URINARY OUTPUT IN CARDIAC DECOMPENSATION

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Urinary output is a variable index of water balance which Rowntree defines, "as the daily relation between the total amount of water entering the organism through the ingestion of liquids and food and the total output of water lost from the body by way of the kidneys, bowels, lungs and skin. In the intake must be included the water of oxidation." The amount of fluid gained through the water of oxidation and the amount lost through the feces may be considered constant, for all practical purposes, when the diet is maintained on roughly the same amount of carbohydrates, fats, proteins and calories each day, given in a fluid, semi-fluid or solid form.

The major portion of fluid excretion occurs through the kidneys and through the skin and lungs. The ratio between these two varies tremendously and is influenced especially by increased activity and increase of temperature. If, however, the patient is controlled by absolute bed rest in a room of fairly constant temperature and he is fever free, the fluid loss in a well person through the skin and lungs may be considered to be roughly constant from day to day and that variations in fluid output will manifest themselves largely in the urinary output.

Addis and Watanabe[2] have observed a considerable variation in urinary excretion from day to day in normal persons. Atwater and Benedict[3] found the water intake with subject in repose to range from 880 to 2440 c.c., an average of 2290 c.c. for forty nine days and with moderate work from 2225 to 4550 c.c., an average for sixty six days of 3700 c.c. The urine output ranged within normal limits from 800 c.c. to 3000 c.c., the average being usually between 1200 to 2000 c.c. Van Valzah, Mc Kinley and Middleton[4], using this data, have arbitrarily stated that the expected urinary output normally approximates sixty per cent of the fluid intake and

that any increase of the output above this point may be termed diuresis.

Cardiac decompensation is always associated with a marked disturbance of the circulation which makes itself evident by variable degrees of impairment of the vital organs and very commonly by the appearance of peripheral edema.[5] Van Valzah, Mc Kinley and Middleton quote from Loeb's (Monograph, Oedema): "Dilatation of the capillaries and veins due to faulty functioning of the circulation is the principal cause of the increased transudation and the subsequent edema in cardiac decompensation, but there may perhaps be in addition an increased filtration pressure responsible for these results. The vascular dilatation is apparently followed by an increased permeability of the capillaries. Retention of sodium chloride plays a role also in the case of cardiac edema, although it is a smaller one than in the case of renal edema. In certain stages of cardiac edema a hydremia seems to occur. As to the occurrence of a true plethora, the statements are contradictory. In addition to the increased transudation, there is a delayed absorption and an alteration in the relations determining the exchange of fluid between tissues and blood vessels. Experimentally produced myocarditic lesions may give rise to lowering of blood pressure and a diminution in the amount of urine under certain conditions, but so far it has not been possible to influence by this experimental method the amount of edematous fluid."[6] Celsus made one of the first observations of importance on the subject of dropsy on which he wrote, "Nor is it improper to measure both the drink and the urine; for if more fluid is excreted than is taken, so at length thereis hope of good health."[7] Schwab, Herrmann and Stone again emphasize the importance of the kidney in the removal of edematous fluid. They are of

the opinion that this circumstance can be combated more successfully by the proper use of diuretics than by mechanical removal of large quantities of fluid in the cases of general anasarca.[8] Richards in an excellent review of kidney function states the views held by Ludwig in 1833. He maintained that "An increment of blood pressure, uncomplicated by increment in velocity or volume of blood flow in the kidney, increases the urine." He also recognized the importance of complete and partial obstruction of the renal vein which he said caused such swelling of the veins of the kidney that tubules were compressed and their lumens narrowed dependent upon the degree of obstruction.[9] Sollmann later considered that stasis in the renal vein caused an obliteration of the filtration space in the capsule of Bowman.[10] Starling established optimal blood pressures for filtration.[11] In later years Eyster and Middleton and others have observed that as the venous pressure rises urinary output decreases and as it falls, diuresis ensues.

This study is limited to those cases of cardiac decompensation which are either arteriosclerotic or rheumatic in etiology. Those of congenital, myxedematous, thyrotoxic, luetic and hypertensive origin are completely excluded. Only cases which were definitely decompensated and which had adequate clinical records for at least seven days were included. Cases dying before the seventh day and cases with coincidental severe ailments which in themselves would modify the course of the disease were excluded, e.g. subacute or acute bacterial endocarditis, carcinoma or advanced tuberculosis.

Routine orders in practically all instances included the Karell or a slightly modified Karell diet, which necessarily restricted fluids to

approximately 1200 c.c. As the patients improved semi-solid and solid food was gradually added, but no allowance was made to include the extra water derived. The daily weight charts were not possible because of mechanical difficulties. Absolute bed rest (without bathroom privileges) was stricly enforced.

This series included 214 cases, which may be classified as follows:

	Surviving	Dying
Arteriosclerotic		
Edematous	78	19
Non-edematous	12	0
Rheumatic		
Edematous	64	22
Non-edematous	18	1

The non-edematous have been discarded. The records of the edematous will be examined under the two etiologic headings. There is very little difference in average age between the fatal and surviving group, but average time of decompensation in the rheumatic group is definitely at a much lower age.

	Surviving	Dying
Rheumatic heart disease	43.7 yrs.	44.0 yrs.
Arteriosclerotic heart dis.	62.6 yrs.	66.7 yrs.

The average stay in the hospital was:

Rheumatic group	37 days	41 days
Arteriosclerotic group	35 days	31 days.

Tables IA and IB give the number and the percentage of the urinary

outputs of the cases recorded above and below sixty per cent of the fluid intake, the point above which has been taken arbitrarily to denote diuresis. This data has been taken from the records of the first twenty one days of hospitalization.

Tables IA and IB chart the daily incidence of the cases with outputs above the sixty per cent level reduced to percentages.

Both graphs show three major characteristics. First, with institution of hospital care, in both the rheumatic and arteriosclerotic heart disease, there is a marked tendency for an initial diuresis, which is more marked and more prolonged in the patients who eventually become compensated. Secondly, the daily output in the surviving cases in both types tends to become relatively fixed at a high rate with but little variation from day to day. Whereas both fatal groups tend to vary greatly from day to day. Thirdly, the majority of the surviving cases show good diuretic response on all but three days. But in the fatal arteriosclerotic group the majority of cases showed diuresis on only seven days and in the rheumatic group only five days.

Tables IIA and IIB group the actual number of cases occurring in various levels of the output-intake ratio percentages. From this, the actual percentage of the output-intake ratio for each type of heart disease as a whole was determined for each day under the two headings - surviving and dying - and plotted on Graphs IIA and IIB.

TABLE IA

ANALYSIS OF THE OUTPUT-INTAKE RATIO IN ARTERIOSCLEROTIC HEART DISEASE .

Days	1		2		3		4		5		6		7		8	
Termination:	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D
	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:
Above 60%	12:43:	1:17:	28:62:	5:29:	41:61:	11:58:	56:80:	10:59:	55:76:	6:43:	54:75:	7:50:	55:81:	9:56:	50:69:	5:36:
Below 60%	16:57:	5:83:	17:38:	12:71:	26:39:	8:42:	14:20:	7:41:	18:24:	8:57:	18:25:	7:50:	13:19:	7:44:	23:31:	9:64:

Days	9		10		11		12		13		14		15		16	
Termination:	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D
	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:
Above 60%	51:75:	5:45:	49:70:	3:30:	42:67:	5:38:	45:67:	5:38:	52:74:	6:43:	47:69:	5:50:	38:63:	3:30:	45:75:	3:27:
Below 60%	17:25:	6:55:	21:30:	7:70:	21:33:	8:62:	22:33:	8:62:	17:24:	8:57:	21:31:	5:50:	22:37:	7:70:	15:25:	8:73:

Days	17		18		19		20		21	
Termination:	S	D	S	D	S	D	S	D	S	D
	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:	No: %:
Above 60%	37:70:	5:45:	40:73:	4:45:	32:64:	3:50:	28:70:	1:11:	25:61:	4:50:
Below 60%	16:30:	6:55:	15:27:	5:55:	18:36:	3:50:	12:30:	8:89:	16:39:	4:50:

S - Surviving
D - Dying

Above 60%) Output-Intake Ratio
Below 60%)

TABLE IB

ANALYSIS OF THE OUTPUT-INTAKE RATIO IN RHEUMATIC HEART DISEASE

Days	1		2		3		4		5		6		7		8	
Termination	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D
	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %
Above 60%	8:31	6:40	27@54	11:58	40:72	8:42	73:41	8:42	38:63	8:42	45:75	10:50	45:72	5:26	39:53	6:33
Below 60%	18:69	9:60	24:46	8:42	16:28	11:58	15:27	11:58	22:37	11:58	15:25	10:50	18:28	14:74	27:47	12:67

Days	9		10		11		12		13		14		15		16	
Termination	S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D
	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %
Above 60%	25:38	7:58	32:54	6:43	27:51	7:47	28:53	6:43	27:54	9:56	26:52	1: 7	29:61	6:50	24:51	1:10
Below 60%	41:62	5:42	25:46	8:57	26:49	8:53	25:47	8:57	25:46	7:44	24:48	13:93	19:39	6:50	23:49	9:10

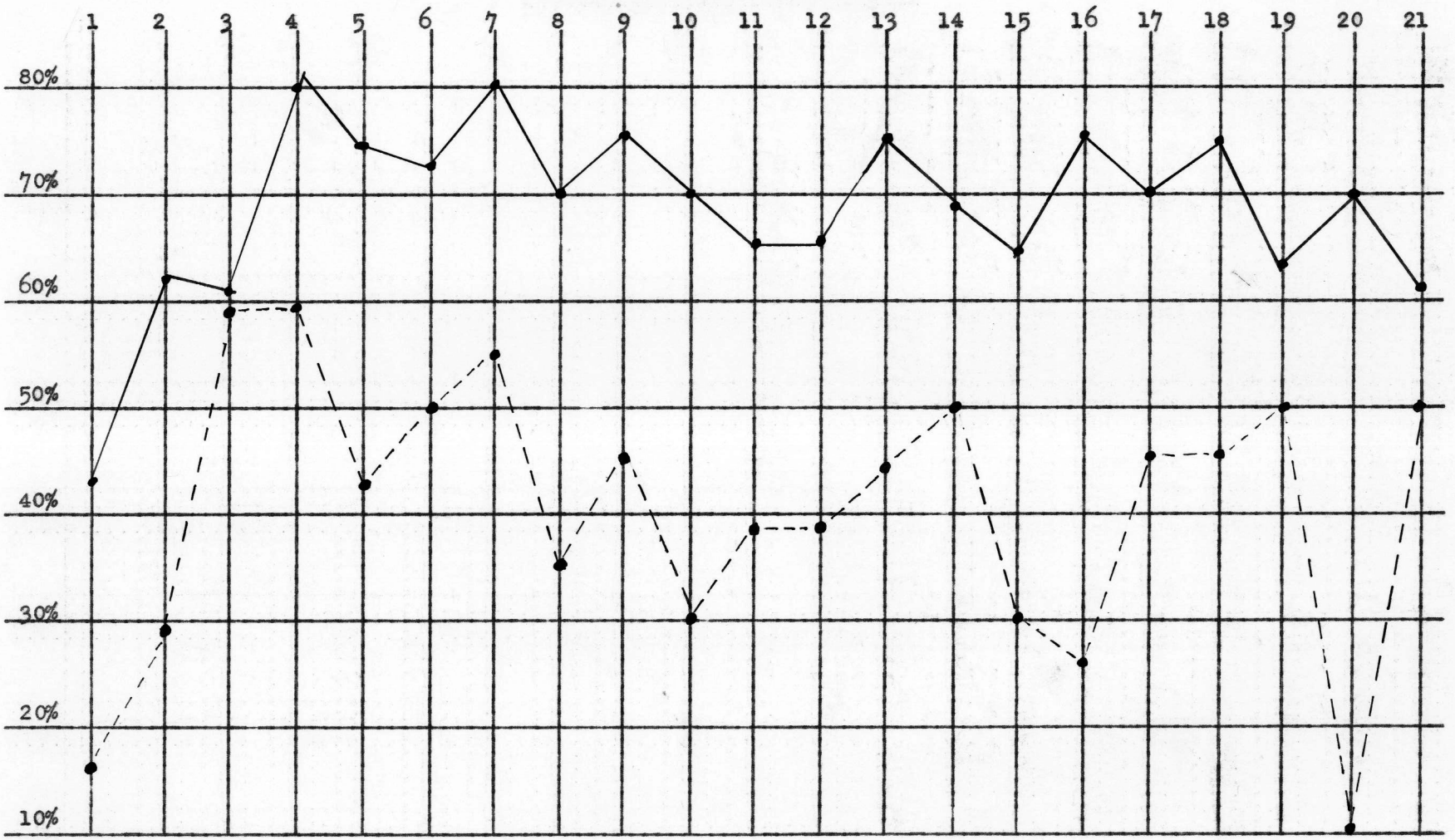
Days	17		18		19		20		21	
Termination	S	D	S	D	S	D	S	D	S	D
	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %	No: %
Above 60%	30:65	2:15	31:70	4:45	28:64	2:20	28:67	4:40	25:61	3:27
Below 60%	16:35	11:85	13:30	5:55	16:36	8:80	14:33	6:60	16:39	8:73

S - Surviving
D - Dying

Above 60%-Output-Intake Ratio.
Below 60%

GRAPH I A

ARTERIOSCLEROTIC HEART DISEASE



GRAPH I B

RHEUMATIC HEART DISEASE

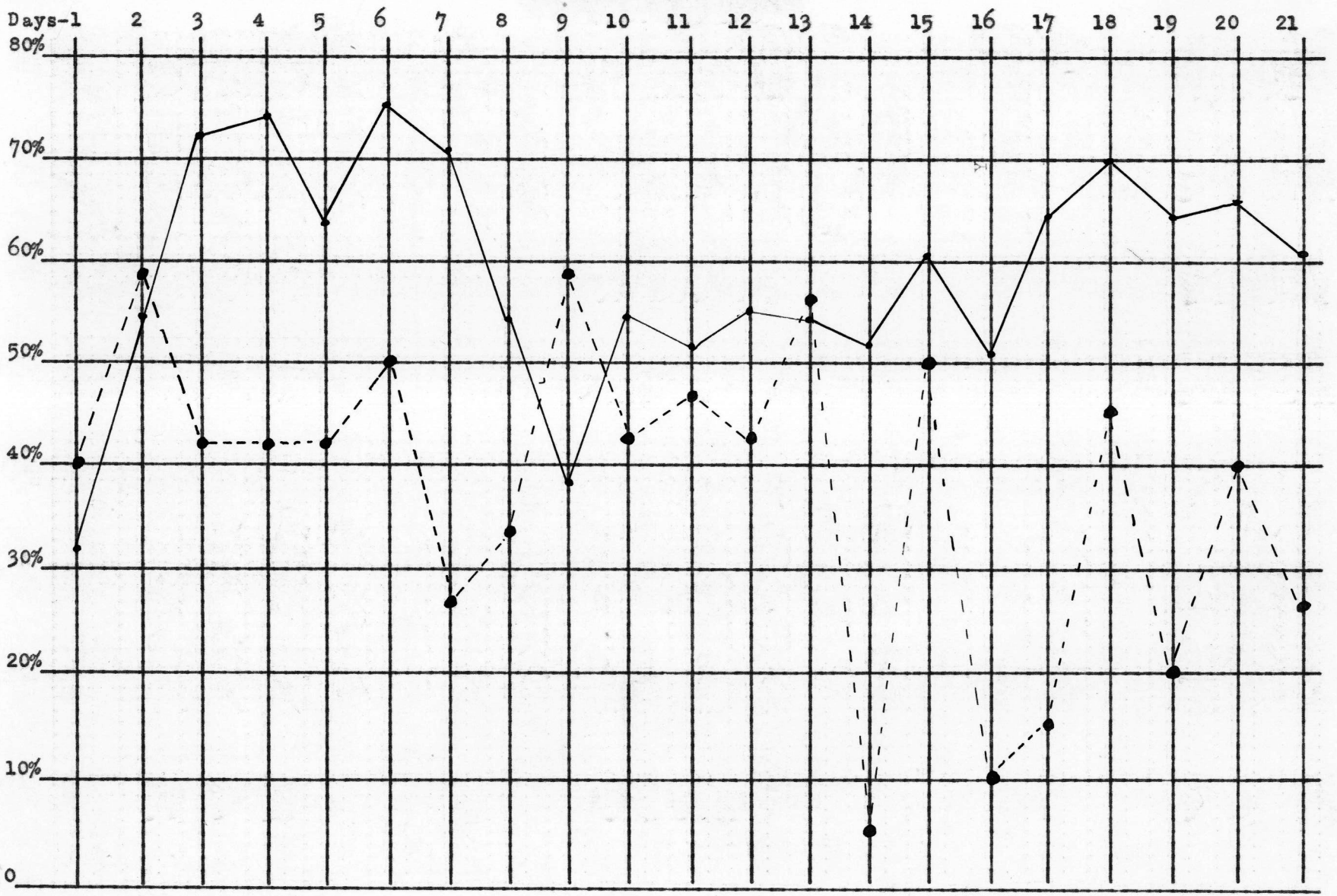


TABLE II A

ARTERIOSCLEROTIC HEART DISEASE

Day		1		2		3		4		5		6		7		8	
Termination		S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D
O-I Ratio	100% +	8	1	19	2	23	2	37	3	39	1	40	1	39	2	37	2
	80-99	1	0	2	2	8	3	4	2	5	1	3	1	4	2	6	2
	60-79	3	0	7	1	10	6	15	5	11	4	11	5	12	5	7	1
	40-59	8	3	7	5	17	6	4	3	8	4	12	3	10	0	10	2
	20-39	6	1	10	6	8	1	6	4	8	3	6	3	3	6	9	3
	0-19	2	1	0	1	1	1	4	0	2	1	0	1	0	1	4	4

Day		9		10		11		12		13		14		15		16	
Termination		S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D
O-I Ratio	100% +	28	2	29	2	24	2	28	2	29	1	28	1	22	1	18	1
	80-99	16	0	9	1	10	1	7	0	12	0	4	1	6	1	10	1
	60-79	7	3	11	0	8	2	10	3	10	6	13	3	10	1	17	1
	40-59	13	3	14	3	12	4	8	5	6	5	13	1	8	3	8	2
	20-39	3	3	6	4	9	4	12	2	10	3	8	4	11	3	6	6
	0-19	1	0	1	0	0	0	2	1	1	0	0	0	3	1	1	0

Day		17		18		19		20		21	
Termination		S	D	S	D	S	D	S	B	S	D
O-I Ratio	100% +	18	3	19	1	17	0	6	0	8	1
	80-99	7	0	8	2	3	0	7	0	9	1
	60-79	12	2	13	1	12	3	15	1	8	2
	40-59	9	2	9	1	13	2	8	4	10	1
	20-39	6	4	5	3	5	1	4	4	5	2
	0-19	1	0	1	1	0	0	0	0	1	1

O-I Output-Intake Ratio

TABLE II B

RHEUMATIC HEART DISEASE

Day		1		2		3		4		5		6		7		8	
Termination		S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D
O-I Ratio	100%+	5	2	9	2	19	5	20	4	19	3	20	1	27	3	13	3
	80-99	2	2	6	3	9	1	9	1	6	2	11	2	7	0	9	1
	60-79	1	2	12	6	12	2	12	3	13	3	14	7	11	2	8	2
	40-59	12	4	12	1	9	4	9	4	12	7	9	2	10	4	18	8
	20-39	4	4	10	6	5	4	6	5	7	3	6	5	8	8	8	1
	0-19	2	1	2	1	2	3	0	2	3	1	0	3	0	2	1	3

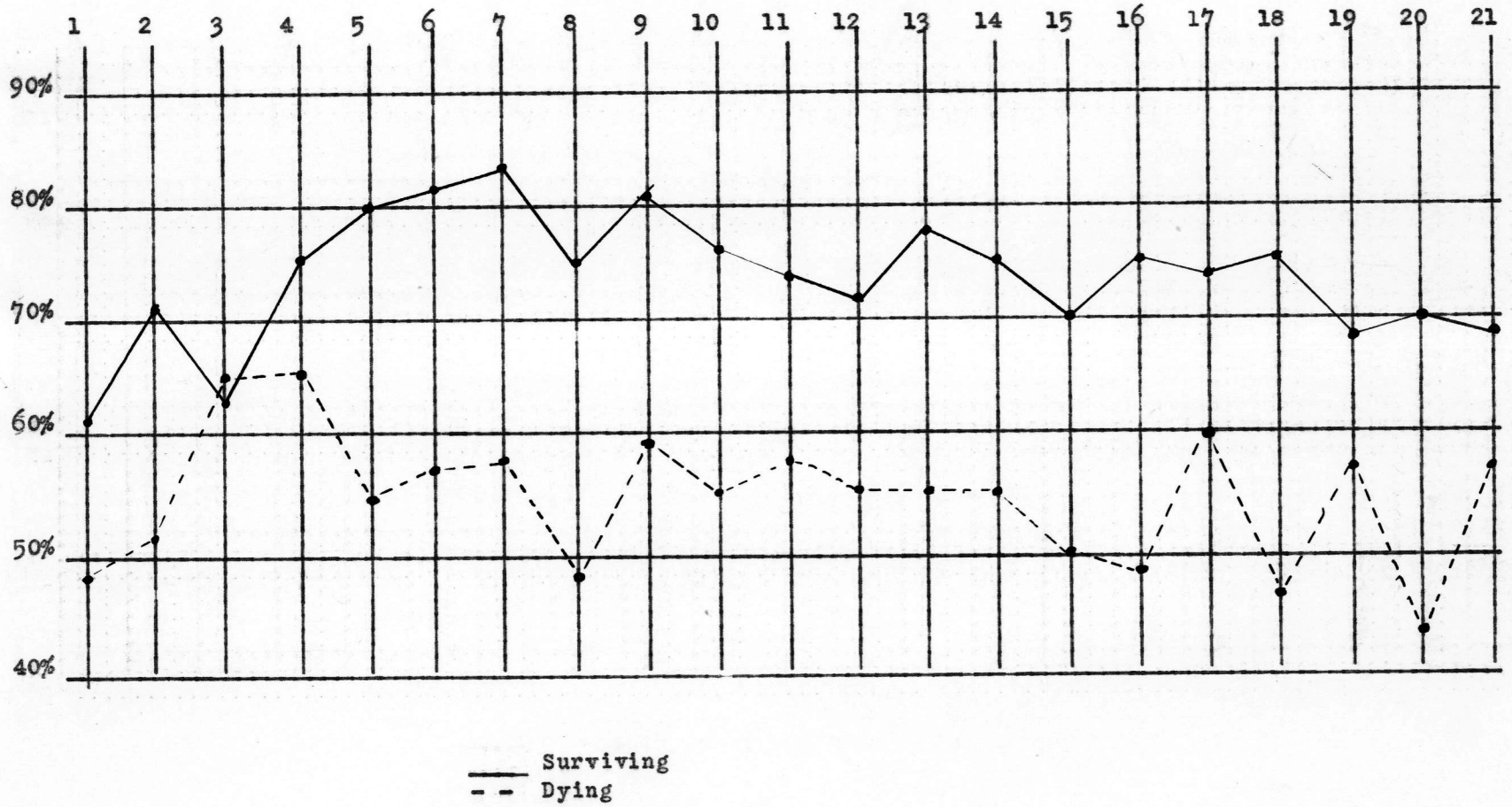
Day		9		10		11		12		13		14		15		16	
Termination		S	D	S	D	S	D	S	D	S	D	S	D	S	D	S	D
O-I Ratio	100% +	14	5	13	2	8	2	10	2	10	3	11	0	9	1	11	1
	80-99	4	0	10	2	10	4	2	2	9	1	10	0	8	3	2	0
	60-79	7	2	9	2	9	1	16	2	8	5	5	1	12	2	11	0
	40-59	21	1	11	3	16	2	11	4	14	0	14	10	8	3	14	4
	20-39	19	8	13	3	10	2	12	3	7	5	7	3	8	2	7	4
	0-19	1	2	1	2	0	4	2	1	4	2	3	0	3	1	2	1

Day		17		18		19		20		21	
Termination		S	D	S	D	S	D	S	D	S	D
O-I Ratio	100% +	11	0	13	3	12	1	14	3	12	2
	80-99	6	1	9	1	3	0	3	0	6	0
	60-79	13	1	9	0	13	1	11	1	7	1
	40-59	8	5	5	1	10	2	9	2	7	4
	20-39	7	5	6	3	4	5	4	3	9	3
	0-19	1	1	2	1	2	1	1	1	0	1

O-I - Output-Intake Ratio.

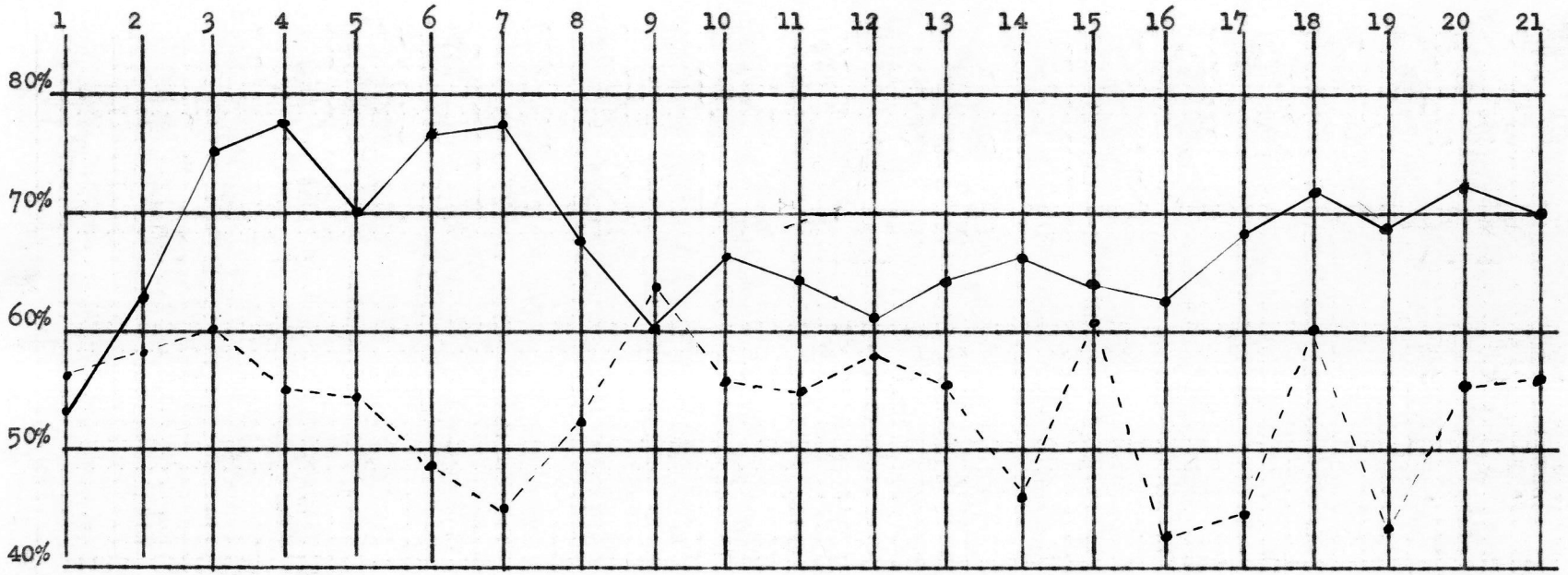
GRAPH II A

ARTERIOSCLEROTIC HEART DISEASE



GRAPH II B

RHEUMATIC HEART DISEASE



___ Surviving

- - Dying

FACTORS IN DIURESIS

REST: It will suffice to state that in cardiac decompensation the heart needs every possible aid to preserve what little reserve is left. Rest is an indispensible means of conserving this reserve by decreasing the demand on the heart by eliminating all but the necessary activities Absolute bed rest, without bath room privileges, was routine in this series with gradual resumption of activity following compensation.

DIET: The Karell diet has been the standard dietary control of edematous, decompensated patients at the Wisconsin General Hospital, except when contraindicated. This includes a daily fluid intake reduced to around 1200 c.c., salt poor, and low protein diet. This regimen is popular throughout the country. However, Herrmann[12] raises the question of an oligoproteinemia commonly present in this condition and wonders if this may not be further increased with resultant increase of edema by restrictions on the protein intake.

DIGITALIS: Since the time of William Withering digitalis has been unexcelled in its use for cardiac decompensation. In his "An Account of Fox Glove and Some of its Medical Uses with Practical Remarks on Dropsy" he speaks of the diuretic, emetic and purgative effects of its use. As a guide to dosage and evidence of toxicity he writes, "Let the medicine therefore be given in the doses and intervals mentioned above; let it be continued until it either acts on the kidneys, the stomach, the pulse, or the bowels; let it be stopped upon the first appearance of any of these effects."

The administration of digitalis will not be discussed further in this paper. However, it will be pointed out that digitalis is not to

be considered as a diuretic per se. Rowntree believes that the direct action of digitalis by stimulation of the vagus would tend to decrease the urinary output and that the diuresis obtained from its use is the direct result of a more effective circulation. This view point is shared by Hatcher[13] and many others, who report that diuresis with digitalis whether direct or indirect occurs only in the presence of edema.

DIURETICS: The mercurial and xanthine diuretics will be considered together. Schwab, Herrmann and Stone have accepted the "Modern Theory of Renal Function of Cushing" as basis for their recent work. Briefly this contention embraces parts of the theory of Bowman and Heidenhein and the Filtration-Diffusion theory of Ludwig. They consider that "The glomerular capsule, as the result of pure physical force, acts as a filter, allowing all of the constituents of the plasma, except colloidal, to pass through it without altering their relative concentrations. This filtrate which may be regarded as deproteinized plasma enters the tubules where active absorption takes place."[14] They have used Rehberg's modification, which adds another process in the tubules namely back diffusion, to study the effects of the various diuretics in their series. By the Rehberg method, creatinine filtration rate, they have determined that the xanthine diuretics act to increase glomerular filtration to a point which exceeds the customary rate of tubular absorption and diuresis ensues. By the same method they determined that the creatinine filtration rate is not increased by the use of mercurial diuretics. They attribute the diuresis thus obtained primarily to impaired tubular reabsorption. Working on this theory they have found that the daily use of a xanthine diuretic supplemented with a mercurial diuretic every two to four days as necessary

gave much better results than when either of these two were used alone or separately. Theophylline sodiosalicylate, Theocin and salyrgan, both used intravenously, have been most efficacious in their hands.

C. Smith and J. R. Wiseman[15][16] have reported on the use of salyrgan for periods of three and five years in patients without ill effects. However, Willins[17] reports venous thrombosis with long continued use of salyrgan intravenously. Novasurol has been proved to be a very good diuretic, but it is often accompanied by toxic symptoms. Mercurin has found much favor used in suppository form by virtue of the ease of administration.

In this series of cases, the use of xanthine and mercurial diuretics was in most instances successful after bed rest and digitalis had failed. There was no noticeable difference of action on the rheumatic and in the arteriosclerotic cases, but the series is small and no generalizations can be made. No data is available for use of the xanthines supplemented by the mercurial diuretics, but both appeared to be quite efficacious when used separately.

VENOUSYPRESSURE: The mechanical load that an increase of venous pressure places on the kidney function has been sufficiently discussed. Eyster and Middleton, in a large series of hospital patients without cardiac affliction, have determined that normally the pressure in the peripheral veins does not rise above 11 cms. of water. It has been the practice at the Wisconsin General Hospital to perform routine venesection of at least 500 c.c. when the venous pressure rises to 20 cms. of water, and is accompanied by signs of respiratory embarrassment. In this series venesection, in many instances, was the impetus which enabled the patient

to acquire compensation. Diuresis was obtained in practically every case, except six cases in which there was no appreciable fall in venous pressure, five of these terminated fatally and in the other instance an insufficient quantity was removed.

PARACENTESIS: The removal of transudations from serous cavities when present in sufficient quantities to cause some mechanical load to cardiac action or respiratory excursion, was in many instances successful in contributing to a good diuretic effect. In most cases several days elapsed before any appreciable diuresis could be noticed.

SCARIFICATION: Multiple scarifications were used in too few cases in this series to draw any conclusions. No diuretic effect was noticed but the patients did obtain some subjective relief.

ARTERIOSCLEROTIC HEART DISEASE

DRUG	ADMINISTRATION	No.	RESULTS		
			+	?	0
Theocin	First	23	17	1	5
	Repeat	5	5	0	0
Diuretin	First	14	10	3	1
	Repeat	3	3	0	0
Salyrgan	First	3	2	0	1
	Repeat	5	3	1	1
Salyrgan with Ammon. Chloride	First	3	3	0	0
	Repeat	8	7	0	1
Mercurin	First	0	0	0	0
	Repeat	0	0	0	0
Mercurin with Ammon.Chlor.	First	2	2	0	0
	Repeat	3	2	0	1

RHEUMATIC HEART DISEASE

DRUG	ADMINISTRATION	No.	RESULTS		
			+	?	0
Thevetin	First	12	8	0	4
	Repeat	4	3	0	1
Diuretin	First	8	3	4	1
	Repeat	1	0	1	0
Salyrgan	First	2	1	1	0
	Repeat	0	0	0	0
Salyrgan with Ammon. Chlor.	First	2	2	0	0
	Repeat	3	2	1	0
Mercurin	First	1	0	0	1
	Repeat	0	0	0	0
Mercurin with Ammon. Chlor.	First	2	1	1	0
	Repeat	2	2	0	0

RESULTS OF VENESECTION

Arteriosclerotic Heart Disease:

	Administration	No.	RESULTS +	?	0
Surviving	First	13	11	1	1
	Repeat	1	1	0	0
Dying	First	1	1	0	0
	Repeat	0	0	0	0

Rheumatic Heart Disease:

	Administration	No.	RESULTS +	?	0
Surviving	First	5	4	1	0
	Repeat	0	0	0	0
Dying	First	10	5	1	4
	Repeat	4	2	1	1

PARACENTESIS

Arteriosclerotic Heart Disease:

	Amount C.C.	Termination	No.	Results +	?	0
Thoracentesis	870	Surviving	25	16	1	8
		Dying	2	1	1	0
Abdominal Paracentesis	4,300	Surviving	5	4	0	1
		Dying	1	0	0	1

Rheumatic Heart Disease:

	Amount C.C.	Termination	No.	Results +	?	0
Thoracentesis	870	Surviving	10	7	2	1
		Dying	6	2	2	2
Abdominal Paracentesis	4,300	Surviving	4	3	1	0
		Dying	0	0	0	0

CONCLUSIONS

1. The removal of edematous fluid by diuresis indicates the re-establishment of effective renal circulation, which reflects general improvement in the systemic circulation as a whole.

2. The presence of a well sustained diuresis in the edematous decompensated cases encourages a more favorable prognosis.

3. As a group the daily variations in the output-intake ratio among the surviving cases do not fluctuate markedly, whereas there is marked variation in those cases terminating fatally.

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