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28

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> DEPARTMENT OF THE ARMY Fort Detrick Frederick, Maryland

INVESTIGATION ON THE ELIMINATION OF A!KALI SALTS

by E. Salkowski Assistant Physician of the Medical Clinic, Koenigsberg

Virchow's Archiv, Vol 53, 1871, pp. 209-234.

I have already reported elsewhere that our knowledge of the elimination of alkali salts is practically nil. There exists e.g. only scant knowledge on the alkali salt content of urine, the most important excretion from the point of view of our understanding of metabolism while Vogel's investigations on the phosphoric acid [content] of urine comprise, according to him¹, more than 1,000 determinations. It is clear that there is thus a great gap here: I cannot hope to fill it with my research, but hope to stimulate interest, and herewith contribute a first paper myself.

The reason for the lack of data on the material under discussion is no doubt due to the fact that alkali determinations in biological fluids give reliable results only when they are carried out carefully, with exact control of the reagents, of distilled water, etc. They are also very time-consuming. Furthermore the potassium determinations with platinum chloride require special precautions because of the ammonia content of the air. I was fortunate enough to be able to use, for my investigation, rooms [laboratory space] into which nobody entered besides myself. Only a small portion of the analyses were carried out in the laboratory of the clinic, but even there nobody else was working at that time, and thus I was sure that the above difficulties were avoided.

The investigations included healthy individuals (i.e. 1) Neubauer + Vogel, 1 <u>Harnanalyse</u> (Urine Analysis) p. 335.

- 1 -

28

free of fever) at various states of food intake and individuals having fever (with partial inanition). They refer to all excreta which I recognized to be sources of considerable elimination: urine, feces, saliva, sputum and blood serum.

Experimental Procedure

1.) For the alkali determination in urine I essentially followed the method of Neubauer, introducing some modifications.

Urine was precipitated with an equal volume of barium hydroxide mixture. 40 cc of the filtrate, corresponding to 20 cc of urine, was measured out. 2 to 3 times this volume was used for very dilute urine. This causes a small error which also applies to the urea determinations and which up to now has been overlooked or neglected. The error is that the volume of the precipitate is not taken into account and is set equal to zero. The error is the greater the more concen-trated the urine. If it is to be avoided one has to wash the precipitate until the filtrate is free of all solid components, and one has then to use the total amount for analysis. Since this procedure makes the determination still more difficult, I have felt justified to neglect the not too considerable error. - It is self evident that in the case of very concentrated urine, whe e one is not sure that the amount indicated is sufficient for precipitation, one has to test whether the filtrate becomes cloudy upon further addition of the barium hydroxide mixture. If my memory is correct I never encountered such a case, (the barium hydroxide and barium nitrate solutions were supersaturated). If the urine is already alkaline, a known volume is evaporated on the water bath until all the ammonium carbonate is driven off and the liquid has become acid. It is then diluted to the initial volume.

According to Neubauer the filtrate should then be evaporated on the water bath, dried and calcined. If I proceded in this manner there always occurred a very violent, sudden combustion or even a true explosion and the material was thus lost. In order to avoid this, I strongly acidified the filtrate with hydrochloric acid and then evaporated; the hydrochloric acid displaces a large fraction of the nitric acid and the combustion proceeds entirely without or only with very minor explosions. Attack of the platinum dish by the mixture of nitric and hydrochloric acid did not occur at this very great dilution. It would indeed be much simpler to use barium chloride as precipitant; I only proceded in the above manner because I had prepared a large amount of pure barium nitrate which I did not want to go to waste. When - as happened quite

- 2 -

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often - the residue burned incompletely, I wetted it with several drops of dilute nitric acid (1 vol sp. g. 1.2 + 5 vols. water) dried it and repeated the calcination. This step depends on skill and experience; if one takes too little nitric acid it does not accomplish its purpose, if one takes too much, an explosion occurs and the analysis is lost. In spite of all precautions this does happen from time to time, and it is perhaps advisable to leave out the precipitation with barium hydroxide mixture altogether.

I dissolved the calcination residue in dilute hydrochloric acid while warming slightly (Neubauer indicates only extraction with boiling water), mixed with ammonia and ammonium carbonate, washed with hot water until the sodium reaction was negative, evaporated to dryness, at moderate heat, in a porcelain dish. Towards the end the material was acidified with hydrochloric acid to drive off the nitrous acid which might still remain. The residue was dissolved in water, transferred to the platinum dish etc. The alkalis were weighed as chlorides. Potassium was always determined by means of platinum chloride, since the indirect titration with silver solution yields inaccurate results because of the small total amount of alkali chlorides.

I paid the greatest attention to the purity of the reagents and to that of the distilled water; each time I used new batches I tested them for purity. This is indeed a very boring but a very essential job since one often finds impurities one never even suspected.

I obtained the barium hydroxide commercially in pure form except for a trace of lime which does not interfere. The barium nitrate can easily be purified by repeated recrystallization of the commercial chemical which always contains soda. The platinum chloride introduces errors most readily. I have had difficulties to obtain commercially pure platinum chloride (I purchased it from three reputed manufacturers); with care, however, it can easily be purified.

The evaporation and calcination was always carried out in a platinum dish. The filtrate after the first precipitation with ammonium carbonate could not be evaporated in a platinum dish because of the large volume involved - such a procedure would have increased the work tremendously. This introduces a small error since the ammonium chloride solution absorbs during the boiling a trace of alkali from the porcelain; this error, however, is so small that it can be neglected in good faith.

- 3 -

The analysis miscarries most often during the calcination of the alkali chlorides because losses may occur through decrepitation. This handicap can only be avoided by very lengthy drying on the water-bath. Special heed has also to be paid to the fact that what is weighed as alkali chlorides really is that. The solution is always somewhat cloudy small amounts of dust etc. suffice to make it so. This, however, has no effect on the accuracy of the results. That the solution should be entirely clear, as required by Fresenius, is, in my opinion, a demand which cannot be realized in practice.

As far as the collection and designation of urine is concerned, I and W. collected samples from 8 AM to 8 AM the next day. A urine carrying the date 23 refers to a sample collected from the morning of the 23rd to the morning of the 24th. Otherwise the periods are always from 2 PM to 2 PM the next day, and urine with the date 23 is then a sample collected between the afternoon of the 22nd to the afternoon of the 23rd. This also applies to sputum and feces. Urine was collected with the greatest care. Patients who seemed not intelligent or reliable enough were not used for the investigation. Since the correct amount is the crux of this investigation, I was as careful as possible in this respect.

For the temperature data, the first number refers to the temperature of the preceding evening and the second value refers to the temperature of the morning of the particular day.

The blood serum was analyzed according to the usual 2.) A weighed amount was evaporated to dryness in the procedure. water-bath (addition of acetic acid seemed to favor the drying process), it was then carbonized in a moderate incandescent heat, the coal was crushed, extracted with hot water to which a little hydrochloric acid has been addea, collected on an ashfree filter, filter and material were dried and calcined, the residue was digested with some water and hydrochloric acid; this - including the insoluble part - was combined with the first liquid [extract] - the mixture was made strongly alkaline with barrium hydroxide, and was left standing for 12 hours etc. I always had to content myself with blood obtained from cupping glass; these cannot be moistened before their application and consequently adhere poorly and yield little blood. It was thus not always possible to obtain usable serum. Only once did the blood come from a vein section (Schakowsky (Table IV).

3.) Pneumonic sputum was treated in the same manner. The entire 24-hour collection - if it was not too large - was

- 4 -

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evaporated and carbonized in the platinum dish. If the amcunt was too large, I evaporated the sputum to dryness in the porcelain dish, removed it from there, with as little loss as possible, pulverized, weighed the air-dried residue (centigrams) and used a part for analysis. I deviated from the procedure in one case only. Since in this case the amount of sputum was very large (between 200 and 250 cc), I found it advisable not to evaporate the whole amount, but to measure it and take an aliquot for analysis. However this could not be done with the genuine sputum. I tried to dissolve the latter by boiling with barium hydroxide; in practice only a small, mostly powdery residue remained. The liquid was always diluted to 400 cc, it was left to stand for some time and then 40 cc were taken for analysis. This was evaporated and calcined with the addition of a few drops of nitric acid in order to introduce some barium nitrate. The volume of the precipitate formed during the boiling with barium hydroxide could be disregarded for the same reasons as in the case of the urine analysis. The latter contained only little calcinable substance, and consisted for the main part of barium carbonate together with calcium, magnesia, traces of sodium, phosphoric acid, traces of hydrochloric acid. Potassium could not be demonstrated.

4.) During the alkali determination of saliva - originating from an angina tonsillaris with stomatitis - I proceeded as for blood serum.

5.) The procedure used for the determination of alkali salts in feces needs special justification. For my purpose I thought it unadvisable to calcine the material directly. In this manner I would also have determined the salt content of the undigested remnants of the food which are still carried in the feces. These however seem not to have to be considered. Borrowing a very apt description of Hermann, the latter seem to cling mechanically to the inner surface of the body. It seemed better to extract the feces with water and to determine the salts in this extract.

The feces were thus placed in a calibrated porcelain dish, weighed, a weighed amount (40 to 50 gms) was removed, carefully triturated with water, then enough water was added to bring up the volume to 600 cc, this was warmed, cooled and the volume was again adjusted to 600 cc. This is filtered. The filtration is rather slow; as soon as 200 cc were collected they were used for ash-analysis.

Collection of thin typhoid bowel movements was carried out as carefully as possible. They were transferred from the bed-pan to a large glass [container] by means of distilled

- 5 -

water. A 24 hour sample was collected in the glass. The sample was diluted further and treated in the previously described manner. In both cases the following small error was introduced: when determining the volume of the sample, the solid particles suspended in the fluid were not taken into account. - The errors are of the same order as those for urine. - When performed, urinary Cl is determined by titration with AgNO3 preceded by fusion with pure saltpeter.

Pathways of Alkali-Salt Elimination

If one considers only one side of metabolism: consumption and replacement - without considering elimination via excretion - it appears that healthy grown persons maintaining more or less a balance between intake and output, - do not need a constant supply of salts in their diet. One can imagine that the salts that are set free upon decomposition of the tissue of which they are part - are used immediately to build new tissue, since they do not have to undergo any change.

Only experience tells that the process is different. We have known for a long time - from Wund's experiments - that withdrawal of sodium chloride causes considerable disturbances already after a few days. As Kemmerichl) has shown recently, this is true, to a greater extent for potassium salts. Kemmerich's experimentd do not apply completely here because he used dogs that were still in a growing state in which the formaticn of tissue, especially muscle tissue, exceeds the decay of tissue and an excess of potassium is thus required which cannot be supplied by the liberated amount [of potassium salts]. However, the pressed meat residues that Kemmerich used to feed the animals still contained a quantity of potassium salts, which could not be extracted with water, but which upon dissolution by the digestive juices are set free and can be used by the organism. This amount must be at least as large as the required excess. Nevertheless the animals waste away because the organism is not as economical and excretes salts whether they are going to be replaced or not. A certain amount of flooding of the body with salts is necessary for the mainte-nance of the metabolism, and Voit has demonstrated directly that increased administration of sodium chloride results in an increase in the entire metabolism.

The reasons for the need for salts are not entirely clear. One reason could be that salts are necessary to bring about a certain concentration of the blood and the parenchymal juices in order to create and maintain the osmotic currents

1) Pfluger's Archiv, V-1 II, p 49.

- 6 -

which are necessary for metabolic processes. Secondly it is certain, though direct proof is still lacking, that the amount of acid that leaves the body is greater than the intake. The sulfur of the proteins underlying the decomposition, and which are, in the last analysis, introduced with the food, leaves the body as sulfuric acid, while there is no noteworthy reformation of organic sulfur compounds from the introduced sulfuric acid. A part of the ingested alkali is thus used to bind sulfuric acid through the formation of acid phosphates and acid urates.

A question, which has to be settled before the amount of alkali salts that leave the body can be determined is: what secretions eliminate a considerable amount of alkali salts from the body?

For healthy, fever-free persons urine is the only secretion that has to be taken into consideration to determine the elimination. The other external secretions occur in too small amounts to matter. The only other secretion produced in considerable quantity, the feces, contain normally very few salte extractable with water. This is apparent from Table 1.

Detuc	Marnmenge E	Hornalo	Notice	NALFOR	Summe D	pci (P
25.	1490 Ccm.O F. 81,5 Grm	29,05	3,412 0,255	5,692 0,068	9,134 0,323	37,7 79,4
	frat	-	3,697	5,76	11,157	35,0
26. 1010 Cc BF. 121,6 C	1940 Ccm. DF. 121,6 Grov.	28,13	2,929 0,316	4,385 0,092	7.314	40,1 77,5
	breng	•	3,213	4,477	7,122	42,1
27.	1735 Ccm F. 126,0 Grui.	28,96	$2,282 \\ 0,190$	4,633 0,073	6,913 0,190	33,0 71,8
	breitg		2,472	4,706	7,178	34,4
28.	1630 Ccm. F. 140,7 Gms.	24,61	2,298 0,287	4,287 0,150	0,585 0.438	34,9 65,62
	duastraile		2,585	1,437	7,023	36,5
29.	1340 Ceia. F. Genicht?	22,91	2,620 0,314	4,208 0,226	0,834 0.540	38,0 62,5
		. –	2,94	4.434	7,374	39,9

Table 1. Observations made on myself

1. Ccm = cc

2. Grm = g. 3. F

3. F = Feces

a. Date; b. Amount of urine or feces; c. Urine; d. Potassium oxide; e. Sodium oxide; f. Total; g. %K

fest = solid; breiig = pasty; dunnbreiig = thin; Gewicht = weight Remarks: 1) I have set the sum total of potassium and sodium oxides equal to 100 and have calculated the percent potassium oxide. This figure is given in the last column. The diet was, on purpose completely unrestricted.

- 7 -

One can see that, compared to urine, the alkali salt content of the feces is very small. At the same time one can see, from the data, the known fact that in feces potassium salts predominate. - The figure of the last day for the feces cannot be considered normal. It should be used as an indication that the distribution of salts is different during diarrhea. To induce this condition I took, on the morning of the 29th 15 gm. infusion of Senna - in two portions. The latter however did not cause large, thin bowel movements, but a large-intestinaland colon catarrh whose symptoms were violent cramps and rather limited bowel movements. I did not repeat the experiment since Schmidt already established that sodium salts predominate in diarrheal feces. The following total amounts were eliminated in the course of the five days:

a) b)	via via	the the	urine feces	KO 13.577 _1.362	NaO 23.205 0.609	KO+NaO 36.782 1.971	36.9%
			Total	14.939	23.814	38.753	38.5%

One can see that proportionally the value for potassium oxide does not change much when the fecal excretion is taken into account; I thus felt I did not have to take it into account. It would however be wrong not to take diarrheal feces into account whose large salt content has been demonstrated by Schmidtl).

For invalids with large secretions the latter will have to be taken into account and their omission may introduce a large error.

1) I was able - on myself - to observe the salt excretion in the saliva during angina tonsillaris associated with stomatitis and considerable salivation.

The saliva collected during 24 hours measured 515 cc. It was thin, slightly turbid, alkaline and some mucus collected on the bottom of the glass. It contained a considerable amount of buccal epithelia and salivary solid bodies. It behaved normally towards reagents; there is no precipitate with nitric acid; slight yellow coloration upon bciling; strong saccharification power. 50 cc were taken for analysis: Found

0.138 KO
0.023 NaO
0.161 KO + NaO.
1) Schmidt, Characteristik der epid. Cholera (Characteristics
of Epidemic Cholera), p. 90.

- 8 -

Thus the total elimination via saliva was 0.697 Ko and 0.116 NaO. Potassium represents 85.7% of the total. The salivary glands thus seem to be elimination organs for potassium. This is also confirmed by the observation of Kemmerich who found that potassium intoxication causes extensive salivation.

Urine of	the same	e day:					
665 cc uri	ne 19.15	gm (ne	appetit	te, [.]	very	little	fever)
Found:	0.205%	КO Г	a 1.363	gm	-		
	0.427%	NaO	= 2.840	ğm			
	0.632%		4.203	gm			

Potassium makes up 32.4% of the total amount. A total of 2.06 gm potassium oxide and 2.956 gm sodium oxide = 5.016 gm total, were excreted during that day.

The potassium makes up 41.1% of the total. If in this instance the saliva had been disregarded one would have committed a considerable error.

2) A considerable amount of alkali salts can at times be eliminated via the sputum. This is illustrated by the following table. This is the case of a gangrenous lung condition, with purulent expectoration, which is in the healing process. Subfebrile state, good appetite.

Table II. Lueneberger.

(/) a) Unin.	
27 700 (20 1212 DCT 2007 92	
	.1
23, 1500 - 1,380 3,255 4,635 29	.8
24. 1100 - 1,167 2,070 4,587 35	3
b) Sputum.	
22. 1,42 2,35 3,77 37	.5
23 1,16 1,78 2,94 39	.5
24. 1,20 2,46 3,68 32	9
c) Sputum +- Urin,	
22. 2,743 5,024 7,767 35	.3
23. 2,54 5,035 7,575 33	.7
24. 2,817 5,455 8,267 34	,0

1. Date; 2. Potassium oxide; 3. Na oxide; 4. K + Na oxide; 5. %; 6. CCm = cc.

There is a certain antagonism between the alkali elimination in the sputum and in the urine. On the 23rd, the potassium content is lower than one would expect for this particular diet; however, there is a corresponding increase in the sputum.

Such cases are to be avoided if one does not want to

- 9 -

complicate the investigations unnecessarily, since in such cases determination of the alkali salts in urine alone would give an entirely wrong picture. I believe however that in the case of pneumonia one can neglect the sputum without incurring too large an error and that one can draw conclusions from the urinary elimination alone. In order to prove my point I present the data of alkali determination in sputum in two cases with pneumonia. I shall refer to these cases later on.

Table	III.	Mertin	s, 16	years (old.	Croupous	<u>pneumonia</u>
	() D3L	Temper.	Harnfaenge		Natro	Kali + Netroa	(2) pči.
	15. Febr.	40,7-40,0	1350	1,985 -	0,163	2,148	92,4
	16	39,5-39,6	1470	2,142	0,431	2,573	82,8
	17	30,7-36,7	1590	1,399	0,271	1,670	83,8
	18	36,8-36,7	665 ')	0,002 pCi	. 0,325 p(L 0,417 pCL	22,1
	19	37,0-37,1	1055	1,530	5,488	7,016	21,8
	20	\$7,1-36,8	1970	3,369	9,022	12,391	27.2
	') alc	àι≜iles. ≃Ω	ot al	1	-		
				b) Stutam	·		
		Dat	Kalı	Natroa	Kali II. Norea		
		15.	0.061	0.188	0940	•u put. ••••	
		16.	0.071	0 275	0.340	90.5	
		17.	0.017	0.134	0,510	20,0	
		18.	0.042	0 1 3 4	0,101	23,9	
		. 19.	0.032	0 699	0,172 A 431	24,3	
		20.	0.025	0.067	0,131	23,4	
			,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,) Πειο -L Santi	0,092	<i>¥1</i> ,1	
		- 15.	2 046	0 341	nu. 0.207	0- 0	
		10.	2 213	0,331	2,397	85,3	
		17	1 4 4 6	0,700	2,919	75,8	
		18	1,440	0,100	1,851	78,1	
		10.					
		19.	1,5/2	5,385	7,157	21,9	
		2 0.	3,394	9,089	12,383	27,3	

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1. Date; 2. Temp. 3. Amount urine; 4. K oxide; 5. Na oxide; 6. K + Na oxide; 7. %.

Remarks

1) Large build, fairly heavy constitution.

- 2) Onset of illness: Noon of llth with chills, shooting pain in the night of the 16-17.
- 3) No appetite, constipation. 4) Sputum: typical of pneumo
- 4) Sputum: typical of pneumonia, amount: rather more copious than usual.

According to the above the alkali salt content of the sputum is usually even smaller than that of normal feces.

- 10 -

Table	IV.	Schakowski,	worker	20	years	old.
		Croupous p	neumonia	1		

O	E)	\odot		ia. C	5) a.) (1)	(E)
Dat.	Tomper.	Hørnmenge	Sp. G.	🕑 Kali	Natron	Summe	րԸւ
24. Doc.	40,0-39,9	620	1026	3,01	6 0,354	3,395	92,3
23	40,5-39,8	760	1026	2,30	1 0,311	2,612	88,1
26. •	30,1-39,8	730	1020	1,70	0,226	1,927	88,2
27. •	40,6-39,7	1270	1024	2,629	0,521	3,150	82,5
28	39,9-39,2	\$150	1024	2,43	5 0,518	2,950	82,5
29	40,0 —3 0,0	1030	1024	1,90	5 0,36	2,266	84,1
30. •	36.0 - 36,2	880	1020	0,86	2 0,625	1,487	58,0
\$1	Debacírai 🛣	1200	1024	1,03	3,984	5,016	20,6
1. Jan.	dito	1570	1022	2,90	5 7,540	10,645	27,3
			b) Spu	lum.			
	Dal	K	sli l	iatron	8umma	pCt.	
	26.	. 0,0	021	0,087	0,1.5	16,3	
	27.+28. pi	ro die 0,0	028	0,170 .	0,198	13,3	
	29.+30.	- 0,0	028	0,112 -	0,140	20,0	
	31.	0,0	20	0,088	0,114	23.1	

* no fever

1. Date; 2. Temp.; 3. Amount urine; 4. Spec. Grav.; 5. K oxide; 6. Na oxide; 7. Total; 8. %; 9. per day.

I feel that I do not have to add the figures, since in this case the amount of alkali salts of the sputum is even more insignificant - considered absolutely or relatively.

Remarks

1) Fairly strong constitution.

- 2) Onset of illness: On the 19th with chills.
- 3) Sputum: typical of pneumonia.
 4) No appetite. Fever-type diet
- No appetite. Fever-type diet. Regular bowel movements, no diarrhea.
- 5) On the 25th, venous section of 10 ounces.

Bamberger¹) has already made an extensive ash-analysis of the sputum of pneumonia during the inflammatory and healing stages. Sodium and potassium behave the same way as in urine of which we shall speak later: in the feverish period potassium exceeds sodium by far, in the non-feverish period the opposite takes place. I had expected the same; however, as one can see these expectations were not borne out. Bamberger himself reports a second case in which the above-mentioned behavior does not take place. He tries to explain his results by the nature of the sputum, which was not too similar to pneumonic sputum. The sputa which I tested had a definite

1) Bamberger, Wurzburger med. Zeitschrift, Vol II, P. 344.

- 11 -

pneumonic character and consequently the ratio seems inconstant.

On occasion other pathological secretions can also remove considerable amounts of alkali salts. I felt that for my purpose, I had to reject cases in which there were considerable other secretions. Furthermore, I also had to reject all cases which had received medication which contained alkall prior to admission to the clinic, or cases which had or developed diarrhea during or as a consequence of medication. All these circumstances limited the number of cases - with fever - which I could use for my investigation.

THE ELIMINATION OF ALKALI SALTS IN HEALTHY, FEVER-FREE INDIVI-DUALS.

It is self-evident that the amount and ratio of potassium and sodium of the eliminated salts depends on the type and amount of the food taken in.

Table V. Observations on Myself

Ŵ	Dal, 🖽	aramea ga	Harasto	🖉 Kali 🖉	Natros	Summ	DIJa
2. Dec	1869.	1230	25,3	3,038	4,588	7.620	39.8
3.	-	1620	27,2	3,191	4,744	7,938	40.2
4.	•	1615	24,8	2,859	3,925	7,784	34.4
5.	-	1665	23,4	3,130	4,428	7,558	41.4
6.	•	1330	25,8	3,501	3,701	7,292	46.7
7.	•	1605	25.7	2,84	3,82	6,660	42,8

1. Date; 2. Amount urine; 3. Urea; 4. K oxide; 5. Na oxide; 6. Total; 7. %.

The diet was mixed, perhaps heavy on meat. The beverage was only water and 300 cc beer/day. As is apparent from the urea data I approximately maintained my nitrogen balance.

Body weight at time of experiment about 45 kg.

- 12 -

There was obtained daily, on an average:

1513 cc urine with 25.69 gm urea, $3.094 \text{ K}_{20} + 4.207 \text{ Na}_{20} = 7.476. - 41.4\%$ Per day and 100 kg: 57 J cm urea 6 87 cm K-0 0.35 cm Naco

57.1 gm urea, 6.87 gm. K_20 , 9.35 gm Na₂0.

Fable	VI.	W.,	25 years	old,	syphilis,	treatment
				by	inunction	

Do Dal	Haramenga	Harnatoff	Kali (Natron 🐨	Summa	pCi. 💮
[₩] 24.	1470	19,8 (2)	1,882	6,291	8,173	23,1
25.	1780	21,9	1,638	7,038	8,675	18,5
26.	1610	18,51	1,823	5,116	6,939	24,8
27.	1630	18,74	1,907	5,770	7,677	24,8
28.	1620	18,46	1,280	5,443	6,723	19,01
29.	1500	17,85	1,395	4,205	6,60	21,1
30.	1690	21,97	2,062	6,945	9,008	23,0
31.	1770	21,24	2,362	6,717	9,079	26,1

1. Date; 2. Amount urine; 3. Urea; 4. K oxide; 5. Na oxide; 6. Total; 7. %.

Remarks:

 \overline{A} diet poor in protein, no meat. No saliva discharge. Physical well-being.

If one compares this table with the previous one, one notes at once the lowering of the potassium as a consequence of the meat-free diet.

Table VII. Sophie Schroeder, 27 years old, small.

(Doi.	Harnmenge	Harnatof	S Kali (4)	Natron 🔗	Summa (5	pCi. Cz.
19.	1390	27,5	3,211	8,188	11,400	28,2
20.	1690	27,0	4,225	7,216	11,441	30,3
21.	1620	23.0	2,819	7,093	9,914	28,4
	(5= Mille	25,8	3,452	7,499	10,92	28,9
	A Mille	1 25,8	3,452	7,499	10,92	2

1. Date; 2. Amount urine; 3. Urea; 4. K oxide; 5. Na Oxide; 6. Total; 7. \$; 8. average.

The patient suffered, to a minor degree, of progressive muscle atrophy. Her metabolism can be considered normal. General state good.

Diet: plentiful, also milk but no meat already several days before observation. Body weight 88 pounds 25 Lth [not identified] on the 25th.

- 13

Table VIII. Schroeder. The same diet with meat.

Date	Harnmeng . 1	larnatoff	Kali	Natron	Summa	pCt. (~~)
22.	1530	26,4 (3)	4,228	7,3:4	11,552	36,5
23.	1250(7)	20,6 (?)	. 3,100	5,513	8,613	36,0
24. 1	1860	30,2	3,701	G,804	10,565	35,0
25.	1690	27,3	4,191	7,977	12,108	34,4
16.	1660	28,9	4,532	8,731	13,263	34,1
27.	1300	25,2	3,172	5,603	8,775	36,2
	(Nittel	26,4(?)	3,636	7,002	10,862	35,4

1. Date; 2. Amount urine; 3. Urea; 4. K oxide; 5. Na oxide; 6. Total; 7. \$; 8. average.

In this case also one can see the effect of meat on the excretion of potassium if the two periods are compared.

I had a very hard time to find a person who was on a fever type diet for some time but did not have too considerable disturbances.

Table IX presents the case of a 16 year old girl who suffered from a peculiar type of deep melancholia, without any other apparent disturbances. She only took liquid, or semisolid nourishment and had to be fed. Later on she refused food completely and had to be fed by stomach tube until she left the clinic (unimproved). This stage lasted a whole month. The sensorium was free. She never wetted herself, but gave indication of her need to pass urine through sighing.

Table IX.Rautenberg, 16 years old.Partial Inanition.

Daw	Haramuaga	Harast. pro dia	Kalire I	Nairon pro dia	Summa C.	pCi Zi
i. g. 2.	800	8,0 3	0,80	1,10 5	- 1,96	40.8
3. u. 4.	1030	8,73	0,829	1,730	2,559	32.2
5. u. 6.	900	. 9,045	0,803	1,735	2,56	31,4
7. u. ä.	620	7,45	0,625	0,862	1,487	41.6
						•

1. Date; 2. Amount urine; 3. urea/day; 4. K oxide; 5. Na oxide/day; 6. Total; 7. %.

Both the absolute amount of alkali salts and their ratio vary considerably for no known cause.

THE RATIO OF THE ELIMINATION OF ALKALI SALTS IN THE CASE OF FEVER.

At first I present the tables which concern acute illnesses accompanied by fever: pneumonia, recurrent fever,

- 14 -

erysipelas. General conclusions will be drawn from these cases.

Table III. Martins. Croupous pneumonia, already presented above (p. 10). I complete the data here by adding the chlorine determinations, in which the chlorine is reported as NaCl. (For greater accuracy the urine is always burned first with saltpeter).

Datum.	J	G	j		3	
15.	0,188	PCL	-	1,838	Gra.	NaCI,
16.	0,231	-	-	3,690	•	•
17.	0,176	-	-	2,898	•	-
18.	0,355	•				
19.	0,823	•		8,683	•	•
20.	0,978	•	-	19,267	•	-
•						

1. Date; 2. pCt = %; 3. Grm = g.

Table X. Grohnert, age 45 years. Croupous pneumonia

Ø	Dal.	🕢 Temp.	Hornmengo	Harnst .	Keli	Natron	Summa	րմեւ 🕞
	16. Noi 1	870. Fieber	CU 850	20,23	2,32	0,314	2,635	88
	17.	.0	1100	32,0	2,288	1,067	3,355	68,2
	18.	fieberfrei	880	29,22	1,038	0,096	1,734	59,9
	19.	. 1	1030	31,93	0,628	2,493	3,121	20,1
	20.+21.	- prò	die 900	25,82	0,547	2,11	2,657	12,5
	22.+23.	•	- 890	23,59	0,917	2,884	3,791	24,1
	24.+25	•	- 1360	27,80	:,513	0,037	7,550	20.0
	26.	•	1930	23,51	1,737	7,392	9,129	19,2
				-				

1. Date; 2. Temperature; 3. Amount urine; 4. Urea; 5. K oxide; 6. Na oxide; 7. Total; 8. %; 9. fever; 10. free of fever; 11. per day.

Remarks:

The medical history has mysteriously been lost. It was a regular c oupous pneumonia which started with chills. Temperature varied between 39 and 40° C. Crises at the end of the 5th day in the night of the 16-17th. On the 26th the patient had to be dismissed at his request. Little expectoration. No diarrhea.

- 15 -

Table X	I. Fey	erab	end, :	20 y <i>e</i>	ear o	ld gi	[r].
	<u> </u>	ecuri	rent :	Cever	·		
G: Det	6 Techner	Harnmene	 Ustasiai	r Katal	Salron	Comme	El . c. E.
13 Jan 183	0 10 2 - 30 7	010	AD G 1030	3 9 817	 	2 0 10	or percer
14	40.0-31.7	1550	1286	1.611	0,12	1 9/1	V1,2
14	16 1 - 35 4	860	34.05	0746	0,310	1,012	70.1
16	36 B - 36 9	530	263:	0,710	0.100	1.000	19,1
10	37.0 36.8	180	40,0J	0,455	0,307	1,004	40,0
17.	37,0 - 30,0	. 400	40,05	0,144	1,115	1,7.17	24,3
10.	30,0-30,5	010	21,03	0,432	3,080	4,512	18,4
19.	. 30,3-30,3	840	18,00	1,243	3,041	4,281	29,0
20.	30,8-30,8	830	14,33	3,134	3,303	4,457	23,9
21.	37,0-39,6	900	13,34	1,520	5,943	7,469	20,41
22.	41,0-40,2	Jolder v	or der Un	lerouchun	g forigego	.05820.	
23.	-40,2 - 40,4	CP 660	14,31	1.755	1,407	3,181	55,8
24.	; 36,8-36,0	1160	23,17	1,694	0,101	1,858	88,2 .
25.	30,6-38,4	420	16,17	0,777	- 0,168	0,945	82,2
28.	37,0-38,0	· 420 ·	15,5	0,773	0,260	1,033	74,7
27.	- 37,0-36,6	. 320	13,03	.0,630	0,234	0,804	73,3
28.	fleberfrei	480	17,28	0.883	2,309	3,192	27.7
29. (<u> </u>	. 690	16,32	1,070	5,083	6,135	17,6
30. Jan. 1870	fieberfrei	1060	19,14	1.462	7.389	8 8 1	171
31.	•	700	12,21	0.032	5.334	0.988	15.9
1. Febr.	•	1240	16,1	1.19	5.02	7 110	16 7
2.	•	1140	15,98	1.528	3.978	5.506	27.7
3.	•	1130	19.43	2.442	6.193	8 6 3 5	28.0
4.	· •	1190	19,23	2,321	4,974	7,295	31,8

1. Date; 2. Temperature; 3. Amount urine; 4. Urea; 5. K oxide; 6. Na oxide; 7. Total; 8. %; 9. Unfortunately discarded before analysis; 10. free of fever.

Remarks:

1) Onset of illness: Morning of Jan. 9, at 11 A.M. with chills. Admission: Afternoon of 12th. First crisis: Night of 13-14th. Relapse morning of 21st. Crisis at noon of the 23rd with abundant perspiration; since then free of fever except for a single rise in temperature to 38.4° C on the morning of the 25th. This passed quickly.

2) Strong constitution. Very slight appetite. During the first day thin and sparce bowel movements.

3) The figures of the first days represent the average of two determinations. The urine of the 22nd, which was particularly interesting, was unfortunately thrown away by the nurse before analysis.

4) No sputum.

- 16 -

Table	XII.	Friedr.	Kamrau,	16	1/2	years	old.
		Ervsi	las fact	le1	_	-	

ſ

Int. C	Temper-II	aromenge	Harnstoffe	Kaly	Natron-	Sunima-	pCi. c.
26. Jen. 1870.	39,3-40	870-3	37,84	3,725	2,453 ^{~~}	6,177	60,3
27.	39,1-40,2	960	42,34	3,234	0,980	4,234	70.8
28.	39,039,4	1230	48,9	4,317	1,895	0,212	69,5
29.	: 39,5-39,0	aicht un	teraucht.				
30.	30,0-30,60	£1270 .	52,58	2,946	0,420	3,300	87,5
31.	39,5-39,4	923	. 37,18	1,505	0,361	1,860	80,7
l. Febr.	37,8-39,8	070	37,83	1,750	1,170	2,532	69,3
2.	37,6-37,3	660	27,06	1,505	0,699	2,204	68,3
3.	38,6-35,0	930	35,71	2,827	2,558	5,385	52.5
↓. `	36,4-36,8	1010	35,65	3,192	3,242	0,434	49,6
5.	fleberfrei	1100	33,0	2,541	4,660	7,141	32,7
û. I	D .	1020	20,78	3,108	4,766	7,874	34,8
7.	•	1220	31,72	2,513	6,503	9,016	28,6
8.	•	1090	26,19	1,284	7,172	9,136	21,7
			·				

1. Date; 2. Temperature; 3. Amount urine; 4. Urea; 5. K oxide; 6. N oxide; 7. Total; 8. %; 9. not analyzed; 10. free of fever.

Remarks:

14.

15.

16.

Upon his urgent request the patient had to be released on the afternoon of the 8th. The observations thus could not be continued.

Onset of illness: Noon of the 24th. Admission: Noon of 25th. Crisis occurred on Feb. 1st. The temperature recorded in the evening of the 31st (37.8° C) seems questionable. Very strong constitution - Fever-type diet until the 3rd, meat from the 4th on. Patient's appetite was very poor on days when he had fever.

	Cro	upous	pnei	moni	a		
Del. O	Tempile II	aramenge	Harnstoff	Kali -	Natron -	Sumaia	
5. April 1870	40,6 - 30,1	8103	25,114	2,351	1,232	3,013	63.9
Ú.	. 39,2-37,5	770	26,56	1,34	1,186	2,526	\$3.1
7.	37,0 - 37,0	800	29,2 -	0,72	1,72	2,14	29.5
8. 、	37,0-37,0	500	\$5,85	0,12	1,115	1,535	27.7
9. ·	37,4-37,2	850 -	29,41	0,646	6,31	4.956	13.6
10.	37,5-37,1	1010	25,96	0,919	6,141	7.06	14.3
11.	37,3-37,0	1180	21,54	1,263	5,439	6,792	18.9
12.	alebertrai	890	22,52	1,29	4.20	5.42	23.5
13.	œ,	900	13,68	0,765	2,358	3,213	23.8
14	÷.	880	14,56	1,065	3,64	4,708	22.7

Table XIII. Bahr, Male, 33 years old.

1. Date; 2. Temperature; 3. Amount urine; 4. Urea; 5. K oxide; 6. Na oxide; 7. Total; 8. \$; 9. free of fever.

16,25 0,971

4,708 22,7

2,530 3,801 23,5

17,70 1,308 4,308 5,730 23,8

830

1200

- 17 -

Remarks:

Onset of illness: March 31st; Admission: Noon of April 4th. Crisis (with perspiration) during the night of 5-6th, free of fever since. Patient feels well but lacks appetite. Feces from 4-5 at noon: 3 thin movements, solid since that time. Little sputum. Fairly weak constitution.

Table	XIV.	Klock,	Worker,	35	years	old.
		Croupous	s pneumor	nia		

Dol. D	Temper. 2	Hernincage	· Kaly#)	Natrons	Summer	pCify
7. April 1870.	39,1-38,6	040	1,818	1,120	2,938	61,9
8.	39,3 - 37,3	450	0,932	1,251	2,183	42,7
9.	39,4-37,8	600	0,726	1,770	2,502	29,0
10.	38,038,1}	940	0,401	2,305	2,706	14,8
11.	fieberfrei 1				· · · ·	40.4
17.	•	800	0,603	3,750	3,355	18,1

Date; 2. Temperature; 3. Amount of urine; 4. K oxide;
 Na oxide; 6. Total; 7. %; δ. free of fever.

Remarks:

Light case with little fever. Onset of illness: Morning of 4th April, admission: noon of the 6th. The crisis occurred already during the night of the 7-8th, then again slight fever at irregular intervals. - Very little sputum.

Fever-type diet. Almost no appetite, no diarrhea. Average constitution. The figures for the urine from the 10th to the 11th are unreliable as are apparently all other data.

Table XV. May, 48 years old. Croupous pneumonia.

Was admitted at noon of the 4th, which was the 5th day of the course of the illness Died on the 5th in the evening. Only one determination available.

510 cc, 20.2 gm urea, 1.560 K oxide, 0.195 Na oxide, thus, the K makes up 88.9% of the total.

Table XVI. Wilhelm Lucas, 16 years old. Croupous Pneumonia

Dat. C 21. Dec. 1870. 22. 23.	Temper 40.1-40.0 30.2-30.2 37.9-36.0	laramen 1000- C: nicht	te op G. – 1029 Vaterouch	(+ Kali - 3,44	Natron 0,77 \	Sucima 4,21	€ pCi.€: 81,7
24. Dec. 3870. 23. 24. 27.	36,436,4 36,636,8 fieberfrei	505 560 970 880	1028 1029 1026 1019	0,520 0,723 2,260 1,528	2,034 4,334 6,431 3,206	3,454 5,002 8,691 4,734	15.1 14,4 26,0 34,4

1. Date; 2. Temperature; 3. Amount urine; 4. Spec. Grav.; 5. K oxide; 6. N oxide; 7. Total; 8. %; 9. not analyzed; 10. free of fever

Chlorine Determination Cl as NaCl

21. C,145 pCf 1,45 Grave 22. incht bestimmt. 23. incht bestimmt. 24. 1,011 pCt. 5,106 Grav. 25. 1,502 - 8,747 -26. 1,606 - 15,38 -27. 1,051 - 0,24 -

1. $pCt_{,=\%}$; 2. Grm = g; 3. not determined.

Remarks:

Onset of illness on Dec. 15th with shivering fits, crisis on the 22nd. Sputum sparce, no diarrhea, poor appetite.

Table IV. Schakowski, worker, 21 years old. Pneumonia. Mostly already reported above. Only the chlorine determinations are reported here.

	C1	as	NaCl	•
24.	0,175	pCr. 🛥	1,085	Grm.
25.	0,165	- =====	1,221	. '
26.	0,170	• 25	1,211	• .
27.	0,120	- 657	1,524	•
28.	0,139		1,599	•
29.	0,132	• . •••	1,568	•
3û.	0,161		1,417	•
31.	0,638		7,87	•
1.	1,024		16,08	•
	•	•	•• •	

pct = % ; Grm = g

- 19 -

If one surveys the course of the potassium and sodium elimination separately one can see, from the tables, at first a drop in the amount of potassium after the crisis until a minimum is reached, and then a gradual increase (with minor fluctuations) during the convalescence. If the observations are continued long enough a higher value is attained than during the feverish period, which is due to the increased food intake. At the extreme the amount of potassium on a day with fever can amount to seven times as much as during a fever-free day. Cf. table XI 2.857 gm on the 13th, and 0.422 gm on the 17th. Such differences, however, are rare; three to four times the amount is more usual. In many cases the amount of potassium during the convalescence period falls below the amount eliminated by a relatively healthy person on a fever type diet (see table IV); the latter amounts to about 0.8 gm.

The scdium behaves differently throughout. With high fever it drops, in urine, to a minimum value, e.g. in Table XI it amounts to 0.12 gm on the 13th. It is considerably reduced in each case amounting to 1 gm. ppl, thus less than in a healthy person on the same diet. - If the observations are started soon after the beginning of the illness, the amount drops during the first days; this is apparently still the effect of the previous dietary intake. If the observations start later the sodium value is immediately low. The amount increases soon after the crises. The increase is often very sharp so that the amount determined on the first day on which an increase is noted sometimes exceeds the total amount determined on all previous days with fever. For instance in table III (Martins) the total sodium oxide content of the 15, 16 and 17th is 0.865 gm, while that of the 18th is 2.161 gm, even though this is only about half the urine, and that of the 19th is 5.486 gm.

In Table XI: 1.135 NaO on the 13, 14 and 15th as compared with 2.896 on the 17th.

In Table IV. (Schakowski) 3.115 gm in 7 days (24-30) as compared with 3.984 gm of the 31st and 7.54 gm on the 1st.

In general the lowest value for potassium occurs when sodium is already on the increase, the percentage is then very low for the former; often, however, when the sodium content is on the increase the potassium is also on the increase. Only very seldem - apparently after very prolonged fever - does the opposite occur: the potassium is still falling while the sodium is already increasing again. The potassium ratio is then extremely low.

These irregularities - like the often considerable

- 20 -

differences in the number of days between the individual fever or fever-free days - are not related to the nature of the problem but are contributed 1) by the arbitrary choice of the 24 hour time interval, 2) by the irregularities of food intake which can never be completely avoided. The irregularity in the individual days are much smaller during the experiments on myself. In these experiments the periods were kept as regular as possible. A general rule is that each individual with fever excretes more potassium than sodium, and that convalescing persons excrete more sodium than potassium, and that furthermore the constitution of the urine exceeds this change-over by one to two 24 hour periods. Case XI retained "fevertype" urine for a remarkably long time, i.e. for four 24 hour periods (during relapse), perhaps in connection with the low diuresis. The transition from one type of urine to the other is sometimes gradual and sometimes abrupt so that the urine of two consecutive days has a completely different character.

If we want to know the reason for this behavior it is again advisable to consider potassium and sodium separately.

To me, the most natural interpretation for the behavior of potassium is that its concentration decreases during a fever-free state as a consequence of the decreased metabolism compared with the feverish state. The increased food absorption can then explain the gradual recovery. We thus assume that the excreted potassium is in fact the potassium which is liberated each day from broken-down tissue (+ that introduced by food). - As far as I can see, no great objections can be made to this interpretation; but even if we assume that the decrease in the potassium excretion during the fever-free period is caused, in part, by the retention of potassium which was excreted during the fever period, and had not been replaced by food, there is still a considerable increase in potassium excretion compared to a healthy individual on the same diet. These findings indicate that the metabolism of tissue rich in potassium is increased during fever. The subsequent increase of potassium is so gradual, that its interpretation as being caused by increased food intake seems to be reascable.

Can the above assumption of stepwise climination associated with liberation from tissue breakdown also be used for sodium? Can we assume, in practice, that during the lively fever metabolism only 1.2-3 decigrams of sodium oxide are liberated during 24 hours, as shown in the tables, or should we assume that the eliminated amount does not correspond to the liberated amount and fever causes retention of sodium salts? Furthermore, should not the food taken in - even at

- 21 -

reduced appetite - contain at least a few decigrams of sodium? The sudden increase of sodium after the crisis, in a few cases, points to the fact that we deal here with a retention during fever. No other explanation could be possible. I have already summarized the striking cases before - strangely enough they all concern young people (Tables III, IV, XI.) This hypothesis is less mandatory in other cases, in these the sodium excretion during fever is larger and the subsequent in-crease can be explained satisfactorily through the increased food intake. c. Tables XII, XII, XIV. I consider it likely that in this instance there may be individual differences. In the cases of the first type it is impossible to evaluate the amount of sodium liberated during fever and the relative potassium value is also worthless. - But even in cases of the second type, where retention is unlikely - or at least there is no compelling reason to assume it - we see nevertheless that the figures for sodium do not exceed those of fever-free persons on a similar diet while the increase of potassium [elimination] is unquestionable. We can thus conclude that the increased metabolism during fever occurs mainly in tissue rich in potassium - a conclusion which however is put in doubt because it is based on such a small number of observations.

A definite answer could probably be obtained if the daily intake of salts in food were determined. I would have liked to proceed in this manner; however, the difficulty to execute such a project on patients proved too great, especially for a single observer. Finding out whether administered sodium salts reappear during fever would also contribute to the elucidation of this question.

The behavior of chlorine in the cited cases corresponds exactly to that of sodium, so that one would have to consider sodium chloride retention, and the current view that its decrease in fever-urin(is due to inanition would be false. Chlorine also manifests a considerable and sudden increase after crisis, when a considerable food-absorption cannot as yet be taken into consideration; cf. case III and IV. I have attempted to approach the question of sodium retention during fever from another point of view. The assumption would be strengthened if it were possible to prove a relative decrease of potassium in exudates and in blood serum.

1

I analyzed the blocd serum of two of the cases: Mertins (Table III) and Schakowski (Table IV) for alkali salts. I also analyzed a typhoid patient (Glogau).

- 22 -

Schakowski, Venous section on the 25th <u>)</u>) 24.474 gm serum yielded 0.220 alkali chlorides and 0.049 potassium platinum chloride = 0.386 KO per thousand 4.437 Na0 4.823 Potassium oxide makes up 8.0% of total. Mertins. Blood from cupping glass on the 16th 2) 13.199 gm serum yielded 0.115 alkali chlorides and 0.028 K platinum chloride= 0.409 KO per thousand 4.729 NaO 5.138 Potassium oxide makes up 8.7% of total. 3) Glogau. Blood from cupping glass. 23.577 gm serum yielded

0.153 alkali chlorides and 0.040 K potassium chloride= 0.323 K0 3.165 Na0) per thousand 3.488 Potassium oxide makes up 9.3% of total.

As far as I know, there exist, in the literature, only two determinations of alkali salts in blood serum of healthy individuals. They were made by Schmidt. If one computes his figures (1 oc. cit. p. 30 and 32) equally for potassium oxide and sodium oxide, one obtains in the first case:

> 0.0095 K oxide 0.1160 Na oxide 0.1255 potassium oxide makes up 7.6% of the total.

In the second case:

0.0067 K oxide 0.0661 Na oxide 0.0723 Potassium oxide makes up 8.6%.

The first of my values lies between the two values found by Schmidt, the two others are very slightly above his. The drop in potassium cannot be seen.

It would be more accurate to compare the fever blood serum with that of the same person immediately after the crisis; however, the withdrawal of so much blood, as would be necessary to obtain sufficient serum [for analysis] would be

- 23 -

unadvisable for a patient who had just passed through a serious, feverish disease. I never had the heart in these two cases, both of which were very serious.

Two determinations which I carried out on healthy individuals gave somewhat higher values.

> 1) Amount of blood serum was not determined. 0.068 alkali chlorides; 0.0225 potassium-platinum chloride

0.0043 K oxide 0.0271 Na oxide 0.0314 13% of the total is K c.ide.

2) 18.433 gm serum 0.144 alkali chlorides; 0.042 potassium-platinum chloride

0.0081 K oxide 0.0695 Na oxide 0.775 [sic] 10.4% is K oxide.

The number of determinations is probably not large enough to draw definite conclusions. However I refrained from further experiments for the moment so as to bring this investigation to a temporary conclusion.

Special reference however should be made to the behavior of alkali salts during typhoid. Before - as will be seen later - I discovered that the feces are a great source of eli-mination, I found upon the examination of urine alone a marked deviation of the results from the cited figures showing a preponderance of potassium in the urine of patients with fever. These figures are not reported here because they are irrelevant in this connection. It is understandable that initially I did not believe that the feces could represent an important elimination pathway for potassium because Schmidt had proven that diarrheal feces have a higher sodium content. - Besides I feel that typhoid patients are unsuitable for such investigation for many reasons. First of all a comparison with persons in a fever-free state partaking the same diet is lacking since most typhoid patients develop a great appetite even when nightly exacerbations still occur. Comparison of figures in the feverish and fever-free state are also unreliable because the long-lasting fever causes considerable consumption and as far as metabolism is concerned - a typhoid patient is a different individual at the beginning and at the end of the illness. Concern for nourishment often forces the replacement of the fever-type diet by a richer one. To this one has to add clouding of the senses which often causes unavoidable loss

1

- 24 -

of excrements. I think that it is sufficient here to present figures for a couple of individuals - especially well suited to be investigated - which indicate the relative increase of potassium during fever. I present first two cases of ileotyphus and one case of exanthematous typhus; diarrhea was absent in all cases.

Table	XVII.	Rosa	Kodien,	21	years	old.
	Exa	nthema	atous tvi	<u>unus</u>		
Bal C	Temper	Harameaga	ep. G. Kali	S, Nate	an Summa	Ź pCi.C

		Dimesen si	a grosser	Beaomm	enheit auf	egeben v	erden.
	40,0-40,2	Jirin alles	in's Bett.	Die wei	itera Untera	uchung .	massle
	40,7-39,1	730	1021	1,117	0,307	1,424	75,5
	40,239,6	1270	1022	2,42	0,326	2,568	87,3
Juai 1870.	40,0-10	740	1025	2,794	0,190	2,985	87

1. Date; 2. Temperature; 3. Amount urine; 4. Sp. g.; 5. K oxide; 6. Na oxide; 7. Total; 8. %; 9. All urine voided in bed. Further investigation had to be discontinued because of too great stupefaction.

Remarks:

Onset of illness: morning of 29 May with chills. Crisis during the night of the 9-10th on the 12th day of the illness.

> Table XVIII. Kugh, 22 years old, worker. Ileotyphus

Dat. 🕑	Temper	Haramenge _	Kalys	Natron	Summa	pCe.
8. Febr. 1870.	39,9-39,8	ر بي ا	Cr.	0) (e	ζ
9.	40,5-39,4	1480	2,250	0,518	2,768	81,3
10.	39,8-38,1	1300	2,34	1,196	3,535	66,2
11.	38,8-37,9					

1. Date; 2. Temperature; 3. Amount urine; 4. K oxide; 5. Na oxide; 6. Total; 7. %.

Remarks:

Onset of illness: 30 Jan after several days of prodromal symptoms. Chills. Admission on Feb. 7th. The data was collected during the last few days of the first period.

Very strong constitution, no diarrhea.

- 25 -

Table XIX. As..., male, 20 years old. Ileotyphus

J Dat.	; Temper - A	arnmenge	Ap. G.	Kali _	Natzon -	Sumina	‴ ⊳Ci
15. Mára 187	1. 41,0-39,5	560	1024	0,958	0,258	1,216	78
16. 17.	40,6 - 40,2 40,5 - 40,4	1055	1024	1,807	0,454	2,321	80,3
18. 19.	40,6-39,9) 40,8-30,91	910	1024	1,456	0,300	1,750	82,9
··· -·			_ ·				

1. Date; 2. Temperature; 3. Amount urine; 4. Sp. g.; 5. K oxide; 6. Na oxide; 7. Total; 8. %.

Remarks:

Onset of illness on 5 March after slight prodromal symptoms. - Strong body build, sensorium almost free. Very little appetite, sparce, diarrheal feces. Profuse intestinal bleeding started on the afternoon of the 19th, the patient expired that evening.

I also analyzed the alkali content of feces on a few days in two cases; for this, of course, I could only take light cases. I have already reported the manner of specimen collection and analysis.

Table XX. William Fr., 16 years old. Ilectyphus

G	. ($\widehat{\Sigma}$	్ •)	Urin.	(F)	6:		ſ.
Dar C	Temper	- Harn	mengo sp.	<u>6</u> G- 1	Kali	Natron	Summa	pCi.
4. Juni.	39,9 - 39	9 ,2 1	010 10	15 1	,147	1,247	2,394	42,8
5.	40,0-3	7,6') 18	540 10	10 1	,104	0,902	2,006	55,0
6 .	40,û-3	8,5*) 11	770 10	12 G. n	icht best	immt.——		•
<u>7</u> .	40,0-3	9,2 1	710 10	11 5	,180	3,505	4,685	25,9
(i)) Bi	d und 1,5	Grm. Ch	iaia dea 4.	Abends.				
(-) Ba	d und 1,5	Grm. Ch	ioio den 3.	Abends.				
U		6	こいい	Stuhl.			(\cdot)	
	3.	0,550 6	ali 0,269	Natron	- 0,8	19 69,7	pCL	
	4.	1,103	- 0,457	•	🚥 1,5	60 70,7	•	
	5.	0,690	• 0,393	•	🖚 1,0	88 63,4	•	
	Ó,	0,735	0,620	-		35 Q3,6	•	•
			(3) c) Ur	ia 🕂 Stul	al.		10-	
	4,	2,25 H	iali 1,701	Natroa	🛥 3,9	34 57 p	Ct.2	
	ʻ 5.	1,794	- 1,300	()	= 3, 0	94 58	•	

1. Date; 2. Temperature; 3. Amount urine; 4. Sp. g.; 5. K oxide; 6. Na oxide; 7. Total; 8. %; 9. not determined; 10. Bath and 1.5 gm quinine on the evening of the 4th; 11. Bath and 1.5 gm quinine on the evening of the 5th; 12. feces; 13. urine + feces.

Remarks:

Onset of illness on 21 May.

- 26 -

Table XXI. Brennert. Ileotyphus

1.	C	i a) Uria					í
Temper.	Harnnirnga	Horn-tall 5	, Kali 🛓	> NatronC	- Sumina ¹	- pci. Cr	j١
39,0-38,8	1205 1	014 sp. G.	0,313	0,009	0,482	63	
39,5-39,8	1930	38,02	0,453	0,424	0,907	53,2	
39,4-39,4	2300	\$1, 03	0,851	0.014	1,595	20,9	
38,8 - 39,2	1930	38,03	0,41	0,624	1,034	39,6	
39,0-39,7	1810	42,5	0,471	0,742	1,213	33,8	
39,6-39,0	2010	41,3 .	0,382	0,724	1,106	31,5	
	·	1C , b) 131					
ð.a t.	Kali	Natro	6 S	a mut	րԸւ.		
12.	1,19	0,47	:	1,00	71,0		
13.	2,20	1,3 1		3,54	62,1		
14.	· 1,37	0,03	:	2,00	68,5		
45.	1,28	0,53		1,31	70 ⁷		
tú,	2,37	1,90	5	4,275	13.4		
17.	1,71	(1,38		3,00			
		c) Unin -	- Faces.				
12.	1,503	0,53	9	2,132	70,1		
13.	2,083	1,70	4	4.457	59,9		
14.	2,221	1,27	4	3,495	63,6		
15.	1,000	113	4	2,841	59,4	•	
16.	2,841	2,64	17	5,188	51,7		
17.	2.092	2.10) ;	4,196	49,9	·	
	(- Temper, 39,6-38,8 39,5-39,8 39,4-39,4 38,8-39,2 39,6-39,0 0.4, 12, 13, 14, 15, 16, 17, 12, 13, 14, 15, 16, 17, 17, 12, 13, 14, 15, 16, 17, 17, 17, 17, 17, 17, 17, 17, 17, 17	Temper. Haranica $33, 6-38, 8$ 1205 1 30, 6-38, 8 1205 1 30, 5-39, 8 1930 39, 4-39, 4 2300 38, 8-39, 2 1930 39, 6-39, 0 2010 Dat. Koh 12. 1, 19 i3. 2, 20 14. 1, 37 i5. 1, 28 16. 2, 37 17. 1, 71 12. 1, 503 13. 2, 683 14. 2, 221 15. 1, 630 16. 2, 841 17. 2, 922	$\begin{array}{c} ()^{(2$	(2 - a) Urin. Temper. Harmings Harnstoff S. Kali S. $30,6-38,8$ 1205 $1014 ep. G.$ $0,313$ $30,5-39,8$ 1205 $1014 ep. G.$ $0,313$ $30,5-39,8$ 1205 $1014 ep. G.$ $0,313$ $30,5-39,8$ 1930 $38,02$ $0,483$ $39,4-39,4$ 2300 $41,03$ $0,851$ $38,8-39,2$ 1930 $38,03$ $0,41$ $39,6-39,0$ 2010 $41,3$ $0,382$ List of the sec. Dat. Kali Natron St. 12. $1,19$ $0,471$ 13. $2,200$ $1,31$ 14. $1,37$ $0,63$ 15. $1,28$ $0,33$ 16. $2,37$ $1,905$ 17. $1,71$ $(-1,138)$ Clint + Faces. 12. $1,503$ $0,539$ 13. $2,683$ $1,754$ 15. $1,6900$ 154 1.04	$\begin{array}{c} (-2,-3) \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \$	$\begin{array}{c} (4 - a) \ Urin. \\ \hline Temper. \\ 30,6-38,8 \\ 1205 \\ 1014 \ ep. G. \\ 0,313 \\ 0,009 \\ 0,452 \\ 0,5-39,8 \\ 1205 \\ 1014 \ ep. G. \\ 0,313 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,452 \\ 0,009 \\ 0,451 \\ 0,47$	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$

1. Urine; 2. Date; 3. Temperature; 4. Amount urine; 5. Urea; 6. K oxide; 7. Na oxide; 8. Total; 9. %; 10. feces; 11. urine + feces.

Remarks:

Onset of illness: Apparently on 24 May, bedridden since 6 June, admission to clinic on the 8th.

Fairly light case. - Strong body build. Appetite not completely lacking, considerable diarrhea.

Both cases have a slight preponderance of potassium salts - the second case is interesting because it shows that even with lasting, strong diuresis the greatest portion of the alkali salts can still take another pathway. During the entire 6 days, during which the investigation was carried out, the amount of potassium and of potassium salts was greater in the feces than in the urine. At present we cannot give any reason for this occurrence.

The alkali-salt content of the urine is, in view of its great volume, particularly low in this case. The percentage is about 1/10 of the normal one; even on the 16th - when it is highest - it is far from attaining 1 part per thousand (0.067%).

- 27 -