ON RENAL DROPSY,
WITH
ESPECIAL REFERENCE TO THE CIRCULATION;
TOGETHER
WITH SOME CONSIDERATIONS RELATING TO PULMONARY
APOPLEXY IN RENAL DISEASE.¹

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SINCE the kidneys provide the chief exit for water, it is to be expected that disease in them such as to obstruct their function should be followed by other disposal of this constituent of the urine. Thus it is a general rule in renal disease, at least in albuminuria, that the dropsy varies inversely with the urine; with the outset of nephritis the diminution of this secretion and the access of dropsy proceed *pari passu*; while should the disease reach an advanced stage, in which the urine is abnormally copious, the dropsy then lessens and disappears. This is

¹ The general conclusions arrived at in this paper were, with the permission of the Council of the Royal Medical and Chirurgical Society, referred to in the Harveian Oration of 1891.

the rule which generally applies to albuminuria, though even here there are so many exceptional cases that it is obvious that other factors are involved beside the amount of the renal secretion. But if we include other renal and urinary diseases together with albuminuria, we find that conditions apart from the amount of discharge by the kidneys take so large a share in the result that dropsy may present itself when the urine is superabundant, and remain absent though the urine is suppressed. In some of the most intense forms of nephritis, where the urine suffers extreme diminution, dropsy may be nearly or quite absent. Dropsy is commonly absent under obstructive suppression, however complete; while the converse condition is seen with diabetes mellitus, where dropsy often presents itself though the urine is in excess. I have witnessed the development of deep œdema under diabetes with a discharge of fifteen pints a day. With such anomalies awaiting explanation, I will proceed to a brief reconsideration of the leading circumstances of dropsy, more especially when of renal origin.

It is not necessary here to dwell upon the fundamental facts upon which dropsy depends. Dropsy fluid is the exudation from the capillaries which constitutes the lymph, possibly altered by morbid action. This may accumulate either because too much has been poured out by the capillaries, or too little carried off, especially by the veins. So far as dropsy is the result of excess of exudation, and of morbid increase of or addition to the normal lymph, it is to the point to inquire by what processes liquids traverse the walls of living vessels. Of the agencies so concerned there are two which are obvious, and a third which is hypothetical.

1. *Filtration*, or transudation under pressure, acting on all the liquid parts of the blood, colloid as well as crystalloid, the albumen as well as the salts.

2. *Diffusion*, or osmosis, acting on the salts, not on the albumen.

3. *A secretory action on the part of the capillary wall,*

TABLE II.—*Constituents of Dropsy Fluids in 100 Parts (Various Observers).*

Authority.	PERICARDIUM.				PLEURA.				PERITONEUM.				CELLULAR TISSUE.			
	Sp. gr.	Solids.	Alb.	Min. salts.	Sp. gr.	Solids.	Alb.	Min. salts.	Sp. gr.	Solids.	Alb.	Min. salts.	Sp. gr.	Solids.	Alb.	Min. salts.
Schmidt, 'Lehmann's Chemistry,' ii, 316	...	...	...	...	...	...	...	...	...	...	...	...	...	...	.86	.106
Bartels, Ziemssen, xv, 106 . . .	1009-6	2-13	...	1-55	...	2-85	...	1-13	1009-6	1-56	.25	1-14	1007-6	1-17	Traces	.77
Schmidt, Gangee, i, 234 . . .	...	...	...	...	3-60	...	.75	2-10	...	2-10	...	.97	...	1-13	...	.90
Hoppe-Seyler, Gangee, i, 232 . .	...	...	...	...	4-24	2-78	...	2-42	...	3-23	1-61	...	...	1-78	.36	...
Frerichs, Diseases of Liver, ii, 44	...	...	...	...	...	...	...	2-42	1-10	...	...	...	...	...	...	...
Simon, 'Chemistry,' ii, 492 . . .	...	...	...	...	...	...	...	1007-5	1-93	.81	.80	...	...	...	...	...
" " ii, 494 . . .	...	...	...	...	...	...	...	...	...	...	...	...	1012	2-4	.70	...
" " ii, 494 (Heller)	...	...	...	...	...	...	...	...	...	...	...	...	1011	...	Very small	1-01
" " ii, 495 . . .	...	...	...	...	...	...	...	...	...	...	...	...	1010	1-32	Too small to weigh	.88
" " ii, 495 . . .	...	...	...	...	...	...	...	...	...	...	...	...	...	2-48	.54	1-56
Frerichs, 'Diseases of Liver,' ii, 44	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
DISEASE OF THE HEART.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Frerichs, 'Diseases of Liver,' ii, 44	...	...	...	...	...	...	...	...	...	1-76	1-18	...	...	...	...	...
DISEASE OF THE LIVER—CIRRHOSIS.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Lehmann, ii, 316 . . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Redenbacher, Day's 'Phys. Chem.,' p. 265	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
" " " "	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Hoppe-Seyler, Gangee, p. 233 : 1st paracentesis . . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2nd " " "	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
P.M.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Frerichs, ii, 44; average of 6 cases	...	...	...	...	...	...	...	...	...	1-55	.61	.84	...	...	...	...
	...	...	...	...	...	...	...	...	...	1-74	.77	.80	...	...	...	...
	...	...	...	...	...	...	...	...	...	1-66	.61	.81	...	...	...	...
	...	...	...	...	...	...	...	...	...	2-26	1-10	...	...	...	...	...

such as has been inferred by Dr. Waymouth Reid—a vital rather than a physical process, which may take a part more especially in the operations of disease, but of which as yet we know but little.

It is clear that the products of the first two processes must be different from each other ; the result of the third, if it exist, would probably differ from both.

I will now inquire into the composition of dropsy fluids in different diseases, with a view to ascertain whether the effusions present such differences under different circumstances as to suggest that their origin is due to different processes, or such resemblances as to imply that their mode of origin is much the same, whatever the disease.

Such effusions have frequently been examined, as the particulars appended sufficiently show. It has been laid down by Schmidt, and no doubt is generally true, that the quantity of albumen in an exudation is dependent on the system of capillaries through which it occurs ; that there is most albumen in pleural effusions, next in peritoneal, least in cellular tissue. By this showing the albuminous character of a dropsical effusion is dependent on its place rather than on its cause. I have ascertained for myself the broad characters, specific gravity, solid matter, albumen, and mineral salts in dropsy fluids from different situations and in different diseases. These observations, though not carried into detail, have the advantage of uniformity of method, and probably afford a fair basis for certain comparisons.

First, as to place : putting aside effusions in the ventricles as not to the present purpose, those in the serous cavities are more albuminous than those in the cellular tissue, the pleural sometimes more so than the peritoneal, sometimes the reverse. Next, as to effusions in the same place under different diseases, the most striking result of the comparison is the smallness of the difference. I will take the amounts of albumen, including in this term whatever is coagulable with heat and acid, and of mineral salts, as ready tests of essential resemblance, and I will include

in a general average my own results and those of others. First, with regard to albumen: pleural effusions gave under renal disease—not for the present distinguishing between lardaceous and other kinds—1·48 per cent. as the mean of six observations. Pleural effusions under heart disease gave a mean of 2·11 (three observations). As to the peritoneum, renal disease gave 1·09 as the mean of albumen in effusions in this cavity (six observations); heart disease a mean of 2·34 (five observations); liver disease a mean of 1·65 (eleven observations); œdema fluid a mean of albumen of ·34 in renal disease (eleven observations), and a mean of ·35 in heart disease (four observations).¹ Thus in the pleura and peritoneum dropsy effusions of every origin are highly albuminous, though not equally so; the albumen in heart disease being, roughly speaking, about twice as much as in renal disease, that in cirrhosis being intermediate. With regard to œdema fluid, this is but slightly albuminous under any circumstances; as between renal disease and cardiac the difference is not appreciable. As to the fixed or mineral salts, the striking fact about them is the uniformity of their quantity in the effusions of dropsy, whatever their origin and whatever their place. A glance at the annexed tables will show that whenever several fluids have been procured from the same patient, those from the chest, belly, and legs have presented these salts in almost exactly the same amount, and that what preponderance there has been has not been always in the same place. This, however, is less to my present purpose than their uniformity under different diseases. To take the peritoneal fluids as an example, these in renal disease gave ·92 as the average percentage of mineral salts (six observations); in heart disease they gave an average percentage of ·78 (four observations); in cirrhosis an average percentage of ·83 (seven observations). Similarly the fluid of œdema gave under renal disease an average of ·92 (twelve observations); under heart disease an average of ·81 (four observations).

¹ Traces or amounts too small to be weighed are calculated as none.

The constancy of the mineral salts under different conditions is more strikingly displayed in the tables than in the recapitulation ; we cannot fail to infer that whatever be the process of their extrusion, it is much the same whatever the organic cause of the dropsy. The normal percentage of the mineral salts in the *liquor sanguinis* is about .85, so that dropsy fluids, whatever their place or origin, contain about as much of these crystalloids as the blood from whence they are derived. These salts, chiefly chloride of sodium and the phosphates, transfuse readily, and probably owe their presence in effusions to a simple osmotic action. But it is sufficiently clear that this action will not explain the presence of albumen, which is colloid and little diffusible, and must traverse either as the result of pressure or of some process other than osmosis. Whatever be the process by which albumen transudes, it is in action in dropsy of all kinds, in renal as well as in cardiac and hepatic dropsy. If intra-vascular pressure, as we cannot doubt, is largely concerned in the production of cardiac and hepatic dropsy, the presumption is that it is not unconnected with dropsy of renal origin.

There will be found in the foregoing table, under the heading of Diabetes, a set of estimations which appear to me to have some significance in regard to the origin of dropsy as between osmosis and transudation by pressure, which, although they do not properly relate to renal dropsy, I will venture to refer to as throwing some light upon it. A woman with diabetic coma had within the space of thirty-two hours no less than twenty-two imperial pints of a saline aqueous liquid injected into her veins.¹ Among the results were slight œdema of the legs, and, as ascertained after death, effusions of small amount into several cavities,—18 oz. of straw-coloured serum into the peritoneum, 12 oz. of blood-tinged serum into the pleuræ, 1 oz. of the same into the pericardium. The organs presented general venous engorgement, which in the brain and lungs was intense. I had thought to find the effusions

¹ 'Transactions of the Clinical Society,' vol. xxiii, p. 130.

especially aqueous, in the expectation that by some process akin to osmosis the blood would be relieved of its superfluous water; but it was clear that increase of pressure within the vessels had more to do with the dropsy than the dilution of their contents. The blood in most of the effusions showed that the pressure had sufficed for corpuscular extrusion; while even the peritoneal liquid, into which no blood had passed, was nearly up to the average in albumen, and not superabundant in water. It is clear that this exudation was brought about less by the alteration in the quality of the blood than by increased intra-vascular pressure.

I will now take into consideration some of the circumstances of renal dropsy with particular reference to the vascular system.

I will place first a class of cases in which total absence or extreme scantiness of urine occurs, with no dropsy or but traces of it. In these there is an apparent deficiency of pressure in the arterial system so far as ordinary observation can testify. There is usually urgent uræmia, and with this much vomiting and inability to take or retain either solids or liquids. A man named Richardson¹ died of calculous suppression which was complete for four days, incomplete for seven. The pulse was at first quiet and regular; latterly it intermitted. He died by syncope. The heart was found to be flabby and uncontracted. He had no dropsy. An exceedingly fat man died after six days of complete suppression of urine due to calculous impaction.² He had a little cedema of the legs which did not increase, and was presumably due to old mitral disease. The pulse was always soft, in the earlier course of the illness from 80 to 72; at last it fell somewhat suddenly to 48 without becoming irregular or otherwise different. He then had repeated attacks of syncope, in one of which he died. The left ventricle was found to be entirely uncontracted, which not only de-

¹ 'Renal and Urinary Affections,' vol. iii, p. 951.

² *Loc. cit.*, p. 953.

clared the manner of death, but appeared to show that the fall in the rate of the pulse was not due to digitalis, which was being administered at the time. A lady died of calculous suppression which was complete for four and a half days.¹ The only trace of dropsy was a little puffiness of the eyelids. The pulse on the fourth day numbered 128; it was feeble, jerking, and irregular. She was extremely prostrate, and died, sensible to the last, by cardiac failure. The ventricles were found to be entirely uncontracted. But it is not necessary to multiply cases in proof of what will be accepted as a matter of common experience, that obstructive suppression involves no dropsy or only traces of it, a feeble pulse, and death by syncope.

I will place next another class of cases, in which extreme scantiness or even suppression of urine was dependent on disease of the kidney substance. A boy of nine had intense scarlatinal nephritis, which proved fatal by way of uræmia with convulsions in five days.² The urine, which was nearly solid with albumen, averaged almost exactly three ounces a day, or less than a pint for the whole period. Dropsy was almost totally absent—at first none, then only a trace in front of the *tendo Achillis*; no fluid in the serous cavities post mortem. There had been obstinate vomiting of food, medicine, and green slimy matter, what was brought up apparently more in bulk than what was swallowed. The pulse was slow and irregular, the patient under digitalis. A boy of ten passed bloody urine for five days without noticeable diminution; then for sixty-eight hours only half an ounce, still bloody, intensely albuminous, and swarming with large casts and renal epithelium. He then died almost suddenly by way of syncope. There had been somewhat urgent vomiting of food and bilious matter; no diarrhœa. The pulse had been weak and irregular, the temperature low. There was no trace of cedema from

¹ Loc. cit., p. 959.

² 'Albuminuria,' edit. 2, p. 95, case of Vallance.

first to last, nor evidence of fluid in any of the serous cavities. There was no post-mortem, but the existence of intense congestive nephritis was beyond question. A man¹ with albuminuria of lardaceous and syphilitic origin had œdema for a time, but lost it under spontaneous diuresis, vomiting, and diarrhœa. The urine then became so nearly suppressed that the last three days of life gave a total of only two and half drachms. There was no recurrence of the œdema, nor was any dropsical effusion found post mortem excepting a small quantity of serum in the peritoneum. Latterly the vomiting had been persistent, the tongue dry. The pulse became extremely feeble, and fell to 52. The patient remained conscious to the last, and died by asthenia. The heart after death was found to be of small size.

The preceding cases were noted without any special purpose, and, as I now regret, without the use of the sphygmograph; but their testimony is clear, and, if it were necessary, could be corroborated by other cases from medical literature. They show beyond reasonable doubt that with suppression of urine the circulation as a rule is feeble, and dropsy in abeyance. In all there was marked uræmia, in most discharge by vomiting, in some by diarrhœa. They were characterised rather by abnormal excretion than by œdema. We cannot fail to associate the absence of dropsy in circumstances which would appear to invite it with the failure of circulation—in other words, with the diminution of arterial pressure.

To turn now to a condition in which arterial pressure is increased and dropsy present, I come to one which is well known, though perhaps not fully understood. With nephritis, whether from scarlatina or cold, dropsy is usually early present, together with increase of arterial tension, not extreme or conspicuous, but generally decided, less evident to the finger than with the instrument. The late Dr. Mahomed² described a pre-albumi-

¹ George Percy.

² 'Medico-Chirurgical Transactions,' vol. lvii, 1874.

nuric stage of nephritis with increased pulse-tension, blood-crystalloids in the urine, and occasionally slight œdema. Dr. Galabin¹ has given tracings in declared but recent nephritis in which, together with albuminuria and dropsy, the tension was decidedly though not extremely increased.

I have been in the habit of late of recording the pulse by means of a Dudgeon's sphygmograph, with a Richardson's lever graduated up to 10 oz.² Each observation has consisted of a series of tracings beginning with 1 oz., and going on ounce by ounce until the pressure was enough to stop the pulse, or at least had reached 10 oz. I believe that such a series gives a more definite presentment of the force of the pulse than can be obtained from the mere configuration of one or two lines, however significant. As providing standards of health, I took twenty-one healthy medical students, and fourteen nurses under thirty years of age. The average pulse of the student gives its greatest increment or movement under a pressure of 2·8 oz.; it drops extremely under a pressure of 6·8 oz., and ceases to be perceptible over a pressure of 8·9 oz. The pulse of the nurse gives its greatest movement under a pressure of 3·2 oz., drops extremely under a pressure of 6·4 oz., and ceases to be perceptible over one of 7·5 oz. Thus it may be said in general terms that the pulse of the healthy adult gives its greatest movement at 3 oz., falls greatly at 6½, and stops between 7 and 9.³

I annex a table which will show the range of the pulse under nephritis in the acute stage and later, and in the presence and absence of dropsy. Dropsy was present at some time in all the cases referred to excepting two.

¹ 'On the Connection of Bright's Disease with Changes in the Vascular System,' 1873.

² Ultimately I found that this was not always enough, and had it weighted up to 20 oz. Only a few observations were made after this change.

³ These averages are probably high, applying as they do to the time of greatest bodily vigour. Of the twenty-one students, six were over six feet in height. In these and other observations, wherever two or more tracings presented the same degree of movement, the maximum has been taken from the latest.

TABLE III.—*Pulse in Acute Nephritis in Relation to Dropsy.*

SPHYGMOGRAPH.

Name.	Age.	Duration of disease when observation made.	Dropsy.	Heart.	Greatest movement at — ounces pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
E. M.	15	10 days	Much oedema	Sounds exaggerated	7	10	Over 10
		14 days	Less oedema	"	6	10	" 9
		26 days	Oedema nearly gone	Increase of dullness; second sound accentuated	4	7½	" 7
		46 days	None	"	4	6	" 7
L. M.	18	2¼ months	"	"	5	7	7
		5 months	None. Convalescent	Hypertrophy evident	5	8	9
		16 days	Oedema diminishing	Second sound accentuated	5	6	8
		34 days	None	"	2	—	6
		53 days	Oedema slight but decided	First sound dull; second sound accentuated	4	—	9
A. H.	19	88 days	Much oedema	"	3	5	7
		17 days	Oedema diminishing	First sound muffled; second accentuated	5	—	8
				"	3½	—	10
A. W.	37	24 days	No oedema	"	3	10	Over 10
		54 days	Considerable oedema	"	3	8	" 5
		91 days	None. Convalescent	"	—	—	" 9
		3 days (?)	No oedema	Normal	4	—	9
		41 days	Slight but decided	"	4	—	9
		43 days	"	"	5	—	9
		47 days	"	"	4	6	7
		85 days	Trace. Convalescent	Dullness; second sound exaggerated	4	—	" 7
G. G.	21	7 days	Oedema	—	1	6	7
		32 days	None	Second sound accentuated	3	6	7
		39 days	"	First sound prolonged; second accentuated	3	7	8
			None. Convalescent	Apparently hypertrophied	6	10	Over 10
		62 days					

SPHYGMOGRAPH.

Name.	Age.	Duration of disease when observation made.	Dropsy.	Heart.	Greatest movement at — ounces pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
M. H.	43	1 day (?)	None	—	7	Over 10	Over 10
L. S.	15	7 days (?)	None. Convalescent	—	5	8	8
A. B.	33	3 months	Much œdema	Dulness; second sound accentuated	5	9	9
H. S.	18	10 days	Much; general	Second sound accentuated	5	9	Over 10
A. G.	26	6 weeks	"	Dulness	—	—	8
M. R.	25	9 days	Edema gone under purging	Accentuated	3	4	4
E. S.	41	5 weeks	General	Second sound accentuated	5	8	9
		17 days	Considerable œdema	Sound accentuated	5	—	9
		23 days	Less œdema	Hæmic (?) murmur	6	Over 10	Over 10
		48 days	None	"	5	—	"
		70 days	Doubtful trace	Dulness	2	10	"
H. B.	6	7 days	Slight general œdema	Second sound accentuated	4	Over 10	"
H. L.	38	2 months	Much œdema fluid in pleuræ and peritoneum	First sound muffled; second accentuated	10	"	"
			"	"	8	"	"
J. B.	20	1 week	None at any time	Pulmonary second sound exaggerated	4	9	"
		10 days	"	"	6	9	"
		24 days	"	"	5	7	"
A. M.	29	36 days	Convalescent	"	4	Over 10	Over 10
		16 days	General œdema; fluid in pleuræ	Second sound exaggerated; hæmic murmur	4	12	16
		18 days	"	"	3	12	16
A. S.	33	6 days	Puffiness not reaching œdema	Pulmonary second sound exaggerated	4	7	10
		11 days	None	"	5	7	9

*Case of Puerperal Nephritis.*¹

SPHYGMOGRAPH.

Name.	Age.	Date of observation.	Dropsy.	Heart.	Greatest movement at — ounces pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
L. F.	20	1 day before labour 4 days after labour 8 days after labour 17 days after labour 27 days after labour 45 days after labour 75 days after labour 127 days after labour	Much general oedema; urine highly albuminous Much less oedema — No oedema No oedema; albumen slight — No oedema; trace of albumen Apparently well; slight trace of albumen	Second sound exaggerated — — — — — Second sound much accentuated Cardiac dulness increased	4 3 4 3 3 3 2 5	6 6 6 7 7 7 7 9	18 14 13 10 12 11 13 14

¹ This case, being of somewhat special nature, is not included in the averages given in the paper.

TABLE IV.—*Pulse in Chronic Nephritis in Relation to Dropsy.*

SPHYGMOGRAPH.

Name.	Age.	Duration of disease when observation made.	Dropsy.	Heart.	Greatest movement at — ounces pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
J. K.	9	4 months	Now none; much formerly	Much hypertrophy	6	—	Over 10
		4½ "	Traces	"	—	—	8
		6 "	Barely a trace	"	3	5	7
		8 "	Oedema increased	"	2	6	9
H. B.	23	7 "	Much general	Dulness, second sound accentuated	7	8	Over 10
M. G.	29	8 "	"	Hypertrophy, final pericarditis	7	Over 10	"
F. A.	3	6 "	"	Hypertrophy	3	8	"
T. H.	25	5 "	None	Dulness, second sound sharp	3	6	7
		+ 1 week	"	"	5	10	Over 10
		6 months	"	"	2	—	"
		7 "	"	"	2	10	"
		7½ "	"	"	4	10	"
		10 "	Trace after walking	"	3	7	9
Miss B.	25	2 years	Much general. Under hard purging	Extremely feeble	—	—	5
M. Y.	49	2 "	Much oedema, ascites, fluid in pleuræ	Natural	6	10	Over 10
J. S.	29	6 months	None	Dulness, second sound accentuated	5	10	10

Taking first the observations made in acute nephritis and when dropsy was present, it appears that under these circumstances the increase in the force of the pulse was constant and decided though seldom extreme. The sphygmograph gave on an average the greatest movement at 4·7 oz., fell greatly at 8·8, and stopped at 9·4.¹ Thus the pulse of acute renal dropsy conveys to the sphygmograph a force of about an ounce and a half over that of health. And this does not represent the whole increase of blood-pressure, for the elevation lasts longer than in health, as is best displayed with Marey's instrument. Looking at the same series of cases in the absence of dropsy, it would seem that there is less pulse-pressure without dropsy than with it, in the proportion of 8·5 to 9; but this relation is complicated by the beginnings of chronic change, as will presently appear. Proceeding to chronic nephritis, it is to be seen that, whether dropsy be present or absent, the tension of the pulse, or at least the force required to stop it, increases with the duration of the disease. The cases recorded pulsate on an average up to 9·6 oz. Not as yet going beyond nephritis, it is certain that renal dropsy is attended with an increase of arterial pressure, though there may be an increase of arterial pressure and no dropsy. The association of increased tension with nephritis and dropsy is illustrated by the early development of hypertrophy of the heart and arteries—at once a result, a proof, and in some sort a measure of the abnormal state of the circulation. Regarding the hypertrophy as a result of the tension, it is apparent that this is enough to react upon the system and produce pathological change. The hypertrophy is found earliest in the left ventricle, where it often becomes marked within a month of the outset of the renal disease; the arterial change becomes amenable to observation somewhat later, but possibly occurs simultaneously. In Table V, at page 342, are instances of this form of hyper-

¹ In these calculations, where the tracing has not stopped at 10 oz., it has always been reckoned as 11; in some cases it should obviously have been higher.

trophy produced in from three to ten weeks. Thus we have conclusive evidence that in early nephritis there is enough increase of arterial tension to give rise to structural change, and presumably enough to modify vascular action and transudation.

This increase of tension must necessarily be attributed to difficulty of arterial exit ; and next comes the question where precisely the difficulty arises, whether in the arterioles or the capillaries. I am perfectly aware that the arterioles are highly muscular, and that, especially under nervous influences, they have the power of shutting off the blood from their several territories. But here we have to do more immediately with blood than nerve, and it is not unreasonable to suppose that the blood may acquire properties which may interfere directly and immediately with its passage through the capillaries, with which vessels it is brought into more intimate relation than with any others. The capillaries have been shown to be contractile, though they are not muscular ; and the fact that cedema presents itself, together with arterial tension, would seem to indicate these vessels as the seat of the impediment. It is not to be doubted that with nephritis the cedema is mainly the result of increased exudation, and that from the capillaries. If the blood-stream were arrested or greatly diminished in the arterioles, we should presume capillary exudation to be diminished rather than increased. It might be suggested as opposed to this presumption that in lardaceous disease cedema appears, though the arterioles are undoubtedly and permanently narrowed. But with regard to this, as will presently appear, the nature of the cedema, as between increased exudation and diminished absorption, is not so clear as to furnish a sure basis for reasoning. Whether in nephritis the capillary hindrance depends on change in the capillaries themselves or in the relation of the blood to them is a question for future investigators ; and, indeed, this part of the subject is one in which physicians need further help from physiologists.

It may be asked whether the series of changes so far indicated is the sole cause of acute renal dropsy. As yet only increased exudation has been had regard to, but there are conditions which at least suggest the possibility of diminished absorption. The frequent association of inflammation of the respiratory organs with acute nephritis cannot but introduce into the problem the question of venous obstruction. I have elsewhere¹ shown what I think is generally recognised, that pneumonia, bronchitis, and pleurisy are, especially in childhood, frequent concomitants of acute nephritis and frequent causes of death. I have more recently had experience of fourteen post-mortems, ten of children, four of adults, in acute renal dropsy of from one week to three months' standing. The condition of the respiratory organs was as follows :

State of organs of respiration in fourteen cases of acute renal dropsy (post mortem).

Natural	1
Pneumonia or pleuro-pneumonia	3
Bronchitis (only)	1
Œdema of lung, congestion, and bronchitis	2
Fluid in pleuræ	1
Fluid in pleuræ with œdema of lung and congestion	5
Fluid in pleuræ with bronchitis	1
	<hr/>
	14

It will be seen that the respiratory organs were natural in only one instance, and that in twelve of the fourteen there was bronchitis, congestion or œdema of the lung, or pneumonia.

Now to appeal to clinical apart from post-mortem experience, in sixty-one cases of acute renal dropsy collected during my experience at St. George's Hospital, the Hospital for Sick Children, and elsewhere, noted at

¹ 'Albuminuria,' 2nd edit., p. 45.

dates varying from four days to three months of the outset, the evidence with regard to the respiratory organs was as follows :

Clinical observations on state of organs of respiration in sixty-one cases of acute renal dropsy, of which thirty-eight were in children under twelve years of age.

No respiratory disturbance noted in	18
Cough ; no morbid signs noted in	6
Large rhonchus or bronchitis only	8
" " " together with dul-	
ness	2
Dulness only	7
Evidence of fluid in pleura only	4
Fluid in pleura and fine crepitation	2
Fine crepitation, coarse or tubular breathing	
(dulness also noted in five)	13
Sounds indistinct	1
	<hr/>
	61

It appears that the respiratory organs were abnormal in over two thirds of the number, while in over one third there was evidence of bronchitis or broncho-pneumonia. Thus on every showing it is manifest that inflammatory states of the organs of respiration, particularly as bronchitis or broncho-pneumonia, occur with significant frequency in connection with recent renal dropsy. This cannot but impede the venous return by the lungs, and must enhance the dropsical state by lessening venous absorption. It is occasionally to be observed in such cases that as the lung clears the dropsy diminishes. And it is worth noting in this connection that the exaggeration of the pulmonary second sound in renal disease generally is even more marked than that of the aortic, as if the tension of the pulmonary vessels were affected certainly not less than that of the systemic. But, nevertheless, it is impossible to assign more than a

secondary place to the respiratory organs in the causation of this form of dropsy, for the dropsy may present itself while these organs are unaffected ; and, furthermore, there is usually an absence of superficial venous turgidity, pallor being present rather than congestion. To these considerations must be added the fact that hypertrophy of the right ventricle, which would necessarily result from much and continued pulmonary obstruction, is in nephritis, as in every form of renal disease, little marked compared with that of the left, as if the essential pathology were more systemic than pulmonary. It is probable that the morbid state of the lung and bronchi arises in common with the dropsy as a parallel result of the condition of blood which produces it, rather than that the respiratory lesion is the essential cause of the dropsical.

It is worth remarking that fluid in the pleura stands in a double relation to the dropsy of which it is part, for it necessarily reacts to its increase by way of obstruction by pressure on the venous outlet.

So far it appears that acute renal dropsy has more to do with the state of the arteries than of the veins ; and that it is associated with arterial tension, whether this condition be its cause or only connected with the cause. Considering the not infrequent occurrence of high pulse pressure, apart from renal disease, without the production of dropsy, I cannot think that the increase of tension in renal disease suffices alone to account for it, though constantly associated with it. The most feasible supposition appears to be that the morbid state of the blood produces a condition of the capillaries which our present knowledge does not enable us to define, but which both obstructs the blood and facilitates transudation.

Passing now from acute or recent renal dropsy to that of the more chronic conditions—I do not yet touch upon the lardaceous state—another phase is presented. With the continuance of the disease, and its change from acute to chronic, dropsy lessens while arterial tension increases. The dropsy often goes entirely while the tension becomes

extreme. This appears to be in contradiction to what has gone before, and I must delay for a moment in the attempt to explain the paradox or reconcile the discrepancy. The disappearance of the dropsy with the prolongation of the disease is a natural process, which is too often attributed to treatment. Diuresis often occurs spontaneously, and must share in the *modus operandi*. But that the quantity of urine passed does not alone determine the presence or absence of dropsy is shown by facts to which reference has already been made—the total absence of dropsy under obstructive suppression; its nearly complete absence in some forms of nephritis where the urine is almost suppressed, but not quite; and, conversely, the establishment even of deep œdema under profuse polyuria in diabetes. It is clear that, in the removal of dropsy, other conditions beside diuresis must intervene; if diuresis removes it, the diuresis must occur under certain conditions. Among the conditions which attend the spontaneous mitigation of renal dropsy are great increase of arterial tension and tangible hypertrophy of the heart and arteries. I would venture to assert as a general law that renal dropsy is antagonised by the cardio-vascular change of renal disease.

It is scarcely necessary that I should do more than appeal to the experience of this Society in support of the position that, be the intermediaries what they may, renal dropsy is usually on the wane when cardiac hypertrophy and pulse-tension have largely declared themselves, and even under the same associations is often totally abolished. Nevertheless I will adduce a few instances which it would be easy to multiply.

CASE 1.—Mary T—, 21 years of age, a maid of all work, had slight sore throat, followed by swelling of the legs and scantiness of urine. Three weeks later she came into the hospital with much general œdema, which soon increased and became conjoined with ascites and hydrothorax. The swelling of the belly from the dropsy within and without was enough to tear the true

skin in lines like exaggerations of the *linea albicantes* of pregnancy; similar but smaller fissures presented themselves on the calves. The urine, at first scanty and albuminous nearly to solidity, became gradually more copious, increasing in the course of six weeks from 8 oz. to 132 oz. as the daily quantity while the dropsy lessened with parallel steps. The peritoneum and the right pleura were tapped and the œdema diminished, until ten weeks after admission all dropsy had disappeared, and she was convalescent excepting that the urine still contained albumen, which finally fell to a trace. Mr. Hale, my clinical clerk, made frequent records of the pulse with the sphygmograph, with the result that some over-tension was present from the first, which increased with the diminution of dropsy, and continued to do so after its disappearance. Evidence of hypertrophy of the heart slowly presented itself, while the second sound became markedly accentuated. The patient was very fat, which probably obscured the hypertrophy for a time, but it was sufficiently clear that this had an inverse relation to the dropsy, as also had the pulse tension. When she was last seen, some months after the outset, the heart appeared to be still large; she was then practically well, except for a minute trace of albumen in the urine.

CASE 2.—Rosa B— had severe scarlatinal dropsy in St. George's Hospital, at the age of 5. Eight years afterwards she was readmitted with chronic albuminuria, but no dropsy save a little puffiness under the eyes. She then had diuresis and thirst, hypertrophy of the heart, tension of the pulse and albuminuric changes in the retina.

The puffiness about the eyes subsided, and she left the hospital completely devoid of dropsy.

CASE 3.—Francis W— had scarlatina at the age of 5, followed by dropsy, which subsided and left him in apparent health, but with a liability to headache and epi-

staxis. At the age of fourteen, strabismus appeared, a year later he began to lose strength and memory, and he subsequently became slightly deaf on the left side. At the age of twenty he was found to have albuminuric changes in the retinæ. The urine was then albuminous and superabundant, and himself anæmic, emaciated, and entirely without œdema. The heart was obviously hypertrophied, and the pulse one of the hardest I ever felt, difficult to compress, and almost impossible to stop.

He had occasional vomiting, constipation, and wakefulness, and frequent attacks of pain below the umbilicus, especially after food. Symptoms of peritonitis were presently superadded, and were followed by death. It was found, as had been anticipated, that the small bowel was the seat of albuminuric ulceration and perforation. The kidneys were in an advanced state of granular contraction; the heart was increased in weight to 15 oz., the left ventricle and the columnæ carneæ much hypertrophied. Some arteries which were examined, those of the kidney and pia mater, were much thickened.

CASE 4.—Bessie B— had scarlatina at the age of 17, followed by swelling of the face, legs, and abdomen. She recovered sufficiently to resume her work as a general servant at a place where she was over-worked and underfed. At the age of twenty-one she began to have diuresis, together with headache, vomiting, and epistaxis. Admitted into St. George's Hospital, she had bleeding at the mouth independently of the epistaxis, profuse menorrhagia, bruise-like marks on the body, hypertrophy of the heart, and a murmur thought to be hæmic. She was in a condition of much exhaustion and anæmia, with a weak and failing pulse. There was no œdema or other dropsy. She sank a few days later, after much dyspnoea. The urine had never been obtainable. The kidneys were found to be granular and atrophied, the left no larger than a horse-chestnut, the right shrunk but to a less extent. The left ventricle was hypertrophied and contracted; the weight

of the heart was 13 oz. The lungs were congested and œdematous.

I have elsewhere¹ related the case of Emily T—, who died at the age of twelve with a clot in the brain as large as a goose's egg. She had had scarlatina but no sequelæ were recognised, nor had she ever had dropsy. The kidneys were exquisite examples of granular contraction, with profuse interstitial growth. The heart was enormously hypertrophied, especially the left ventricle, and weighed 8½ oz. The valves were healthy. The arteries as examined in the pia mater showed enormous thickening of their walls, both of the muscular and fibrous coats.

There is a common combination of circumstances which I could exemplify numerously, as no doubt could other Fellows of this Society,—chronic albuminuria with the granular kidney, hypertrophy of heart, arterial thickening and tension of pulse, and either no dropsy at any time or only a little, and that in the past.

To give some instances in brief :

Hannah G—, now 40 years of age, single, was first admitted in 1884 with highly albuminous urine, slight œdema in both legs, venous thrombosis in one ; no evidence of cardiac hypertrophy, but some hardness of pulse. In 1887 she was readmitted. The œdema was now either totally absent or present only as a trace. The urine was less albuminous, the pulse hard, the left ventricle tangibly enlarged. In March, 1891, she was again admitted, now quite without œdema, though traces occasionally reappeared until the following July, after which there was none. She was much attenuated, the legs shrunk as if dropsy were a *minus* quantity. The urine contained now only a trace of albumen ; it was of low specific gravity and alkaline. The chief troubles were nausea and headache, which were treated with success as uræmic. The cardiac dulness was enormously increased, the first sound muffled

¹ 'Albuminuria,' 2nd edit., p. 194; also p. 308.

and prolonged, the second accentuated. The pulse was tense to the utmost experience of renal disease ; it felt like a tendon larger than the radial artery ; it could not be completely stopped with the finger, while under the greatest pressure of the sphygmograph, ten ounces, it gave a bold tracing.

George R—, aged 55, a somewhat intemperate marble polisher, began two years before admission to pass much water, and to be occasionally breathless, faint, and giddy. When seen the respiration was laboured, and markedly of the Cheyne-Stokes character. The veins of the neck were turgid. The heart was greatly enlarged, especially to the left, and had the rhythm of the galloping horse, which signifies reduplication. The first sound was muffled, the second sounds accentuated, the pulmonary most so. The pulse was quick and excessively hard, not to be stopped with the finger, while with the sphygmograph a strongly declared tracing was given under the highest attainable pressure, that of ten ounces. The lungs were full of sharp, moist sounds. The urine was pale, contained a trace of albumen, and deposited uric acid.

There was no history of cedema, nor was any evidence of this or of any other form of dropsy to be detected. He had long suffered from insomnia, and shortly after admission became delirious. Measures suitable in uræmia were enforced, and decided though temporary benefit was obtained from a venesection of 14 oz. He died six days after admission. The post-mortem failed to reveal any dropsical effusion. The kidneys, of unequal size, weighed together 9 oz. ; they were red, granular, cysted, and fibrotic. The heart weighed 24 oz. ; the hypertrophy was chiefly of the left ventricle, though both were hypertrophied and dilated. The auriculo-ventricular openings were dilated, the valves healthy (see outline of heart, Fig. 31).

Both lungs were loaded with frothy fluid ; in the

lower lobe of the left was a mass of pulmonary apoplexy.

The foregoing cases have been selected as typical. The first (Mary T—) shows the gradual removal of renal dropsy under recovery from nephritis coincidently with hypertrophy of the heart and increase of tension. Three others (Rosa B—, Francis W—, and Bessie B—) display as remote sequelæ of scarlatinal nephritis the establishment of the granular kidney with cardio-vascular hypertrophy, and complete or nearly complete removal of the dropsy which had once existed. In all the arterial tension had been such as to cause hæmorrhagic processes. A fifth case exhibits hypertrophy of the heart and arteries and cerebral hæmorrhage in connection with renal fibrosis, to which no dropsy had, as far as was known, ever been annexed. The remaining cases exemplify, as has been indicated, great hypertrophy of the heart, great arterial thickening safely inferred though not exposed to view, extravagant arterial tension, together with absence of dropsy, absolute or nearly so, at least by the time the cardio-vascular change was fully accomplished.

The general and irresistible conclusion is that cardio-vascular hypertrophy, with the arterial tension which is associated with it, have together an inverse relation to renal dropsy. As this state of the circulatory system establishes itself there is frequently, perhaps generally, diuresis; this may be an intermediary *modus operandi*, and presents a point of view in which the inverse relation may be regarded. General increase of tension, involving as it must the renal arteries, tends to cause increase of urine, so that renal disease becomes its own diuretic.

Observations made during life must be supplemented by others made after death. A large degree of cardiac hypertrophy, as is well known, attends the chronic granular kidney; a smaller but yet a decided degree succeeds upon acute nephritis: with the lardaceous

kidney there is generally none, never much. As a general rule with albuminuria, not lardaceous, the hypertrophy begins with the disease and increases as it continues. In five or six weeks from the outset of nephritis it is usually recognisable. When it has attained a considerable measure the dropsy, as I have demonstrated clinically, usually lessens or disappears.

I annex a list of well-marked cases of renal disease, showing in each condition the amount of dropsy in relation to the state of the heart.

TABLE V.

TABLE V.—*State of Heart P.M. in relation to Dropsy.*

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Edema.
<i>Males :</i>						
J. S.	32	Large, white; right atrophied from stone	—	Scanty; albuminous; bloody	Slight	Slight
G. M.	31	Cortices swollen, yellowish; surfaces uneven	16 oz.	Scanty, dark, highly albuminous	Much	Much
J. D.	40	Large, white; acute nephritis	22 oz.	Scanty, dark, highly albuminous	Much	Much
J. R.	18	Large, white	25 oz.	Scanty, pale, highly albuminous	Much	Much
H. H.	15	Large, white	14 oz.	Scanty; highly albuminous; high sp. gr.	Considerable	Considerable
R. W.	7	Large, white	8½ oz.	Scanty; highly albuminous; sometimes bloody	Much	Much
J. G.	2	Large, white; acute nephritis	5 oz.	Albuminous	Much	Much
A. W.	2½	Large, white; scarlatinal	4 oz.	Scanty, dark, albuminous	Latterly 0	Latterly 0
J. M.	7	Scarlatinal nephritis	9 oz.	Scanty, smoky, highly albuminous	Much	Much
C. M.	5	Scarlatinal nephritis	5 oz.	Highly albuminous; bloody	Much	Much
W. W.	22	Large, white, smooth	21 oz.	Scanty; bloody; high sp. gr.; highly albuminous	Extreme general	Extreme
J. M.	12	Large, white	13 oz.	At first highly albuminous, latterly less so	General	Much
J. K.	9	Large, white	15½ oz.	Scanty; highly albuminous	Extreme	Extreme
J. K.	48	Right kidney congestive nephritis; left atrophy from obstruction of artery; aneurysm	Right 8 oz.; left 4 oz.	Scanty; highly albuminous	Much	Much

* This mark indicates that the heart is

Nephritis. No Evidence of Mitral Regurgitation.

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
0	0	Much	2 years	2 years	15 oz.*	—	Left ventricle hypertrophied; valves natural; slight pericarditis.
Much	Little	Much	10 weeks	10 weeks	12 oz.	—	Pericarditis; valves natural.
Much	Some	Some	8 weeks	10 weeks	14 oz.	—	Left ventricle hypertrophied; valves natural.
Much	Much	Much	2 mos.	3 mos.	14 oz.	—	Slight hypertrophy of left ventricle; valves natural.
1½ pints	0	0	3 mos.	3 mos.	5 oz.	—	Natural.
Some	0	Some?	10 weeks	14 weeks	6½ oz.	—	Hypertrophy, especially of left ventricle; valves natural.
0	0	Much	3 weeks	3 weeks	3½ oz.	—	Left ventricle dilated; valves natural.
0	0	0	1 month	Scarlatina 2½ mos.	2½ oz.	—	Some hypertrophy of left ventricle; valves natural.
0	0	Some	16 days	Scarlatina 9 weeks	5½ oz.	—	Left ventricle hypertrophied.
Much	Some	0	3 weeks	Scarlatina 6 weeks	3½ oz.	—	Valves healthy.
Full of sero-purulent fluid	0	Great	4 mos.	4 mos.	11 oz.	—	Healthy.
0	0	Ascites	13 mos.	13 mos.	6 oz.*	—	Both ventricles hypertrophied, especially left
8 oz.	0	Much	9 mos.	9 mos.	7 oz.*	—	Hypertrophy of left ventricle; slight of right.
0	0	0	3 mos.	3 mos.	28 oz.*	—	Left ventricle much hypertrophied; valves natural

shown in section in the annexed figures.

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Edema.
<i>Males:</i> A. M.	29	Intensely congested ; smooth	14½ oz.	Albumen equal to two thirds; scanty	Considerable	Considerable
<i>Females:</i> S. W.	30	Large, white ; acute nephritis	16 oz.	Very scanty; albuminous	Much	Much
E. L.	33	Large, pale, congested ; acute nephritis	16 oz.	Scanty, latterly suppressed ; highly albuminous	Much	Much
E. B.	15	Large, white	15 oz.	Scanty, smoky, highly albuminous	Considerable	Considerable
J. I.	2	Acute nephritis ; scarlatinal	3½ oz.	Albumen bloody, not scanty	Much	Much
A. F.	7	Scarlatinal nephritis	5½ oz.	Scanty, smoky, albuminous	Much	Much
E. A.	6	Large, mottled, smooth	13 oz.	Scanty ; highly albuminous	Much	Much
H. L.	10	Large, white, smooth	7½ oz.	Scanty ; nearly solid with albumen	Extreme	Much
C. B.	19	Large, white, smooth	19 oz.	Scanty ; highly albuminous	Edema	Much
M. G.	29	Large, white, becoming granulated	10½ oz.	Extremely scanty, pale, albuminous	Much	Much
F. C.	3	Large, smooth ; nephritis	6 oz.	Scanty, smoky, highly albuminous	Extreme	Extreme
M. J.	49	Mottled, smooth ; advanced nephritis	15 oz.	Scanty ; nearly solid with albumen	Much	Much
V. E. ¹	9 mos.	Acute parenchymatous ; smooth	5½ oz.	—	0	0

* This mark indicates that the heart is

¹ This case entered also under Pulmo

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
Some in both	0	0	12 days	15 days	13½ oz.*	—	Valves natural; left ventricle thought to be hypertrophied.
0	0	Much	6 weeks	6 weeks	13 oz.	—	Soft; mitral orifice contracted.
0	0	0	1 month	4 mos.	12 oz.	—	Valves natural; pericarditis.
Some	0	Much	3 mos.	3 mos.	8 oz.	—	Left ventricle hypertrophied; right ventricle dilated; valves natural.
6 oz.	0	0	6 days	Scarlatina 3 weeks	2½ oz.	—	Slight thickening of mitral edge.
18 oz.	2 oz.	0	4 weeks	Scarlatina 9 weeks	6 oz.	—	Hypertrophied; valves healthy.
Some	Some	Some	2½ years	2½ years	4½ oz.	—	Left ventricle much hypertrophied; valves healthy.
5 oz.	½ oz.	Some	3 mos.	3 mos.	6 oz.	—	Left ventricle hypertrophied; valves healthy.
0	0	0	10 mos.	10 mos.	10 oz.	Systolic murmur latterly	Recent ulcerative endocarditis.
Much	Some	Much	8 mos.	8 mos.	14 oz.*	—	Hypertrophy of both ventricles, especially left; recent pericarditis.
Much	Extreme	Much	9 mos.	9 mos.	2½ oz.*	—	Left ventricle slightly hypertrophied.
Much	0	Much	2 years	2 years	10 oz.*	—	Apparently normal; valves healthy.
0	0	0	—	Diarrhœa 2 weeks	5½ drms.*	Pulmonary apoplexy	Valves healthy; left ventricle thought to be hypertrophied.

shown in section in the annexed figures.
 nary Apoplexy.

State of Heart P.M. in relation to Dropsy. Granular

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Edema.
<i>Males :</i>						
D. J.	45	Well-marked granular	12 oz.	Chronic polyuria; latterly scanty; sp. gr. 1028; albumen = $\frac{1}{4}$	Some	Some
J. W.	20	Granular scarlatinal	—	Pale; sp. gr. 1011; albuminous; much polyuria	0	0
J. B.	28	Highly granular	14 oz.	Pale; albuminous; latterly nearly suppressed	Slight	Slight
R. H.	55	Granular	12 oz.	Highly albuminous; somewhat scanty; albumen = $\frac{1}{4}$	Much	Much
G. L.	59	Granular; gouty	6 oz.	Latterly pale and scanty	Some	Some
C. D.	14	Well-marked granular	4 $\frac{1}{2}$ oz.	Excessive; low sp. gr.; albumen slight	0	0
J. S.	52	Granular, cysted, shrunk	—	Excessive, pale, highly albuminous; sp. gr. 1015	None latterly	0
Mr. T.	50	Granular; capsule adherent	—	Pale; sp. gr. 1012; albumen much	Latterly	Latterly
H. T.	39	Well-marked granular	6 oz.	Excessive; sp. gr. 1011; albumen = $\frac{1}{4}$	0	0
B. H.	60	Granular; tubal nephritis	—	Early excess, latterly scanty; sp. gr. 1017; albumen = $\frac{3}{8}$	Much	Much
W. C.	47	Well-marked granular	7 oz.	Pale; sp. gr. 1013; albumen = $\frac{1}{8}$	Traces	Traces
J. B.	79	Advanced granular	7 oz.	—	Slight cedema	Slight
H. T.	43	Granular, gouty	9 oz.	Excessive; sp. gr. 1006; albumen = $\frac{1}{16}$	None, save in pleuræ	0

* This mark indicates that the heart is

Kidney. No Evidence of Mitral Regurgitation.

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart	Mitral regurgitation.	Heart, pericardium, infarcts.
40 oz.	0	0	1 month	1 month	21 oz.	—	Left ventricle hypertrophied; valves natural; recent pericarditis.
0	0	0	—	16 years	15 oz.	—	Left ventricle much thickened; valves normal.
Slight	0	0	13 mos.	13 mos.	18 oz.	—	Left ventricle much hypertrophied; valves natural.
Much	0	Much	2 years	2 years	26 oz.	Much bronchitis	Valves good; mitral slightly thickened, aortic beaded; recent pericarditis.
0	0	0	3 weeks	6 weeks	26 oz.	—	Left ventricle much hypertrophied, right ventricle somewhat.
0	0	0	—	—	8 oz.	—	Left ventricle highly hypertrophied; valves healthy.
0	0	0	—	2 years	22 oz.	—	Greatly hypertrophied, chiefly on left side; valves healthy.
1 pint	0	0	3 mos.	15 mos.	—	Loud reduplication of first sound	Enormous hypertrophy of left ventricle; valves natural.
0	0	0	—	2 years	24½ oz.	—	Left ventricle hypertrophied and dilated; valves natural.
Much	0	Much	12 mos.	Some years	25 oz.	—	Left ventricle hypertrophied; valves natural.
0	0	0	—	—	26 oz.*	Apex murmur 8 years ago, none latterly	Both ventricles hypertrophied; valves natural; recent pericarditis.
0	0	0	—	—	16 oz.*	—	Left ventricle hypertrophied; fat on surface; left valves thickened, but competent.
3 pints	0	0	—	Gout 3 years	23 oz.*	—	Both ventricles hypertrophied; valves healthy.

shown in section in the annexed figures.

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Œdema.
<i>Males :</i>						
C. W.	64	Granular, contracted	10 oz.	Albumen = $\frac{1}{2}$	0	0
E. G.	44	Granular, contracted, gouty	8 oz.	Scanty ; sp. gr. 1015 ; albumen = $\frac{1}{4}$	Much	Much ; none p.m.
G. W.	58	Granular, gouty	6 oz.	Pale ; sp. gr. 1013 ; albumen = $\frac{1}{8}$	Slight œdema	Slight
P. C.	35	Horseshoe, granular	8 oz.	Amount normal ; sp. gr. 1013 ; albumen = $\frac{1}{2}$	None seen during life	Trace p.m.
<i>Females :</i>						
A. J.	51	Extremely granular	10 oz.	Alkaline ; albumen = $\frac{1}{4}$; sp. gr. 1012 ; pale, clear	Little	Slight
E. T.	12	Granular ; highly fibrotic	2½ oz.	—	0	0
B. B.	21	Granular ; one atrophied	—	Polyuria 2 or 3 years ; pale, latterly darker ; sp. gr. 1018 ; albumen = $\frac{1}{4}$	0	0
L. L.	62	Granular	4 oz.	Pale ; sp. gr. 1012 ; highly albuminous	Some œdema	Some
M. W.	21	Granular, scarlatinal	3 oz.	Pale, copious ; much albumen	Some	Traces
E. F.	38	Granular, wasted	8 oz.	Generally scanty ; albuminous	Great generally	Much
E. S.	35	Granular, wasted	7 oz.	Highly albuminous ; quantity not known	None	None

* This mark indicates that the heart is

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
0	0	0	—	—	16 oz.*	—	Left ventricle hypertrophied; some fat on surface; valves healthy.
0	0	Much	5 weeks	Gout some years	10 oz.*	—	No marked hypertrophy; valves healthy.
0	0	0	—	—	22 oz.*	—	Left ventricle hypertrophied and dilated; recent pericarditis.
0	0	0	—	6 mos.	18 oz.*	—	Left ventricle hypertrophied; valves natural.
0	0	0	1 year	Over 1 year	26 oz.	—	Both ventricles hypertrophied; valves natural.
0	0	0	—	—	8½ oz.	—	Left ventricle hypertrophied; valves natural.
0	0	0	—	4 years	13 oz.	—	Left ventricle much thickened; valves natural.
0	0	0	3 weeks	3 weeks	13 oz.	—	Left ventricle hypertrophied; mitral valve somewhat contracted, aortic thickened; no murmur.
Much turbid	0	0	1 year	1 year	14 oz.	Murmur at base (hæmic?)	Cavities dilated; valves natural; recent pericarditis.
2 quarts	4 oz.	Distended; tapped often	7 years more or less	7 years	17 oz.	Mitral murmur for last week or two only	Left ventricle greatly hypertrophied; mitral and aortic valves thickened.
None	None	None	—	?	11½ oz.*	—	Left ventricle hypertrophied; valves healthy.

shown in section in the annexed figures.

*Heart P.M. in relation to Dropsy. Granular Kidney and Nephritis
Mitral Murmur, apart from in*

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Edema.
<i>Pulmonary</i>						
<i>Males :</i>						
J. F.	56	Advanced, granular	14 oz.	Albumen = $\frac{1}{10}$; sp. gr. 1020—1025	Much	Much
T. W.	54	Small, red, granular	—	Scanty; highly albuminous	Much	Much
R. M.	70	Congested; surfaces rough	—	Orange; sp. gr. 1030; albumen = $\frac{1}{4}$ — $\frac{1}{2}$	—	0
T. S.	24	Granular; left shrunk, contained stones	8 oz.	Copious; sp. gr. 1010; albumen little more latterly	Much	Much
T. G.	62	Granular, cysted; contained infarcts	12 oz.	Albumen = $\frac{1}{2}$ — $\frac{3}{4}$; sp. gr. 1016	Much	Much
W. G.	65	Granular, cysted; infarcts	10 oz.	Scanty, high-coloured; sp. gr. 1022; albumen = $\frac{1}{10}$	Considerable	Considerable
H. E.	54	Granular	14 oz.	Very scanty; highly albuminous	Much	Much
G. R.	55	Granular	9 oz.	Formerly superabundant, latterly thought to be scanty; albumen slight	0	0
<i>Females :</i>						
E. R.	36	Granular, cysted	10 oz.	Clear, albuminous; sp. gr. 1020	Much	Much
E. K.	53	Granular	10 oz.	Scanty, lithatic, highly albuminous	Much	Much

* This mark indicates that the heart is

with Evidence of Mitral Regurgitation, in Pulmonary Apoplexy or dependent disease of the Heart.

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
<i>Apoplexy.</i>							
Some	Slight	—	Over 6 weeks	8 mos.	26 oz.*	Pulmonary apoplexy	Great hypertrophy of left ventricle; mitral opening dilated; valves natural.
4 pints	—	Much	2 mos.	6 mos.	21 oz.	Pulmonary apoplexy	Both ventricles hypertrophied and dilated; no valvular disease.
1 pint bloody	—	Some bloody	—	6 mos.	21 oz.	Pulmonary apoplexy	Flabby; both sides dilated; valves healthy; roughness on pericardium.
—	—	—	—	3 mos.	18 oz.*	Pulmonary apoplexy	Left ventricle much hypertrophied; right ventricle hypertrophied and dilated; valves natural.
Much	—	Some	—	10 weeks	20 oz.	Profuse hæmoptysis	Left ventricle hypertrophied and dilated; valves natural.
Some bloody	—	Some bloody	4 mos.	4 mos.	21 oz.*	Pulmonary apoplexy; mitral murmur	Left ventricle hypertrophied; right ventricle hypertrophied and dilated; valves efficient, somewhat atheromatous.
Much	—	Much	3 weeks	3 years	17 oz.	Threw up a large quantity of blood, apparently from lungs	Left ventricle dilated and soft; aortic valve slightly calcareous.
0	0	0	—	2 years	24 oz.*	Pulmonary apoplexy	Both ventricles dilated and hypertrophied; valves natural.
—	—	Some	3 mos.	24 years?	15 oz.	Pulmonary apoplexy; systolic murmur at apex	Left ventricle hypertrophied; all valves natural.
Much	—	Much	3 weeks	3 years	17 oz.*	Pulmonary apoplexy	Both ventricles dilated; all valves natural.

shown in section in the annexed figures.

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Edema.
<i>Females :</i>						
V. E.	9 mos.	Large, smooth, mottled ¹	5½ oz.	Not obtained	0	0
<i>Other Evidence of</i>						
<i>Males :</i>						
J. B.	47	Very small, granular; gout	—	Scanty, turbid, highly albuminous	Little	Little; none latterly
T. E.	54	Advanced granular	5 oz.	Clear, pale; sp. gr. 1009; albumen = $\frac{1}{4}$	Much; probably pericardial	Much
W. S.	45	Finely granular; cortex shrunk	—	Loaded with albumen and lithates	Much	Much
P. H.	42	Advanced granular; gout	7 oz.	Excessive, latterly scanty; albumen = $\frac{1}{8}$	Much	Much
W. J.	66	Granular	12½ oz.	Copious; low sp. gr.; slightly albuminous	Much	Much
<i>Females :</i>						
E. F.	37	Granular	8 oz.	Pale; highly albuminous; scanty	Much	Much
J. B.	18	Granular, shrunk	—	Diminished; sp. gr. 1012; much albumen	Slight; latterly none	Slight; latterly none
<i>State of Heart P.M. in relation to</i>						
<i>Males :</i>						
W. P.	11	Large, pale; caseous mass in one; caries	12½ oz.	Increased; low sp. gr.; albumen = $\frac{1}{4}$	Much	Much
F. G.	52	Marked lardaceous; syphilitic	9 oz.	Scanty, albuminous	Much	Slight
C. T.	28	Large, white, syphilitic	24 oz.	Early excess, latterly quantity normal; highly albuminous	Some	Some

* This mark indicates that the heart is

¹ Child rickety and ill-nourished. Died

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
0	0	0	—	Diarrhoea 2 wks.	5½ drms.	Many pulmonary apoplexies	Left ventricle thought to be somewhat hypertrophied; valves healthy.

Mitral Regurgitation.

0	0	0	13 mos.	13 mos.	18 oz.	Systolic murmur at apex	Both ventricles hypertrophied and dilated; aortic valve competent; atheromatous.
—	—	Some dark	2 weeks	?	28 oz.	Lungs congested; liver nutmeg	Left ventricle enormously hypertrophied; mitral region pouched; mitral valve stiff; pericarditis; fluid in pericardium.
Much	0	0	1 month	1 month	22 oz.	Murmur; congestion of lungs	Left ventricle hypertrophied; mitral valve thickened, but competent.
0	0	0	8 weeks	Gout 12 years	36 oz.	Doubtful murmur at apex; lungs congested	Cavities large; great hypertrophy; mitral valve thickened; recent pericarditis.
Much	Little	Ascites	2 mos.	10 years	29 oz.*	Systolic murmur at apex	Left ventricle greatly hypertrophied; right ventricle hypertrophied and dilated; valves natural; recent pericarditis.
2 quarts	4 oz.	Much	6 years	6 years	17 oz.	Systolic murmur at apex	Left ventricle greatly hypertrophied; few patches of thickening on mitral and aortic valves.
0	0	0	5 mos.	2 years	16 oz.	Mitral systolic murmur	Left ventricle extremely hypertrophied; right ventricle dilated; mitral valve slightly thickened.

Dropsy. Lardaceous Kidney.

0	Much	Much	5 mos.	3 years	4½ oz.	—	Normal.
2 pints	Some	17 pints	7 mos.	13 mos.	11 oz.	—	Normal.
0	0	0	Over 4 mos.	—	12 oz.	Edge of mitral thickened	Otherwise normal; no hypertrophy.

shown in section in the annexed figures.
of diarrhoea.

Name.	Age.	Kidney.	Weight of kidneys.	Urine.	Degree of dropsy.	Edema.
<i>Males :</i>						
J. P.	43	Pale, waxy, syphilitic	16 oz.	No polyuria; pale; highly albuminous	Trace	Trace
J. G.	42	Early lardaceous; syphilis and phthisis	—	Highly albuminous	Much	Much
G. B.	7	Large, white; stone in one; suppuration	9 oz.	Highly albuminous, pale; sp. gr. 1020	Much	Much
W. P.	40	Smooth, pale, lardaceous; disease general; syphilitic	—	Very scanty; much albumen	Much	Much
G. H.	35	Well-marked lardaceous; syphilitic	13 oz.	Excess; sp. gr. 1009; much albumen	Trace; latterly 0	Trace; latterly 0
J. S.	47	Large, lardaceous; phthisis	—	Diuresis; highly albuminous	0	0
P. F.	34	Large, white; nephritis superadded; syphilitic	—	Much albumen; nephritis superadded latterly	Slight cedema	Slight latterly
W. H.	17	Well-marked lardaceous; phthisis	16½ oz.	Scanty, highly albuminous; sp. gr. 1020	Much	Much
J. W.	36	Pale, cysted, lardaceous	17 oz.	Nearly solid with albumen; pale; sp. gr. 1012	None latterly	0
W. H.	30	Advanced lardaceous (dysentery)	12 oz.	Trace of albumen; sp. gr. 1010	0	0
A. P.	23	Early lardaceous (bronchiectasis)	18 oz.	Nearly solid with albumen	0	0
T. S.	12	Early lardaceous (psoas abscess)	11½ oz.	—	0	0
<i>Females :</i>						
L. B.	20	Advanced lardaceous; suppuration	9 oz.	Alkaline; highly albuminous; diarrhoea	Much	Much
S. F.	6	Not advanced; caries	6 oz.	"Fair quantity"	Some	Some
C. G.	28	Large, white; syphilitic	20 oz.	Excessive, pale; highly albuminous	0	0
L. A.	15	Well-marked lardaceous; caries	6 oz.	Albuminous; quantity not stated	Some	Trace
M. H.	22	Well-marked waxy; suppuration	21 oz.	Lithatic; highly albuminous	Edema; latterly 0	Some; latterly 0
E. M.	31	Well-marked lardaceous; syphilitic	20 oz.	Copious; low sp. gr.; highly albuminous	Latterly 0	Some; latterly 0
A. H.	36	Highly lardaceous; phthisis; large vomicae	13 oz.	Scanty; lithatic; highly albuminous	Much cedema	Much

* This mark indicates that the heart is

Pleural dropsy.	Pericardial dropsy.	Peritoneal dropsy.	Duration of dropsy.	Duration of disease.	Weight of heart.	Mitral regurgitation.	Heart, pericardium, infarcts.
0	0	0	Over 2 mos.	2 years	11 oz.	—	Pale, flabby; valves natural.
0	0	0	2 weeks	1 year	12 oz.	—	Pale; mitral atheromatous, aortic thickened.
0	0	Ascites	1 year	14 mos.	3 oz.	—	Small, firm, lardaceous
0	0	Much	4 mos.	4 mos.	8 oz.	—	Natural.
0	0	0	Trace 3 years ago	3 years	—	—	Natural in size and structure.
0	0	0	—	8 mos.	8 oz.*	—	Healthy.
$\frac{1}{2}$ pint	0	0	Recent	Over 3 years	8 oz.*	—	Healthy.
$\frac{3}{4}$ pint	0	1 pint	7 mos.	12 years	6 oz.*	—	Right side hypertrophied and dilated; valves healthy.
0	0	Some	Some 6 mos. ago	6 mos.	12 oz.	—	Pale, soft; patches of atheroma on left valves.
0	0	0	—	7 mos.	13 oz.	—	Natural.
0	0	0	—	2 years	12 oz.*	—	Normal.
0	Slight	0	—	2 years	5 oz.*	—	Valves healthy.
20 oz.	—	—	3 mos.	13 mos.	6 oz.	—	Healthy.
—	—	—	5 mos.	1 year	2 oz.	—	Slight deposits on aortic valve; otherwise natural.
0	0	0	1 year	8 years	6 dr.	—	Left ventricle thought to be hypertrophied.
0	0	Some	1 month	12 years	—	—	Natural.
—	—	—	3 weeks	6 mos.	11 oz.	—	Left ventricle slightly thickened; mitral valve beaded.
0	0	0	13 mos.	4 years	9 $\frac{1}{2}$ oz.	—	Valves natural.
0	0	0	5 weeks	2 years	7 oz.*	—	Recent vegetations on aortic; otherwise natural.

shown in section in the annexed figures.

The state of the heart is, of course, from post-mortem observation ; the condition as to dropsy, to be contemporary, is mainly post-mortem also, though I have included cedema present during the last few weeks, since this form of dropsy is more noticeable during life than afterwards. The cases are mainly from my own notes, and were taken without selection, as they presented themselves. In some the heart is displayed in section.

I have been in the habit for some time past of drawing sections of the heart in various conditions, and venture to bring some of the results before the Society. I began by procuring transverse sections of the ventricular region,¹ which were cut from the heart, mounted in glass, and then traced upon paper ; latterly I have been content to expose a section without detachment, trace it upon glass, and thence transfer to paper. I need scarcely add that great care was taken to make the section always at the same place and in the same direction, at right angles to the septum and as high up as practicable without cutting the valves. With so irregular a body as the heart some discrepancies in the fall of the section were probably inevitable, but nevertheless this method would appear not ill suited to demonstrate the thickness of the walls and the size of the cavities (see figures appended).

With these helps I will gather the more prominent facts from the pathological evidence. With simple nephritis there is hypertrophy, though the duration of the disease does not often allow this to become great. With children this was often extremely evident in the exaggeration of the columnæ carneæ, but it is difficult to express in relation to a fixed standard. After the age of eighteen and upwards, leaving out two cases which were complicated by the atrophy of one kidney, the heart of the male averaged just under 13 oz., that of the female just under 12 oz. If Peacock's estimation be accepted,

¹ Twenty-two such sections were made and mounted by Dr. Delépine, seven by Dr. Penrose. I desire to express my great obligations to those gentlemen.

and 10 oz. and 9 oz. taken as the respective weights of health, the increase with each sex under nephritis is nearly 3 oz. The sections show no general and obvious departure from the condition of health. In one (Maggie Grant) there was distinct dilatation, together with hypertrophy of both ventricles. As hypertrophy of the heart may be regarded as a sure sign of increased arterial tension, the state of the organ confirms the testimony of the sphygmograph in associating tension with nephritis. With both are conjoined scanty urine and abundant dropsy. Thus abundant dropsy is here associated with increased arterial pressure and slight cardiac hypertrophy.

Proceeding now to the granular kidney, this condition in relation to dropsy must be divided into two classes, one where the heart is little altered except by hypertrophy, the other where it has undergone changes which allow of mitral regurgitation. In the first class, that of simple hypertrophy, the heart of the adult male presents an average weight of $20\frac{1}{2}$ oz., that of the adult female one of $15\frac{2}{3}$ oz.

In section it is seen that there is enlargement of both ventricles, both by hypertrophy and dilatation. The preponderating change is hypertrophy of the left ventricle, but in some instances there is increase also of the cavity. The changes in the right ventricle are less marked, not always apparent. In some the cavity is enlarged without corresponding attenuation of the walls, so that some degree of hypertrophy must be inferred. The comparatively small degree of change in the right ventricle is apparent on contrasting renal hearts with those of valvular and other diseases. It will be seen that under renal disease the thickening of the left ventricle will bear comparison with that in any other condition; not so that of the right, which, although somewhat enlarged, is conspicuously less so than in some other states.

As regards simple cardiac hypertrophy with the granular kidney, some degree of dropsy was present in two thirds of the cases (sixteen out of twenty-four), more often in

trifling than considerable amount, so that the effusion is here small while the cardiac hypertrophy is great. As to the urine, this in many cases was excessive, but not constantly so. Roughly it varied inversely with the dropsy, being usually superabundant when dropsy was absent. Taking the cases where there had been such changes in the heart as to give rise to pulmonary apoplexy or a mitral regurgitant murmur, we find that the average weight of the heart of the adult male becomes 23 oz., that of the adult female 16 oz. The sections show (as the symptoms infer) that dilatation has been largely superadded to the hypertrophy. This is manifest in both ventricles, both processes being the more marked in the left. Dropsy now increases. "Much dropsy," instead of presenting itself in a sixth of the cases, is present in two thirds, and together with this scantiness of urine becomes the rule instead of the exception. In addition to the effects of renal disease we have those of valvular inefficiency, and the dropsy which is superadded is as much cardiac as renal. That the hypertrophied heart of renal disease is apt after a time to give way has long been recognised, but I am not aware that pulmonary apoplexy has attracted attention in this relation. I will venture to bring to notice one or two examples of this association in somewhat more detail than was possible in the table. One instance, that of George R—, has been already introduced (page 340 and Fig. 31).

CASE 1.—Eliza R—, aged 36, who had been in St. George's Hospital twenty-four years previously with scarlatinal dropsy, was at last admitted with much and increasing œdema, bronchitis, and dyspnœa. The urine was albuminous. The sounds of the heart were abnormal, the first short and sharp, the second markedly accentuated. I myself never detected a murmur, but Dr. Myers, the Medical Registrar, recorded one systolic at the apex. While in the hospital hæmoptysis presented itself, and continued somewhat freely for several days.

The dropsy increased, so that it became needful to puncture the legs; after much discharge they became erysipelatous, and finally gangrenous with a fatal conclusion.

The kidneys were found to weigh 10 oz.; their capsules were adherent, their surfaces roughened and lobulated. The cortices were somewhat diminished; they were of a pale reddish colour, and contained cysts. The heart weighed 15 oz.; the left ventricle was hypertrophied, and contained several masses of decolourised coagulum; all the valves were natural. The lungs were congested; in the lower lobe of the right were four masses of pulmonary apoplexy, each about the size of a walnut.

CASE 2.—John F—, aged 56, had albuminous urine, increasing œdema, much evidence of hypertrophy of the heart, which had a galloping rhythm involving a reduplication, and at times a soft, systolic apex murmur. Upon these ensued rather severe hæmoptysis, Cheyne-Stokes breathing, and ultimately much delirium. During life there had been doubt as to whether the symptoms were primarily renal or primarily cardiac. After death the only uncertainty was whether the case should be regarded as one of granular kidney with its results, or one of general fibrosis in which the kidneys participated. The kidneys were characteristically granular, though not extremely advanced in this state. They each weighed 7 oz.; they were slightly granular on their surfaces, generally cysted and fibrotic. The heart weighed 26 oz.; both ventricles were hypertrophied and dilated, the left most so. The orifices were dilated, especially the mitral and tricuspid; the valves themselves were healthy excepting a little thickening of the aortic, which, however, was not such as to interfere with its action. A section of the heart is represented (Fig. 33).

In the upper lobe of the right lung was a very large mass of pulmonary apoplexy, several inches in diameter; smaller patches of extravasation were scattered through-

out the left lung. The liver was congested and nutmeggy.¹

CASE 3.—Emma K—, aged 53, came into the hospital after two years of indefinite bad health and a fortnight of dropsy. The urine was scanty, lithatic, and albuminous to one half. The apex of the heart was displaced to the left, the sounds at first free from murmur, though afterwards a low systolic murmur presented itself at the apex. The œdema became deep and general, and, together with much dyspnœa, there was found to be fluid in the right pleura, and 60 oz. were taken away. Before the operation a small quantity of bright blood was coughed up, and more afterwards until the approach of death, ten days later. The kidneys were found to be red and granular, together weighing 10 oz. The heart weighed 17 oz. The left ventricle was much hypertrophied, and dilated with ante-mortem clot entangled in the muscoli papillares. The mitral valve had a healthy edge; there was some atheroma near its base of attachment, but not such as to interfere obviously with the action of the valve. The other valves were natural. The right ventricle was dilated and slightly hypertrophied, the right auricle hypertrophied and dilated and contained ante-mortem clot. The heart is shown in sections (Fig. 30). There were many masses of pulmonary apoplexy in both lungs; the largest occupied nearly the whole of the right middle lobe. The lower lobe of this lung was collapsed.

CASE 4.—Thomas S—, a potman, aged 24, was admitted into the hospital under Dr. Cavafy, to whom I am indebted for the use of the case. He had formerly been treated by Mr. Rouse for stone in the bladder. For six weeks before his last admission he had suffered from headache, vomiting, and shortness of breath. The urine was

¹ There were points of interest in this case to which I have not adverted. They have been described by Dr. Delépine, 'Path. Trans.,' vol. xli, 1890, p. 2.

abundant, of low specific gravity, at first slightly then more albuminous. He had no dropsy, but much bronchitis, and marked albuminuric retinitis with loss of sight. After death, which was preceded by much dyspnoea and delirium, a stone was found in the left kidney, which was atrophied, dilated, and granular. It weighed only 1 oz. The right kidney was also granular; it weighed 7 oz., and contained an old infarct. The heart weighed 18 oz. The left ventricle was much hypertrophied; the mitral valve showed incipient traces of atheroma, but the edge was normal and the valve practically so. The other valves were normal. The right ventricle was dilated and hypertrophied. The heart is shown in section (Fig. 28). The right lung was congested and oedematous, and presented an old infarct in the lateral aspect of the lower lobe over which the pleura was stained. In the lower lobe of the left was a grey pneumonic patch. The cæcum was extensively ulcerated, with blood in the mucous coat; there were also ulcers in the sigmoid flexure and in the duodenum.

In these cases the pulmonary hæmorrhage presents itself as essentially renal; it was not due to disease of the valves, but of the kidneys. Dilatation of renal origin allowed of regurgitation, to which the hypertrophy gave mischievous force. More commonplace evidence of mitral regurgitation with the advanced granular kidney, independently of disease of the mitral valve proper, is to be recognised in the development of a mitral regurgitant murmur in cases where no disease of the valvular structures has been found after death, though the orifices have no doubt been expanded. The tables furnish examples of this. I can also offer the following as a matter of purely clinical observation. With the advanced granular kidney, hypertrophied heart, hard pulse, and renal asthma, it has more than once happened to me to find a loud regurgitant mitral murmur under the asthmatic attacks when in the intervals there was none. The murmur has

TABLE VI.—*Pulse in Lardaceous Disease in relation to Dropsy.*

Sphygmograph.

Name.	Age.	Duration of symptoms when obser- vation made.	Dropsy.	Heart.	Greatest movement at — pressure.	Decided drop at — ounces.	No tracing beyond — ounces.
A. H. Phthisis	36	Edema 2 weeks 1 month 6 weeks 7 " 1 day later	Some edema on tibia Ditto Baggy edema at ankles Much, baggy about feet On the increase	Sounds short and sharp — — — Weight 7 ounces	3 3 3 2 2	4 5 4 4 3	5 7 6 6 5
W. H. Phthisis	17	Dropsy 6 months	Much baggy edema below knees	Right side dilated; weight 6 ounces	2	5	6
L. S. Phthisis	23	Phthisis 6 months	No dropsy	Cardiac dulness increased Pulse somewhat incompressible	R. L., 1	9 4	10 7
A. E. Syphilis	40	Occasional edema 7 years	Baggy edema about ankles. Face free	Much dulness —	2 —	6 6	6 6
M. S. Syphilis	33	Progressive 3 months	None	Dulness increased	1	5	6
S. G. Disease of hip; amputation	20	Disease of hip 6 years	Slight edema; 2 months of leg	No obvious change	1	4	7

been accompanied with palpitation and increased force of the pulse.

To add to the facts of renal dropsy before attempting to form any general conclusions, it is necessary to say a word about lardaceous disease. I regret that my opportunities of obtaining pulse-tracings in this condition have been few, though the post-mortem observations have been more numerous. The tracings show that arterial tension, so far as it has been examined, is generally below par, and this is the case even though œdema is present.

The low pulse-tension accords with the state of the heart as observed after death, which with adult males gives an average of $10\frac{3}{4}$ oz., with adult females one of $8\frac{1}{2}$ oz. These are so nearly the normal weights that it may be safely concluded that under lardaceous disease there is generally no hypertrophy. Looking at the heart in section, it would seem that this organ in lardaceous disease is weaker, rather than stronger, than in health; the wall of the left ventricle is somewhat thinner than in health, and the cavity of full size, as if under diastole or slightly dilated. Thus the observations of each kind, upon the pulse and upon the heart, present a general agreement, and appear to warrant the conclusion that whatever be the immediate cause of lardaceous dropsy, increase of arterial tension has nothing to do with it. The change special to the disease has the effect of narrowing the arterioles and presumably impeding the current within them, notwithstanding which, as has been seen, neither arterial tension nor cardiac hypertrophy results. If it be thought that these manifestations are in closer relation to the capillary system than to the arterial we may cease to marvel at the discrepancy. But another fact presents itself which requires explanation. Œdema may occur though the arterioles be thus narrowed and the blood-supply to the capillaries correspondingly diminished. If the œdema be due to excess of exudation we have this increase, while the blood-supply on which it depends is diminished.

This consideration, together with the position of the œdema, which affects the legs rather than the face, may suggest that the dropsy of lardaceous disease has at least some association with lessened absorption and cardiac failure; but further investigation is needed before any theory of lardaceous dropsy can be confidently advanced, further than that it is essentially different from other kinds of renal dropsy. As with all, it has a general inverse relation to the amount of urine. It is to be noted that lardaceous disease is sometimes obviously succeeded by nephritis, and the symptoms modified accordingly; but this is not common enough to interfere with the general statements which have been made.

To place together the conclusions which are led to by the several lines of the inquiry, it will probably be accepted as not admitting of doubt that—

1. Osmosis alone is not sufficient to account for dropsy, whether renal or of other kinds.

2. With the access of the dropsy of nephritis there is increase of arterial pressure. With a further increase of arterial pressure as the disease becomes chronic, and cardio-vascular changes are established, there is diminution of dropsy.

3. Finally, the heart gives way under the pressure to which it is subjected, with mitral regurgitation, pulmonary apoplexy, and increase or recurrence of dropsy.

All but the first are matters of simple observation, and none, I think, admit of question. As to the explanation of dropsy itself, and of the various changes which go with it, I desire to speak with a full consciousness of the difficulty and complication of the subject, and a sense that nothing can be as yet advanced which is more than provisional. With this admission I will revert in somewhat more detail to some of the points which have been touched upon, and will annex such inferences as present themselves.

Not only are the laws of osmosis or dialysis insufficient to explain the albuminous effusions of renal dropsy, but the similarity of these effusions to those which depend, as in heart disease, upon modifications of intra-vascular pressure suggests that their mode of origin cannot be entirely dissimilar.

The effects of osmosis are probably to be recognised in the presence of urea and sugar in dropsical effusions under certain circumstances ; and this process has been thought to be largely concerned in renal dropsy. Dialysable impurities may be presumed to be abundant in uræmic blood, and dialysis might be thought to be mainly concerned in the attendant dropsy, were not the effusions albuminous. So far as the mineral salts may be taken as the measure of dialysis, this would appear to be much the same in all kinds of dropsy. The connection between dropsy and uræmia as a matter of clinical experience is by no means simple ; for this state of blood may be extreme, even unto death, while serous effusion is conspicuously absent.

Anæmia and hydræmia are both to be considered in relation to renal dropsy. Anæmia is so often associated with this that it may be supposed to have some necessary connection with it. It is at least clear that both dropsy and anæmia can be traced, directly or indirectly, to the same cause, in impurity of blood. Anæmia unconnected with renal disease does not always give rise to dropsy, however extreme. Dropsy is usually entirely absent with idiopathic or pernicious anæmia, where it should be abundant could impoverishment of blood by itself give rise to it. With chlorotic anæmia œdema presents itself occasionally and to a slight extent. Here the anæmia is generally more marked than with renal disease, the dropsy less. But there *is* œdema sometimes, and here a consideration of some interest presents itself, though I desire to advance it with caution, as based as yet upon few observations. I found that with chlorotic anæmia, notwithstanding the prevailing debility, the tension of the

pulse was increased. This observation took me by surprise ; but so far as the evidence of eight cases and of Dudgeon's sphygmograph can be trusted, it appeared to be decided. Anæmia was marked in all, œdema present in five. At all stages there was evidence of more than normal arterial pressure, but it is enough to say that movement was obtained on an average up to a pressure of 9·6 oz., 2 oz. beyond the average of the nurse. In one of these cases of chlorotic œdema I may mention that the Medical Registrar discovered a patch of retinal hæmorrhage as another indication of undue arterial pressure. If further and more varied observations should accord with these, we may discern in chlorotic anæmia and the dropsy attending it something beyond mere poverty of blood, and find an analogy with renal disease. In both we have a toxic element, in one from deficiency of uterine discharge, in the other from a deficiency of renal, and we may infer in both capillary obstruction, with exudation as its accompaniment and arterial tension as its result.

We now come to hydræmia, theoretically distinct from anæmia, however associated with it clinically. Scantiness of urine is one of the most constant attendants of renal dropsy, though the companionship is not without exceptions. Urine may be totally suppressed, and that as the result of renal disease, without any trace of dropsy, as has been already pointed out ; while in disease not renal—in diabetes, for example—dropsy may present itself while the urine is superabundant. Thus the quantity of the urine does not bear a perfectly simple relation to œdema, whether renal or diabetic, and so far as scantiness of urine may be taken to imply hydræmia, the complication extends itself to this condition.

Insufficiency of urine, other evacuations and drink remaining unaltered, must result in retention of water in the blood, increased fulness of vessels, and presumably increased filtration through their coats. Where this is not apparent, as with obstructive suppression, it is possible that the vessels are relieved by other evacuations, notably

by vomiting. Taking all the facts together, considerable importance must be attached to hydræmia in connection with renal dropsy, though this condition *per se* does not appear sufficient to cause it. Cohnheim¹ has shown that blood diluted by nearly half its mass of a saline aqueous liquid may be passed through the vessels of a dog without any indication of œdema. I have already alluded² to a case of diabetic coma in which twenty-two pints of a watery solution were introduced into the circulation in thirty-two hours, with only traces of œdema and but little dropsy of any other kind. The legs, where the œdema was most marked, just above the ankle pitted only to the depth of an eighth of an inch. The small amount of dropsy which ensued upon this artificial and extravagant hydræmia appeared, as has already been shown, to be due rather to pressure than dilution ; more to increase in the contents of the vessels than to their watery character.

So far as hydræmia is connected with renal dropsy, it may be inferred from the albuminous character of the effusions that the process is rather by filtration, or extrusion under pressure, than by dialysis.

The obvious insufficiency of the current explanations of renal dropsy have led to the substitution of theories which I can but briefly advert to. According to the high authority of Cohnheim, acute renal dropsy deserves the description which was given to it before Bright associated it with renal disease, when it was termed inflammatory dropsy, and looked upon as a general inflammation of the subcutaneous tissue. For my own part, I think that the general absence of the ordinary signs of inflammation of the cellular tissue, together with the character of the effused fluid, is sufficient to negative this view. Dr. Waymouth Reid has made a suggestion which he has not fully developed, and which perhaps is scarcely mature for discussion. He has been led to suppose that the dropsy of kidney disease is brought about by some process allied to

¹ Cohnheim's 'Pathology,' vol. iii, p. 1279.

² See p. 322.

secretion on the part of the capillaries, and connected with "the protoplasmic activity" of their walls. He would thus regard the process as vital rather than physical; and would, I presume, look upon the effusion as vicarious, and the poisoned condition of blood as its excitant. If I am correct in thus presenting this view, it must at least be observed with regard to it that the vicarious secretion is often absent when the uræmic intoxication is most extreme, as with obstructive suppression, and sometimes with the advanced granular kidney. It is to be hoped that we shall hear more from Dr. Waymouth Reid on this matter. In the meanwhile it is certain that the dropsical process is not a perfectly simple one; if any such vicarious secretory action is concerned, it does not follow that other modes of action are to be excluded.

In acute renal dropsy (to limit attention for the present to this) the sphygmograph gives evidence of increased arterial tension, which is confirmed by the hypertrophy of the left ventricle.

Unless this arterial pressure is arrested short of the capillaries, we have a condition which must promote *exudation* from them; and the normal *absorption* also must be in many cases lessened by the inflammatory changes in the lungs, which with acute nephritis are the rule rather than the exception. But there are circumstances, among others the state of the right ventricle, which indicate that the part of the lungs and veins in renal dropsy is less important than that of the capillaries and arteries. It cannot be doubted that renal dropsy is essentially a matter of increased exudation, not diminished absorption, and that of this the capillaries are the seat. The process is connected probably with some change in themselves or in their relation to the blood, associated certainly with increased pressure behind them.

No argument is now necessary to connect the arterial tension and cardio-vascular hypertrophy with obstruction to the arterial exit. This must be either in the arterioles or the capillaries. If the obstruction is in the arterioles in the increasing dropsy of acute nephritis we are met with this

difficulty, that the capillaries receive less but they discharge more, at least by the lateral exit; less blood enters them while more lymph leaves them.

It is well known that the arterioles have a power of muscular contraction which is used largely in obedience to the nerves, and which regulates the supply of blood to the various territories of nervous and vascular distribution. But this is not all. Bright thought that the capillaries presented a hindrance to the blood in his disease, and the progress of knowledge since has done much to enforce his conclusion. It was long ago shown by Poisseuille that liquids traversed capillary tubes with different degrees of facility according to the composition of the liquids.

In the living body the processes of inflammation were shown to involve changes of which the capillaries were the especial, and apparently the primary seat, which modified their diameter and otherwise affected the passage of blood through them, conceivably by giving adhesiveness to their walls. Though muscular fibres were not to be recognised in their coats, it was shown by exact experiment¹ that the capillaries were not only elastic, but possessed of vital contractility; thus variations in capillary resistance and in capillary diameter were placed beyond surmise. Whatever the influence of the nervous system upon the arterioles, great as it may be, we cannot but infer that it is upon the capillaries that must be exerted the direct influence of the blood in its character and by its contact. The coats of these vessels are penetrated by the exuding serum, while the blood maintains only a superficial contact with the arterial channels. Accepting as a probability that the capillaries are directly influenced by the uræmic blood so as to oppose a hindrance, be it of what sort it may, to its passage, we can understand the increase of tension, the increase of exudation, and something of renal dropsy.

Proceeding to chronic renal disease and the granular

¹ Paper by Drs. Roy and Graham Brown on "The Blood-pressure and its Variations in the Arterioles, Capillaries, and Smaller Veins," 'Journal of Physiology,' 1879-80, part 2, p. 323.

kidney, we reach the apparent incongruity which has been stated in the diminution of dropsy with the further increase of arterial tension. With this association there is increased hypertrophy of the left ventricle, and thickening and narrowing of the arterioles. The inverse relation of the hypertrophy and the effusion, up to a certain point, is beyond question, however brought about.

The usual increase of urine must of course have its part in the process, but other conditions are certainly present, and must be taken account of. The thickening and constriction of the arterioles must cut off the blood from the capillaries, and presumably lessen pressure in them and exudation from them. The hypertrophy of the left ventricle with the increased thickness of its wall in relation to the cavity, as yet undilated, must give to this chamber an expansile power in diastole altogether beyond what is proper to it in health. The suction power thus increased must tend to draw upon the pulmonary circulation, and ultimately upon the veins and interstices, tending to pump the latter clear of any fluid which may have been effused. It is suggested that it is largely by this process, more or less of which would seem to be physically inevitable, that the antagonism between renal dropsy and cardiac hypertrophy is accomplished.

The giving way of the heart is the beginning of the end, and the return of dropsy its immediate result. The greatly hypertrophied ventricle, together with the mitral orifice, stretches, and mitral regurgitation occurs, enforced by a ventricular vigour unusual with this lesion. All the results of mitral regurgitation present themselves; pulmonary apoplexy frequently, and dropsy which may be truly called cardiac, though not cardiac *ab initio*.

Lardaceous disease presents itself as apart from the pathological series which has been traced. Increase of arterial tension and cardiac hypertrophy appear to have no concern with it. Excepting that the effusion varies more or less inversely with the amount of urine, it would not be safe to say more at present with regard to this result of

the disorder than that it is different in its origin from other kinds of renal dropsy.

Putting this conclusion aside, the points of resemblance and of difference between renal dropsy and cardiac dropsy come into prominence. In both the accumulation, as is well known, is essentially, however modified, what exudes from the capillaries and constitutes the lymph. Under heart disease there is diminished absorption without any presumption of increased exudation, or not to any important extent. Under kidney disease there is increased exudation without any necessary or constant diminution of absorption. Both are essentially connected with modifications of blood-pressure,—in one from obvious obstruction to the venous return, in the other from some presumably obstructive condition of the capillaries which is attended with enhanced exudation from them, and associated with increased pressure within the arterial system.

It only remains for me to record my obligations to those from whom I have received help which I have not already acknowledged—to my colleagues without exception for unrestricted access to and permission to use their cases; to Dr. Delépine for aid in many ways beyond what has been already mentioned; to Dr. Rolleston in connection with the pathological aspects of the inquiry; and to a series of excellent and interested clerks, among whom I must name Mr. Pickthorne, Mr. Wilson, Mr. Thomson, Mr. Romer, Mr. Goldsmith, Mr. Little, Mr. Elliott, and Mr. Woodhouse for frequent and willing assistance.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iv, p. 115.)

NOTE.—All the sections shown in the forty-seven figures on pages 375 to 385 were reduced in the same proportion (nearly one third), so that correct comparisons can be made both as to size and shape.

DESCRIPTION OF FIGURES 1—8.

Heart in Health.

Sections of the heart in health made as described in the text. The exact forms and dimensions were reproduced by tracing, and ultimately reduced by photography.

This plate displays eight hearts in varying states of systole and diastole. All were free from valvular disease and apparently healthy, obtained from cases of suicide, accident, and brief illness. The ages of the subjects range from seventeen years upwards. The weights of the hearts, counting only adults, including both sexes, varied from 7 ounces to 14 ounces.

FIG. 1.—Male, aged 24. Weight 12 oz. Suicide.

FIG. 2.—Male, aged 28. Weight 13½ oz. Accident.

FIG. 3.—Male, aged 17. Weight 11 oz. Accident.

FIG. 4.—Male, aged 21. Weight 8 oz. Perforation of bowel.

FIG. 5.—Female, aged 52. Weight 14 oz. Acute bronchitis.

FIG. 6.—Female, aged 42. Weight 11 oz. Poison.

FIG. 7.—Female, aged 26. Weight 7 oz. Perforation of stomach.

FIG. 8.—Female, aged 17. Weight 6 oz. Perforation of bowel.

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

FIG. 6.

FIG. 7.

FIG. 8.

DESCRIPTION OF FIGURES 9—17.

Nine similar Sections of the Heart in Nephritis.

The last two cases, Kybert and Saunders, present decided hypertrophy, but as these were complicated with antecedent atrophy of one kidney, it is probable that the cardiac change was partially due to this cause. Some of the remaining cases show slight hypertrophy, none very much. With the boy Kelly the hypertrophy is decided; with the woman Grant there is hypertrophy together with dilatation. In all but one there was dropsy, generally considerable where the hypertrophy was slight.

FIG. 9.—Violet Ellwood, aged 9 months. Weight $5\frac{1}{2}$ dr. No dropsy. Pulmonary apoplexy.

FIG. 10.—Florence Cox, aged $3\frac{1}{2}$ years. Weight $2\frac{1}{2}$ oz. Much dropsy.

FIG. 11.—John Kelly, aged 10 years. Weight 7 oz. Much dropsy.

FIG. 12.—John March, aged 12. Weight 6 oz. Much dropsy.

FIG. 13.—Maggie Grant, aged 29. Weight 14 oz. Much dropsy.

FIG. 14.—Maria Yockney, aged 50. Weight 10 oz. Much dropsy.

FIG. 15.—Arthur Maddison, aged 29. Weight $13\frac{1}{2}$ oz. Slight dropsy.

FIG. 16.—James Kybert, aged 48. Weight 28 oz. Left kidney atrophied. Much dropsy.

FIG. 17.—John Saunders, aged 32. Weight 15 oz. Right kidney atrophied. Much dropsy.

FIG. 9.

FIG. 10.

FIG. 11.

FIG. 12.

FIG. 13.

FIG. 14.

FIG. 15.

FIG. 16.

FIG. 17.

DESCRIPTION OF FIGURES 18—27.

Granular Kidneys without Signs of Cardiac Failure.

Ten sections from old cases of the granular or fibrotic and contracted kidney, not conjoined with murmur or pulmonary apoplexy as signs of cardiac failure. The hypertrophy of the left ventricle is generally well marked, sometimes extreme; that of the right ventricle absent or slight, though in some instances it is to be recognised, notably in those of Weber and Fowler. The absence or slight measure of the dropsy in connection with the larger hearts is noticeable. Worby appears to present an exception; his case was one of nephritis and dropsy recently imposed upon an old fibrotic kidney. Fowler, Weber, Windover, Copcutt, and Crann illustrate the rule.

FIG. 18.—William Worby, aged 61. Weight 25 oz. General dropsy.

FIG. 19.—Peter Copcutt, aged 35. Weight 18 oz. Trace of œdema.

FIG. 20.—Emily Smith, aged 35. Weight $11\frac{1}{2}$ oz. No dropsy.

FIG. 21.—George Windover, aged 58. Weight 22 oz. Slight œdema.

FIG. 22.—Henry Fowler, aged 43. Weight 23 oz. No œdema.

FIG. 23.—Charles Stocken, aged 58. Weight 13 oz. Slight œdema. Phthisis.

FIG. 24.—John Bartlett, aged 79. Weight 16 oz. Trace of œdema.

FIG. 25.—Edward Gooch, aged 44. Weight 10 oz. Dropsy.

FIG. 26.—Charles Weber, aged 64. Weight 16 oz. No dropsy.

FIG. 27.—William Crann, aged 47. Weight 26 oz. Trace of œdema.

FIG. 18.

FIG. 19.

FIG. 20.

FIG. 21.

FIG. 22.

FIG. 23.

FIG. 24.

FIG. 25.

FIG. 26.

FIG. 27.

DESCRIPTION OF FIGURES 28—33.

*Granular Kidneys, with Signs of consequent Mitral Regurgitation;
Valves natural.*

Six sections from cases of the advanced granular kidney with signs of cardiac failure; five with pulmonary apoplexy, one with a mitral murmur only. The hypertrophy in all is great and dilatation for the most part evident, most so in the cases of Ruddick, Spaul, and Killick. There was much dropsy in every case but one.

FIG. 28.—Thomas Spaul, aged 24. Weight 18 oz. Much dropsy. Pulmonary apoplexy.

FIG. 29.—Walter Gurwood, aged 65. Weight 21 oz. Much dropsy. Pulmonary apoplexy.

FIG. 30.—Emma Killick, aged 53. Weight 17 oz. Much dropsy. Pulmonary apoplexy.

FIG. 31.—George Ruddick, aged 55. Weight 24 oz. No dropsy. Pulmonary apoplexy.

FIG. 32.—William Jones, aged 66. Weight 29 oz. Much dropsy. Mitral regurgitant murmur.

FIG. 33.—John Ford, aged 56. Weight 26 oz. Much dropsy. Pulmonary apoplexy.

FIG. 28.

FIG. 29.

FIG. 30.

FIG. 31.

FIG. 32.

FIG. 33.

DESCRIPTION OF FIGURES 34—39.

Lardaceous Disease of the Kidneys.

Six hearts from cases of lardaceous disease of the kidney. In one, Fyndley, nephritis has been recently superadded. The general weight is low compared with healthy hearts belonging to the same sex and age, the left ventricle generally weak with diminished muscular substance and increased cavity. The contrast between the heart of lardaceous disease and that of the granular kidney, in regard to the power of the ventricle, is striking. It is not necessary to draw attention to the obvious circumstance that the disease antecedent to the lardaceous was generally of a wasting kind. Dropsy was present in three of the six cases, in one due to superadded nephritis.

FIG. 34.—John Sedgewick, aged 47. Weight 8 oz. Phthisis. No dropsy.

FIG. 35.—Alma Hunt, aged 36. Weight 7 oz. Phthisis. Much cedema.

FIG. 36.—Patrick Fyndley, aged 34. Weight 8 oz. Syphilitic caries. Slight dropsy latterly.

FIG. 37.—Thomas Scales, aged 12. Weight 5 oz. Caries. No dropsy.

FIG. 38.—William Hall, aged 17. Weight 6 oz. Phthisis. Much dropsy.

FIG. 39.—Alfred Perrin, aged 23. Weight 12 oz. Bronchiectasis. No dropsy.

FIG. 34.

FIG. 35.

FIG. 36.

FIG. 37.

FIG. 38.

FIG. 39.

DESCRIPTION OF FIGURES 40—47.

A selection of Hearts in various Conditions of Disease for comparison.

It is seen that the left ventricle with the granular kidney nearly equals in bulk that belonging to any disease primary to the heart. The right ventricle, on the other hand, presents greater hypertrophy under cardiac disease or malformation than ever under kidney disease. The greatest hypertrophy of the right ventricle is shown in the case of Pearce (Fig. 47) as the result of congenital disease. The right ventricle here is absolutely thicker than the left. Considerable hypertrophy of the right ventricle is also shown in connection with mitral disease and emphysema (Cairns, Fig. 40).

FIG. 40.—Martin Cairns, aged 58. Weight $15\frac{1}{2}$ oz. Mitral regurgitation, emphysema, and bronchitis. Much dropsy.

FIG. 41.—Edwin Rose, aged 36. Weight 24 oz. Pericardial adhesions. Bronchitis. Œdema.

FIG. 42.—Louisa Jones, aged 21. Weight 10 oz. Fatty degeneration. Anæmia. Œdema of feet.

FIG. 43.—Walter Banks, aged 28. Weight 33 oz. Aortic and mitral valves diseased. Little dropsy.

FIG. 44.—Maria Ware, aged 32. Weight 8 oz. Phthisis. No dropsy.

FIG. 45.—Georgina Hough, aged 50. Weight 27 oz. Mitral regurgitation. General dropsy.

FIG. 46.—George Paulger, aged 60. Weight 26 oz. Aortic stenosis. Œdema.

FIG. 47.—William Pearce, aged 19. Weight 20 oz. Congenital pulmonary stenosis. Perforation of septum ventriculorum. Phthisis. Slight œdema. The ventricle placed uppermost in the diagram is the right; it is seen to have absolutely thicker walls than the left.

N.B.—The thin line which appears on the outside of some of the hearts shows the amount of fat upon them.

FIG. 40.

FIG. 42.

FIG. 41.

FIG. 43.

FIG. 44.

FIG. 45.

FIG. 46.

FIG. 47.