⁴ made to the Pathological Society on the occasional presence of renal aberrant vessels, and their power of producing hydronephrosis (Fig. 2). The subject, it is true, was not original, but it had never been so clearly demonstrated. From that day to this I have never operated on a kidney without examining the capacity of the renal pelvis of the kidney I was operating on. If I found the slightest dilatation, I examined visually the pelvis and the ureter, and tried to determine the cause of the increased capacity of the pelvis. I was astonished to find that fully 80 per cent. of cases of marked renal pain, not caused by stone, tubercle or cancer, was characterized by the presence of a dilated pelvis. To me it was the hall-mark of renal pain, and I used to gauge the size of the pelvis by the number of finger-tips I could introduce easily into the renal sinus by rolling in that structure. Every house-surgeon I have had since 1894 has felt and confirmed this condition.

DESCRIPTION OF SPECIAL PLATES.

Fig. 6.—Kidney in section. A two finger-tip pelvis, showing flattening of papillae.

Fig. 7.—Pelvo-ureteric junction kinked (c).

Fig. 8.—A three finger-tip pelvis, showing much hollowing of pelvis.

Fig. 9.—An aberrant leash of vessels running to the lower pole under the insertion of the ureter—severely bending the pelvo-ureteric junction.

Fig. 10.—A mal-inserted ureter running along the back of the pelvis, entering this cavity about its middle, after traversing its coats obliquely.

I learnt that the pelvis which admitted one finger was about normal (Fig. 3); if, however, it permitted a search of even one papilla (Fig. 4) it was *being* distended; the ureter in such a case would be *fixed*, but the junction of this with the pelvis would be softened and would easily bend on axial rotation of the kidney. The true capsule would be thin and the fatty capsule would be but slightly attached to the true capsule. I learnt that if two finger-tips could be admitted into the pelvis the kidney would be elongated (Fig. 5), the calyces would be wider than normal, and the papillae would be felt easily (Fig. 6), and their points would be flattened—all conditions pointing to back pressure—the pelvo-ureteric junction would be grooved by a kink (Fig. 7). The true capsule would be thicker than normal, but not adherent, and the fatty capsule firmly adherent to it in patches. Very soon I could tell from a mere glance at the cortex or a touch with the finger on it as to what degree the pelvis was dilated. If the surface of the kidney was deeply divided into lobules like a mammalian kidney, I knew it would dimple here and there under the pressure of my fingers, that the kidney would be much elongated, the cortex thinned, the true capsule more adherent, and the pelvis would admit, when the urine had been aspirated, three or four finger-tips (Fig. 8). I called these "canoe-shaped kidneys," from their fancied resemblance to that form of boat. I very soon learnt, moreover, that with a canoe-shaped kidney I was bound to find either a fixed mechanical obstruction to the pelvic orifice, just where the ureter joins the pelvis, or a ureter greatly dilated from some form of obstruction near the bladder. We will consider the latter in a lecture later on, and glance slightly at the obstruction at the pelvo-ureteric junction. This was either in the form of under-running bow-stringing abnormal vessels, 16 per cent. (Fig. 9), or a misplaced "malinserted" ureter, 8 per cent. (Fig. 10). I have never found a "congenital stricture or valve." I do not believe such exist, and I think the term will in course of time disappear from your textbooks.

My clinical inferences and operative interventions were as follows: A "one finger" capacity meant a very slight dilatation, and I searched for an upper polar artery to cut across, so as to prevent intermittent diuresis, and fixed the kidney to prevent any vessel drag.

With a "two finger," I tried to splint the weakened bend at the pelvo-ureteric junction with fat packing, with more careful fixation of the kidney, and by placing it more obliquely than normal (76 per cent. of cases).

With the more marked hydronephrosis, due to aberrant lower polar artery and vein, under-running or over-running the ureter at the pelvo-ureteric junction—16 per cent.—I merely cut the vessels across between ligatures, and I was well satisfied to see the inner (aortic) ends disappear with a jerk, like a conjurer's coin, and the pelvic cavity commence emptying itself with a worm-like action. I then fixed the kidney.

If my patient came late—that is, with an infected pelvis, pyonephrosis, and an attenuated rotten parenchyma—I took the shell away.

If I found a malinserted ureter which ran between the coats of the pelvic cavity, I either cut its orifice out and stitched it into a hole in the pelvis lower down, or I repaired as in pyloroplasty.

You notice I say nothing about large hydronephrosis—feelable, and visible to the clinician—I do not include them; they are diagnosable, and form a clinical group to themselves. Of course these observations I have outlined to you were the first steps. They are charmingly crude and unscientific, but they saved many a dismal failure in the operative treatment of aching kidneys, and I advise you to follow them unless you are in a position to do expert work. Nowadays an expert can generally tell you before the operation, if the patient be a female, not only the capacity of the pelvis, not only the exact cause of the pain, but also the nature of the pelvo-ureteric junction obstruction, and whether rotation exists rather than a drop in the organ.

During the last decade, led by Kelly of Baltimore, the capacity of the renal pelvis in women has been gauged by means of the ureteric catheter. It can be done in men, but it is not so easy. The pelvis is injected through the catheter, and directly pain is evoked the amount of fluid used is measured, and the character of the pain caused by the distension is noted.

There is some difference of opinion as to the normal capacity of the renal pelvis, but it can be said with certainty that, when it contains over 2 oz., hydronephrosis exists, this being usually accepted as the proper standard. Kelly and others, however, affirm that anything over 3 drachms indicates a pathological condition, and Freeman has found, in numerous instances, that pain is produced in the normal kidney by the insertion of this amount of fluid.

Still more recently, as you will see by these beautiful radiographs, the capacity of the renal pelvis has not only been gauged, but also portrayed by injecting into it a solution of 10 per cent. of collargol (Voelcker, Braasch), or colloidal silver oxide (Uhle, Pfabler, Makinney and Miller), and then radiographing the patient (pyelography).

AXIAL ROTATION OF THE KIDNEY A CAUSE OF URETERIC KINK.

In my earlier work I suspected that much of the simpler forms of bent or kinked ureter was the net result of three causes: Some increase in the vertical mobility of the organ, some softening of a dilated pelvis, and some axial rotation of the kidney. I did not possess then, as I do now, the means of estimating accurately the amount of vertical mobility and the existence of axial rotation of the kidney before operation, so that my material is small, but I have no doubt my earlier convictions are correct.

THE CHARACTER OF THE PAINS INDUCED BY DISTENSION OF THE RENAL PELVIS.

The pains—and I use the plural advisedly—are varied. Let us disassociate them and then combine them in our own mind for the clinical picture as a whole:

First, the pang of the sensitive orifice of the pelvis. This opening, like the other urinary orifices—namely, that of the lower ureter, of the bladder, of the membranous canal of the urethra—is highly sensitized. The pain which marks its mechanical abrasion, closure, or distension is felt in the hollow of the loin; just where an angle is formed between the last rib and the erector spinae. It is quite localized, and can be covered by the point of the thumb; it is stabbing and severe—often enough to make the patient vomit—but it merges into the discomfort which is due to the distension of the pelvis. This latter, if the pelvis is uninfamed, is a dull aching, diffused and covered by the hand placed on the same loin area—ileo-costal angle. As the distension increases so that the capacity of the pelvis is reached, the symptom most marked is that of abdominal distress—pain in the belly, generally over the front of the same side—and finally sharp renal colic, with all its characteristic symptoms. The attack, however, is shorter than real renal colic, for the pelvis empties gradually; the pain is under the control of hot application or hot drinks, or at most a little morphine suffices.

There is also a drag pain on the vessels in between the attacks—this is very definite in certain cases—but relieved in an hour usually by lying down, returning on rising. This pain is due, however, merely to the mobilized organ, and is relieved by a kidney belt.

With an infected pelvis a new form of pain is added, and when the inflamed pelvis is occluded the pain is agonizing. In addition to the local pain, the radiations go down to the groin and leg, and the relief from rest, time and morphine is less, for it depends on the subsidence of the actual attack of pelvitis. Generally with the infected renal pelvis, bladder symptoms are present also, such as frequency of urination, pain at the urinary outlet, and others, but we will consider those in another lecture.

As the pelvis gets larger, and the distension is more marked, we may get a characteristic sleep posture. The patient sleeps on the painful side, violent thirst is occasionally complained of, although only one kidney is affected.

You might remark: We do not need these extra clinical signs, the kidney must now be palpable and visible on the belly. By no means; if it were, conservative surgery would be too late; the kidney, though it has a distended pelvis, should be still unfeelable to the diagnostician. This is the time to interfere to save the kidney. This is also the dangerous time for the patient who, because no stone is seen in the radiograph, is advised to leave the treatment to medicine—a danger which Sir Henry Morris has so

ably demonstrated recently, and which I trust this lecture will also emphatically reflect.

Let me speak quite plainly. Any large, easily-felt hydronephrosis is the evidence of failure; any large, easily-felt pyonephrosis is the evidence of culpable neglect—both might have been avoided. Both will need mutilation, not surgery, for their cure, and if mutilation then curtailment of strength and life, in the place of that which early conservative surgery has in its power to confer.

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An Address

ON THE

PREVENTION OF HEART DISEASE.

DELIVERED BEFORE THE ULSTER BRANCH OF THE
BRITISH MEDICAL ASSOCIATION.

BY

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OF THE BRANCH.

[DR. CALWELL, after thanking the members for electing him, and referring to the necessity of brevity and condensation in their proceedings owing to the very large amount of useful and important scientific work to be gone through, proceeded as follows:]

Many years ago I brought before the Ulster Medical Society a short paper on "Some Possibilities of Preventive Medicine." I was fresh from the study of Koch's work on tuberculosis, and citing what had been done in the past with regard to other diseases, I ventured on the hope that tuberculosis in time would slowly diminish to vanishing point as a national disease. To-day I might point to the tropics as well as to our own country as illustrating further possibilities and larger hopes. At home, amongst the most recent work, respiratory affections are being approached by the clinical bacteriologists, and a scientific knowledge of their cause may reveal the path of prevention or of cure. Cancer is slowly and stubbornly yielding up her secrets; but the darkest blot is undoubtedly the increase of insanity and weak-mindedness. Despite the repeated warnings of our alienists, the public pay but little heed, and were it not for the more advanced educationalist the outlook would be indeed black.

In this light, a short investigation into the possibilities of the prevention of heart disease might be both instructive and interesting; and as I shall have to deal with some rather tiresome statistics I shall throw them upon the screen, and both save your patience and more clearly demonstrate the results.

First, we must study the extent of the problem. Out of a total number of 520,456 deaths in 1908 in England and Wales, 49,891, or 9.59 per cent., occurred from heart disease, or a proportion of 1 death in 10.41. In Ireland out of 76,891, 9,020 were due, or a proportion of 1 in 8.8; in Ulster 3,396 out of 28,696, or 1 in 8.4. We see, then, that heart disease is returned to the Registrar-General as a much commoner cause of death in Ireland than in England; that in Ulster it is slightly greater than the average of all Ireland.

TABLE I.—SHOWING NUMBER OF DEATHS DURING 1908 IN
ENGLAND AND WALES.

Causes of Death: England.

	Males.	Females.	Total.
All causes	268,714	251,742	520,456
Heart diseases	23,918	25,973	49,891
Blood vessels	15,114	16,222	31,336
Rheumatic fever... ..	871	934	1,805
Rheumatism of heart ...	156	207	363
Tuberculosis (all forms) ...	34,182	21,898	56,080

TABLE II.—SHOWING NUMBER OF DEATHS DURING 1908
IN IRELAND.

Causes of Death: Ireland.

	Males.	Females.	Total.
All causes	38,219	38,672	76,891
Heart disease	4,401	4,619	9,020
Blood vessels	1,508	1,743	3,246
Rheumatic fever	91	70	161
Rheumatism of heart ...	21	37	58
Tuberculosis (all forms) ...	5,568	5,725	11,293

You see in Table II the number of deaths due to tuberculosis in all its forms, 1 in 9.2 deaths, and have thus a means of realizing the comparative mortality of two of the great causes of death.

Let us next examine the subdivisions given by the Registrar-General under the heading "Heart disease." We can understand the first six headings, but a moment's consideration will show us that the last heading must embrace a large number of undetermined causes; this is due partly to the laxity of present-day registration, and partly to inevitable inability to ascertain the exact cause. It probably embraces apoplexy, aneurysm; perhaps ruptured stomach or intestine; perhaps violence and poisoning. It is a huge collection of causes which it is impossible to analyse. For the present we must simply accept the heading, "Syncope, heart disease," but should keep in mind that in a large proportion the cause of death is probably due to some other affection than heart disease.

TABLE III.

Heart Disease: England and Wales, 1908.

	Males.	Females.	Total.
Valvular	7,611	8,752	16,363
Pericarditis	237	195	432
Hypertrophy	181	85	266
Angina pectoris	617	312	929
Dilatation	1,670	1,685	3,355
Fatty degeneration	1,133	1,417	2,550
Syncope, heart disease (not specified)	12,469	13,527	25,996

TABLE IV.

Heart Disease: Ireland, 1908.

	Males.	Females.	Total.
Valvular	1,083	1,195	2,278
Pericarditis	13	15	28
Hypertrophy	18	4	22
Angina pectoris	59	23	82
Dilatation	126	124	250
Fatty degeneration	161	125	286
Syncope, heart disease (not specified)	2,941	3,133	6,074

Comparing Tables I and III and II and IV, in England and Wales you notice that the proportion of deaths from rheumatic fever and rheumatism of heart to deaths from all forms of heart disease is 1 to 23; in Ireland it is 1 to 41; Ulster is intermediate, 1 to 36. On the other hand, the proportion of valvular disease to syncope in England and Wales is 1 to 1.5; in Ireland it is 1 to 2.6. The Registrar-General in England draws attention to the fact that deaths registered as due to "valvular disease" are slowly rising in England but those due to "syncope" are falling. His interpretation is that diagnosis and registration is becoming more accurate, and so the vague general term is being less used. If this be correct, our diagnostic capabilities in Ireland or our care in registration