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THE GLOMERULAR PRESSURE IN THE ISOLATED MAMMALIAN KIDNEY.

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Introduction.

The measurement of the glomerular pressure and its variations under experimental conditions is an essential preliminary to the physical analysis of the rate of formation of the glomerular fluid and its subsequent fate in the tubules. But even the less ambitious requirement of assigning a minimum value to the glomerular pressure, high enough to justify the filtration hypothesis, has induced many writers on the kidney to hazard a guess as to its value, based in part on the histological appearances of the lengths and diameters of the blood vessels concerned, and in part on the demands of the filtration hypothesis itself.

Ludwig [1844] recognized that his hypothesis necessitated an exceedingly high glomerular pressure, and founded his belief in this on histological observation, and on hydrodynamical considerations which will be mentioned in detail below but are quantitatively unimportant in this connection.

Two kinds of attempt to arrive at a more exact estimate of the glomerular pressure have been made, both of which depend on a rather drastic experimental intervention with the renal function, namely the identification of conditions which abolish the secretion of urine. First, the least arterial pressure consistent with the formation of urine has been shown to be in the neighbourhood of 40 mm. Hg [Ustomovitsch, 1870; Grutzner, 1875; Starling and Verney, 1925, and others], and it has been contended that the glomerular pressure in these circumstances cannot exceed this value, nor according to the filtration hypothesis can it be less than the 30 mm. represented by the osmotic pressure of the plasma proteins. It may be remarked, however, that the flow of urine more usually ceases if the arterial pressure falls below about 75 mm., even in the isolated kidney where there is no question of reflex vaso-constriction, and that the lower arterial pressure mentioned can

only produce urine if the blood contains large concentrations of some diuretic substance. Since neither the effects of diuretics on the glomerular pressure, nor the changes of glomerular pressure with arterial pressure are known, these experiments throw little light on the value of the glomerular pressure under more normal conditions.

The second method of arriving at the glomerular pressure depends on measuring the ureter pressure which just suffices to abolish the secretion of urine. Various values have been assigned to this pressure by different observers. For example, Loebell [1849] found 7–10 mm. Hg, Hermann [1859] 40–60 mm., and Heidenhain [1883] 64 mm.; Gottlieb and Magnus [1901] and Henderson [1905] occasionally obtained the latter value, but often found much lower values, and ureter pressures as high as 120 mm. Hg (B.P. 155 mm. in dog) have been recorded by Ozorio de Almeida [1927]. Now, according to the filtration-reabsorption hypothesis, when the urine flow is abolished by ureteral obstruction, the reabsorption of water and certain other substances proceeds until the concentration of substances to which the tubules are impermeable reaches a value such that the osmotic resistance brings the reabsorption process to a standstill. The liquid in the tubules is then stationary, and the ureter pressure is therefore equal to the intracapsular pressure, which is assumed to differ from the glomerular capillary pressure only by an amount equal to the osmotic pressure of the plasma proteins. The divers accounts of the maximum ureter pressure would suggest, therefore, that the glomerular pressure is a very variable affair. But the differences in the recorded values of the maximum ureter pressure probably depend mainly on differences in the quantities of diuretic substances employed by the observers, for this pressure undoubtedly increases with the concentration of no-threshold diuretics in experiments on the isolated kidney, though this relation is quite unaccountable in the simple terms of the filtration-reabsorption hypothesis given above. One must therefore either suppose that such diuretics produce their effect by increasing the glomerular capillary pressure, which is contrary both to the evidence included in the theory named and to my own preliminary observations, or confess that the relation between the maximum ureter pressure and the glomerular pressure is not yet understood.

The glomerular pressure in the mammalian kidney is, therefore, a quite unknown quantity. In the frog's kidney, however, it has been estimated by microscopic observation of the glomerular capillaries submitted to an external pressure just sufficient to obliterate them. L. Hill and McQueen [1921] put the pressure at 5–10 mm. Hg; Hayman

[1927], who applied the pressure through a micropipette inserted into the intracapsular space after blocking the tubule, obtained values varying from 4 to 52 cm. of water, which corresponded on the average to about one-half the pressure in the aorta. The hydrostatic conditions in the mammalian and amphibian kidneys are, however, widely different; this and other differences such as the presence of cilia in the tubules of frog's kidneys [Ebbecke, 1931] suggest that no close analogy between them can safely be assumed.

The filtration-reabsorption theory has been placed almost beyond dispute so far as the frog's kidney is concerned [Richards, 1929], but its application to the mammalian kidney is based on no such secure experimental foundations. Apart from the analogy, of somewhat uncertain validity, with the frog's kidney, the belief in glomerular filtration in the mammalian kidney rests mainly on the histological appearance of the glomerular membrane which has been supposed to be too simple in structure to enable it to perform a secretory function. The recent demonstration [Straub, 1929; A. V. Hill, 1930], that even so simple a membrane as that separating the yolk from the white of a hen's egg normally sustains large differences of osmotic pressure between its surfaces has, however, robbed this contention of much of its force. At present the filtration-reabsorption theory, like certain variants of the secretion theory, is unassailable as a possible hypothesis, but indefensible as a unique interpretation of the results of experiment on the mammalian kidney.

In putting forward a method of measuring the glomerular pressure, therefore, it is evident its results may be called upon to testify for or against the rival possible hypotheses of renal secretion; consequently it is a matter of crucial importance that the reasoning on which the method is based should not disguise an inference from one of these hypotheses. I believe that the argument given below is free from this objection.

THE MEASUREMENT OF THE GLOMERULAR PRESSURE.

Direct measurement of the pressure in the glomerular capillaries appears to be impracticable in the mammalian kidney owing to the inaccessibility to microscopic observation of the Malpighian corpuscles; these are situated too far from the surface of the organ for this purpose, and are additionally obscured by the liberal blood supply of the tissues surrounding them.

The present communication is concerned to show that, at least as a first approximation, a simple quantitative law relates the four

hydrostatic pressures which are of chief interest in connection with the secretion of urine:

$$\frac{\text{Glomerular pressure}}{\text{Arterial pressure}} = \frac{\text{Ureter pressure}}{\text{Venous pressure}},$$

where the ureter and venous pressures are so chosen that, taken one at a time, they will produce the same reduction in the rate of formation of the urine.

All the variables other than the glomerular pressure can be measured in the isolated mammalian kidney by methods detailed in previous communications [Winton, 1931 *a*, and *b*]. The value of the glomerular pressure so obtained is a statistical average rather than a measure of the pressure in any individual capillary; but this is hardly a disadvantage, for in correlations between the glomerular pressure and the rate and composition of the urine secreted, it is just such an average which is presumably the significant factor.

The derivation of the equation depends on the assumption that when the pressure in the renal vein is raised, the pressure in the renal artery being constant, the increment of pressure at any intermediate point in the renal circulation varies as the difference between the original pressure at that point and the pressure in the renal artery. Such a relation would hold for any series of rigid tubes connected end to end and traversed by a liquid at a velocity low enough to avoid turbulence. It would not hold accurately for a series of distensible tubes, such as constitute the renal circulation, but reasons will be given below for supposing that the error so involved can be neglected for our present purpose. There is no good reason to suspect turbulence in the blood flow in the kidney.

The application of this principle to the measurement of the glomerular pressure is illustrated in Fig. 1. The straight line PR, representing the fall of blood-pressure from the renal artery to the vein, defines the horizontal axis, which consequently bears an irregular relation to the anatomically linear scale along the path followed by the blood from the artery to the vein. If the glomerular pressure is OS, and equal to the ordinate QT, T will by definition represent the glomerulus on the horizontal scale. Now if the venous pressure be raised to RR', the fundamental assumption mentioned in the previous paragraph implies that the pressure gradient can be represented by a straight line PR', where the anatomical representation by the horizontal scale has undergone no change. The new glomerular pressure will thus be $Q'T$. By similar triangles PQQ' and PRR',

$$\frac{QQ'}{RR'} = \frac{PQ}{PR},$$

and by similar triangles *PQS* and *PRO*,

$$\frac{PQ}{PR} = \frac{PS}{PO}.$$

Hence

$$\frac{QQ'}{RR'} = \frac{PS}{PO},$$

i.e. the increment (ΔG) in the original glomerular pressure (G) is related to the arterial pressure (A) and the venous pressure (V) by the equation

$$\frac{\Delta G}{V} = \frac{A - G}{A}.$$

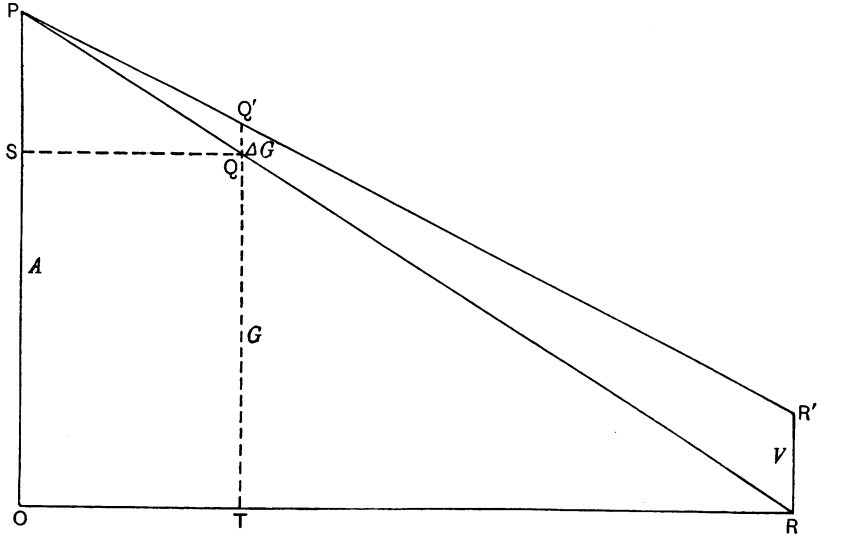

Fig. 1. Diagram illustrating the relations between the arterial, glomerular, and venous pressures in the kidney. Ordinates≡blood-pressure. Abscissæ≡an irregular anatomical scale extending from the renal artery (O) to the renal vein (R); the intermediate points are defined by the condition that the blood-pressure at any point lies on the straight line PR.

In a previous paper [Winton, 1931 *b*] it was shown that the increment in the glomerular pressure due to an increase of venous pressure can be expressed as the difference between the venous pressure and the ureter pressure which, taken one at a time, will produce the same reduction in the urine flow, that is:

$$\Delta G = V - U,$$

where V is the venous pressure that produces an increment ΔG in the glomerular pressure, and retards the urine flow as much as would a ureter pressure U.

Hence $$\frac{V-U}{V} = \frac{A-G}{A},$$

or $$\frac{U}{V} = \frac{G}{A},$$

which is the relation to be derived.

Experimental.

Direct verification of this relation is at present impossible for reasons already given. The degree to which it will yield predictable consequences can, however, be tested. Perhaps the most striking consequence of the relation is that the ratio of the equivalent venous and ureter pressures is a constant, independent of the degree of reduction of the urine flow which they bring about.

This prediction was put to the test in a series of experiments on the heart-lung-double-kidney preparation of the dog; the urine flow of one of the pair of kidneys was retarded by an increase of ureter pressure, and that of the other by an increase of the venous pressure. These pressures were varied at about the same time in such a way as to keep the rate of secretion of the two kidneys equal. The pressure in the renal arteries of both kidneys was kept equal and constant in any one experiment, and apart from this, the technical details of the experiments were exactly as described in previous communications [Winton, 1931 *a*, *b*].

The variation of the urine flow with progressive ureteral and venous obstruction in such an experiment is illustrated in Fig. 2. The numbers in the figure, representing the ratio of ureter to venous pressure, show that there is no systematic change in this ratio with the urine flow over a wide range of the rate of urine secretion. The product of this ratio and the arterial pressure is equal to the glomerular pressure of the kidneys, before the pressure in one of them had been raised by the increase of venous pressure. The experiment shows, therefore, that the glomerular pressure calculated in this way is independent of the absolute heights to which the ureter and venous pressures happen to have been raised in order to provide the data for its measurement.

The curves in Fig. 2 also demonstrate the characteristic independence between the urine flow and both the ureter and the venous pressures, when these pressures do not exceed a value usually in the neighbourhood of 10 mm. Hg. Within this region the ratio of the ureter to the equivalent venous pressure is indeterminate. Pressures large enough to abolish the urine flow also yield indeterminate ratios. Inside these boundaries the ratio of the ureter pressure to the venous pressure which retards the

urine flow equally is as nearly constant as can be expected in an experiment depending on the survival and parallel behaviour of two isolated organs.

The same constancy of the ratio of equivalent ureter and venous pressures is shown in Table I, where it is incorporated, under the heading of glomerular pressure, with the arterial pressure which remained at

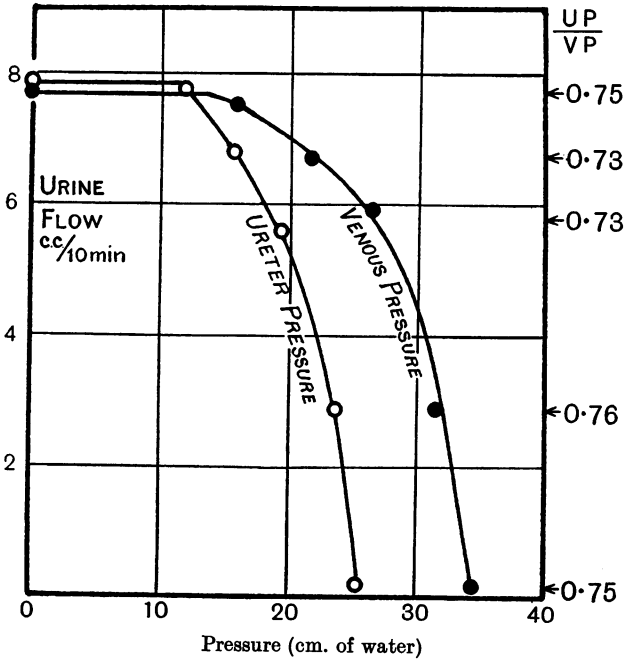

Fig. 2. Curves showing the variations of urine flow in a pair of kidneys, perfused by a heart-lung preparation, when the ureter pressure was raised in one kidney and the venous pressure was raised in the other. The values of the two pressures were adjusted at different levels so that the urine flows of the two kidneys were kept approximately equal. The ratio of the ureter and venous pressures, which produce the same change in the urine flow, is seen to be independent of the absolute heights of these pressures.

the same level in any one experiment. The table summarizes the successful experiments performed in this connection—successful in the sense that only if a pair of isolated kidneys secrete at equal rates and continue to do so for a considerable period do they provide suitable material for glomerular pressure measurements. Experiments indicating the degree of parallelism which may be expected in such a pair of kidneys under identical conditions were recorded by Canny, Verney and Winton [1930].

The experiments recorded in Table I provide representative values of the absolute glomerular pressure in kidneys isolated and surviving

TABLE I. Heart-lung-double-kidney experiments in which the urine flow of one kidney was reduced by raising the venous pressure, and that of the other by raising the ureter pressure. The pressures were chosen so as to keep the two urine flows approximately equal. In these circumstances the ratio of the ureter to the venous pressure is substantially independent of the urine flow, and equal to the ratio of the glomerular pressure to the arterial pressure.

			Urine flow (c.c./10 min.)			
Exp.	Venous pressure (cm. aq.)	Ureter pressure (cm. aq.)	Cf. venous pressure	Cf. ureter pressure	Glomerular pressure (mm. Hg)	Arterial pressure (mm. Hg)
1	0	0	7·7	7·9	—	117
	15·7	11·7	7·4	7·7	88	
	21·5	15·6	6·7	6·8	86	
	26·2	19·2	5·9	5·6	86	
	31·2	23·7	2·9	2·9	89	
	34·2	25·5	0·2	0·2	88	
2	0	0	4·2	4·2	—	110
	18·0	10·5	2·7	2·9	61	
	23·0	12·5	2·0	1·8	60	
	27·0	15·5	1·6	1·5	63	
3	0	0	3·7	3·6	—	100
	41·3	32·5	1·0	1·0	79	
	37·8	30·0	1·8	1·8	79	
	34·3	26·2	2·0	1·9	76	
	29·5	21·6	2·3	2·2	73	
	23·7	19·0	2·5	2·4	80	
	17·4	13·7	2·9	2·8	73	
	0	0	3·2	3·0	—	
4	0	0	6·4	6·3	—	120
	29·5	21·5	2·2	2·4	88	
	22·7	16·5	0·5	0·5	88	
	13·7	10·0	0·3	0·4	88	
5	0	0	12·0	11·8	—	116
	20·5	12·0	8·1	8·1	68	
	21·2	14·9	7·0	7·0	72	
	29·1	17·2	6·1	6·2	68	
	35·1	20·3	5·3	5·2	67	
	40·2	26·0	3·6	3·6	75	
6	0	0	3·2	2·8	—	120
	24·1	15·0	1·6	1·2	75	
	18·7	11·5	2·1	1·7	74	
	31·4	20·2	0·8	0·6	78	
	0	0	2·0	1·6	—	
7	0	0	3·2	3·3	—	116
	15·5	10·5	1·8	2·0	79	
	12·5	8·5	3·0	3·1	79	
	14·8	10·2	2·2	2·3	80	
	18·0	13·2	1·5	1·6	85	
	20·5	15·0	1·1	1·2	85	

under the conditions specified. In certain experiments the ureter and venous pressures were progressively raised, while in others, they were

first raised to a value which nearly abolished the urine flow and then progressively lowered; both procedures yielded unchanging values for the calculated glomerular pressures at different periods of the experiment. The glomerular pressure may therefore be regarded as approximately constant for periods of one or two hours in the circumstances named, and independent of the degree to which the urine flow has been reduced.

As might be expected, the values of the glomerular pressure obtained in different experiments varied considerably; as a rough approximation, however, the pressure in the glomerular capillaries may be taken as about two-thirds of that in the renal artery; the standard deviation from this value in the seven experiments described is about 10 p.c., and indicates the variation among kidneys taken from different animals.

The analysis of the thirty individual values of the glomerular pressure can provide an estimate of the random error which is involved when the glomerular pressure is measured in this way. To this end, the deviation of each value from the mean glomerular pressure for its particular experiment is calculated, and the root-mean-square of these differences obtained. The standard deviation of the glomerular pressure is thus found to be 2 mm. Hg, and indicates the magnitude of the uncertainty due to imperfection of the experimental technique. The smallness of the experimental error is in part due to the steepness of the relevant parts of the pressure-flow curves shown in Fig. 2.

A HYDRODYNAMIC MODEL ILLUSTRATING SOME PRESSURE-FLOW RELATIONS IN THE ISOLATED KIDNEY.

In order to render the implications of the fundamental pressure equation of the kidney vivid enough to be of use in the further analysis of renal activity, I have found it convenient to consider a model such as that shown in Fig. 3. In it the pressures representing the arterial, glomerular, venous, and ureter pressures can be varied independently and measured, and the impedances corresponding with the vas afferens, the vas efferens, and the glomerular membrane can be controlled by screw clips. The "reabsorption" of water from the tubules is arranged to be little affected by small changes of "ureter" pressure by making its rate the resultant of the pull of a long column of water and a small adjustable orifice; in the absence of any such "reabsorption" the "ureter" pressure would have to reach the "glomerular" pressure before urine flow ceased. In the kidney itself the ureter pressure would have to reach the intracapsular pressure before it abolished the urine flow

unless there were withdrawal of water from the tubules by some route other than the ureter.

The significant relation between the distal ends of the tubules and the venules embracing them is simulated in the model by allowing the "urine" to pass through a thin-walled rubber tube before emerging into

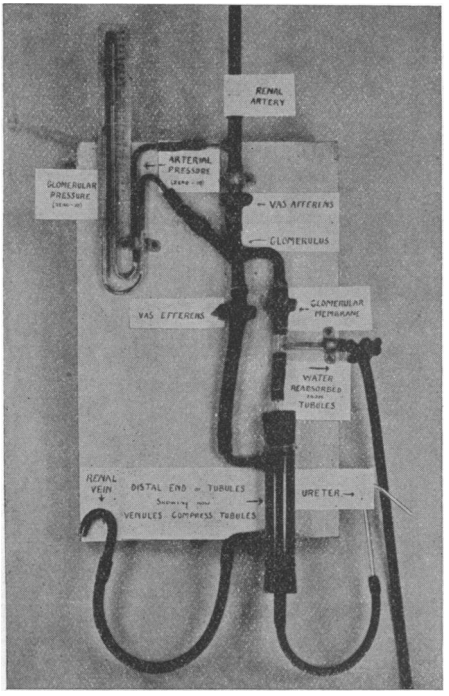

Fig. 3. Model showing how the fundamental pressure relation arises in the kidney. The model is an arrangement of glass and rubber tubes with water flowing through them. Screw clips represent the impedances due to the vas afferens, the vas efferens, and the glomerular membrane. The arterial and glomerular pressures are each recorded by mercury manometers, and the venous and ureter pressures can be varied by raising or lowering the respective outlets.

the "ureter," and by enabling the "blood" between the "vas efferens" and the "vein" to pass through a chamber surrounding this rubber tube. This is effected by an arrangement like the arterial resistance in the heart-lung preparation [Knowlton and Starling, 1912], modified, at the suggestion of Dr L. E. Bayliss, so as to render the closure of the rubber tube complete even with small external pressures, by making two short longitudinal slits in the middle segment of the tube, and then

occluding them by sticking the sides of the tube together with rubber solution.

In the model, as in the kidney, an increase of glomerular pressure increases the urine flow, while an increase of ureter pressure decreases it. A rise of venous pressure can be seen to increase the pressure recorded by the glomerular manometer, and yet to reduce the urine flow by compressing and so raising the back pressure in the "tubule." Hence the pressures applied to the vein and ureter, which produce the same retardation of the urine flow, are different, the venous pressure exceeding the ureter pressure by an amount equal to the increase of glomerular pressure associated with the former. An increase of ureter pressure has a negligible effect on the glomerular pressure owing to the urine flow being relatively slow compared with the rapid blood flow from the vein.

The action of drugs on the kidney can be simply illustrated on the model. Caffeine increases the glomerular pressure, the urine flow, and the blood flow: loosen the screw-clip "vas afferens." Low concentrations of adrenaline increase the glomerular pressure and the urine flow, but decrease the blood flow: tighten the screw clip "vas efferens." Higher concentrations of adrenaline reduce the glomerular pressure, the urine flow, and the blood flow: constrict the clip "vas afferens." The no-threshold substances increase the urine flow by retarding the reabsorption of water from the tubules: constrict the clip controlling the withdrawal of water. Pituitary extracts reduce the urine flow by increasing the rate of reabsorption of water: release the same clip. Substances, if such there be, which affect the permeability of the glomerular membrane, produce effects comparable with those of varying the appropriate screw clip. The experiments on which this summary of the action of drugs on the isolated kidney is based will be described in future papers.

This model is something more than the purely analytical class of model, such as that illustrating the tension-length-time relations in plain muscle [Winton, 1930]. Such models are intended only to describe the exact relations between the variables concerned, with no implied reference to how in fact the living organ brings about those relations. The model of the kidney here proposed, on the other hand, is less exact in the simulation of the quantitative properties of the kidney, owing to the turbulence set up where the water passes the screw clips; but it purports to bring about the relations concerned by anatomically parallel means, and to render visible the changes of glomerular pressure which are inaccessible to direct measurement in the kidney. Despite the drawback mentioned, screw clips have been preferred to stream-line resistances on

account of the facility with which they enable the effects of changes in the different resistances to be studied.

Discussion.

The experimental errors involved in pressure measurements of this kind have been found to be small, but the systematic difference between the pressure computed in this way and the true glomerular pressure can for the moment only be assessed by a scrutiny of the likely sources of systematic error in the fundamental assumptions concerned. Later it is hoped to amplify this estimate of the accuracy of the glomerular pressure measurement, by discovering whether consistent correlations can be established between its variations under experimental conditions and properties of the kidney which are believed in part to depend on it.

In the simplifying abstraction on which the reasoning underlying the calculation of the glomerular pressure was based, certain properties of the kidney were falsified. For example, both the uriniferous tubules and the renal blood vessels were treated as rigid rather than distensible tubes. This introduces two sources of error:

(1) An increase of venous pressure produces a greater change of blood-pressure near the venous end of the circulation than near the arterial end; the distension of the blood vessels near the venous end will be greater than that near the arterial ends; the associated increase of glomerular pressure will therefore be less in the kidney than it would be in a rigid structure. This error will increase with increasing values of the applied venous pressure, and is in the direction corresponding to the observed value of the venous pressure being too high in proportion to the hypothetical rise in glomerular pressure.

(2) An increase of pressure in the distal ends of the tubules dilates the tubules only when it is effected by increase of ureter pressure, and not at all or much less so when the pressure in the whole kidney is also raised by increase of venous pressure; this is shown by histological observation of kidneys fixed after obstructing their ureters or veins; consequently there is a relative reduction in the pressure difference between the capsule and the ureter when the pressure in the latter is raised. The estimate of the increase of glomerular pressure, due to an increase of venous pressure, in terms of the difference between that venous pressure and the ureter pressure which retards the urine flow equally, is therefore in error in the sense that the measured ureter pressure is too high. The magnitude of the error will increase with increasing values of the ureter pressure.

In the calculation of the glomerular pressure the ureter and venous pressures appear as a ratio; the two classes of error considered result in the observed values of both of these being too high by amounts which vary with the absolute values of each. If the error in each pressure were the same percentage of that pressure—a not unlikely approximation—the value of the ratio between the two pressures would be exactly the same in the rigid system from which the pressure law was derived, and in the kidney which deviates from this system by including distensible tubes. The word "error" is used in this argument to refer to this deviation.

Since it has been shown experimentally that the ratio of the equivalent ureter and venous pressures is in fact independent of their absolute values, it may be inferred that these errors do not affect the value of the ratio, and that they are either too small to produce detectable consequences, or equal and opposite in their effects on the value of the glomerular pressure obtained by this method. A peculiar relation between these factors, which would not affect the value of the ratio is possible, but hydrodynamically quite unlikely. This contention may be taken to cover and to dispose of certain other possible "errors" which would vary with the value of the applied ureter or venous pressures.

If Ludwig's proposition, that the height of the glomerular pressure is in considerable part due to the conversion of kinetic energy of the blood to potential energy, were true, this would introduce another class of error. However, if typical values of the diameter of the renal artery and of the blood flow, characteristic of the isolated kidney in these experiments, be taken, and even if it be supposed that the whole of the kinetic energy of the blood in the renal artery has been converted into potential energy represented as hydrostatic pressure in the glomerular capillaries, only about 2 mm. Hg can be attributed to this factor. As the reduction of blood flow due to the increase of venous pressure occurring in these experiments is small, the influence of this consideration on the method described can be regarded as an error of the second order.

In conclusion it may be noted that the values of the glomerular pressure, amounting to about two-thirds of the arterial pressure, would be accepted by advocates of the filtration-reabsorption hypothesis as adequate to justify the hypothesis; but as methods of estimating the fall of pressure across the glomerular membrane and along the tubule are perhaps now in sight, it would seem unprofitable to speculate in detail on whether it will equal or exceed the values found for the glomerular pressure.

The simple law relating the four pressures considered above may not have reached its final form, and may have to be corrected if its consequences prove to deviate appreciably from quantitative properties of the kidney yet to be studied. In the absence of other sources of information, however, it provides a means of estimating the glomerular pressure and its variations, which may be employed with some confidence in the further analysis of the behaviour of the kidney without prejudging the issues raised by the classical rival hypotheses concerning renal secretion.

Summary.

1. The law governing the hydrostatic pressures concerned in the formation of urine may be expressed approximately in the form:

$$\frac{\text{Glomerular pressure}}{\text{Arterial pressure}} = \frac{\text{Ureter pressure}}{\text{Venous pressure}},$$

where the ureter and venous pressures are so chosen that, taken one at a time, they induce the same reduction of urine flow.

2. This relation is deduced from the effects of raising the ureter and venous pressures and inferences from them previously recorded, and from an approximate hydrodynamic treatment of the blood circulation in the kidney.

3. The theoretical treatment leads to the prediction that, other things being equal, the ratio of the ureter and venous pressures, equivalent in the above sense, should be constant, whatever the degree of the reduction of the urine flow. This was verified in experiments on the heart-lung-double-kidney preparation of the dog, which are shown to justify the main premises on which the treatment was based.

4. This pressure relation provides a means of measuring the glomerular pressure and its variations. In the isolated kidney the glomerular pressure is about two-thirds of the arterial pressure. The standard deviation corresponding to the random experimental error is 2 mm. Hg. Some possible systematic errors are considered.

5. A model is described which illustrates how these pressure relations are brought about in the kidney and how drugs influence the rate of formation of urine.

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