

agents. What exactly is the active principle which produces these nutritional disturbances we do not know. Sometimes it is tobacco, sometimes tobacco and alcohol combined, sometimes it is other poisons, as opium, hashish, iodoform, etc., and sometimes it is a toxin, as, for example, the toxin of diabetes. I have long ago contended that I did not believe it was tobacco alone, or its active principle, nicotine, which was the essential poisonous agent, but that one or more of the many principles freely present in tobacco-smoke, or some toxic influence which they liberate in the system, must be held accountable for the disease. Sachs contends that even in the tobacco cases certain complex chemical combinations occur in the stomach, and there is a resulting transformation of the normal gastric juices into acids of the fatty type which combine with nicotine to form substances which are more injurious than the simple tobacco-bases themselves. Experimental work now being carried on in Chicago under the direction of Dr. Ossey Wood indicates that certain stomachic toxins are capable of causing in animals blindness, probably of the type under consideration.

UREA-ESTIMATIONS IN CASES OF TYPHOID FEVER TREATED BY THE BRAND-BATH METHOD.¹

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E. B. C., a bookkeeper, aged twenty-five years, was admitted to the hospital of the University of Pennsylvania, December 25, 1896. His family and personal history had no bearing on his existing illness, which began apparently on December 17th with a severe headache. He had headache also two days earlier, but this disappeared and did not return. On the 24th, the day before admission, diarrhoea set in, he having previously been constipated. On this day, too (the 24th), he had a small epistaxis. There had been some cough for several weeks. On admission at 3.30 P.M. his temperature was 102.8° F. At 8.30 in the evening it was 103.8° F. His tongue was slightly coated, but not dry; his belly somewhat distended. The spleen was decidedly enlarged, but there were no rose-colored spots on that day. These were unmistakably present on the 28th. On the day of admission the urine was examined and a small albuminuria found. To this were added, on the 27th, hyaline casts. The diagnosis of typhoid fever seemed sufficiently justified, tubing at 102.2° F. was commenced on the 27th. The effect upon the temperature was prompt, lowering it from 104.4° to 98.4° F., at the fourth bath, received at 11.45 of the 28th. On this day a copious purulent expectoration, tenacious and bloody, was noted, containing also numerous diplococci, but the physical signs of a croupous pneumonia

¹ Read before the Association of American Physicians at its meeting in Washington, May, 1897.

could not be elicited. The aluminaria on this day had increased to one-fourth bulk, and hyaline casts were numerous.

We were not deterred from tubbing by these symptoms, though the bronchitis was deep-seated. Indeed, there was probably a bronchopneumonia. It was thought that this would be a suitable case on which to make a study of the uriae. The diet was the usual liquid diet of typhoid fever, wholly milk.

We began to collect the urine on the 29th, when but eleven ounces and a half were gathered, representing a part only of the twenty-four hours' quantity. This contained 2.9 per cent. of urea. The daily amount was measured and the urea estimated daily for a month succeeding, until convalescence was thoroughly established. The results are:

		Quantity		Urea.	
December	30, 1896	24 oz. (720 c.c.)	2.52 percent.	18.32+ gms. (283.72 grains.)	
"	31, "	40 " (1200 ")	2.65 "	32.11+ " (495.60 ")	
January	1, 1897	48 " (1440 ")	2.14 "	20.46+ " (480.00 ")	
"	2, "	66 " (1980 ")	1.64 "	32.80+ " (506.22 ")	
"	3, "	56 " (1680 ")	1.89 "	31.98+ " (494.43 ")	
"	4, "	60 " (1800 ")	1.77 "	32.16+ " (496.20 ")	
"	5, "	45 " (1350 ")	1.77 "	24.76+ " (382.15 ")	
"	6, "	44 " (1320 ")	2.14 "	28.61+ " (440.00 ")	
"	7, "	52 " (1560 ")	1.64 "	25.84+ " (398.84 ")	
"	8, "	32 " (960 ")	1.51 "	14.57+ " (225.93 ")	
"	9, "	48 " (1440 ")	1.89 "	27.33+ " (421.84 ")	
"	10, "	48 " (1440 ")	1.51 "	21.96+ " (333.88 ")	
"	11, "	68 " (2040 ")	2.14 "	44.07+ " (680.00 ")	
"	12, "	40 " (1200 ")	2.52 "	30.53+ " (471.50 ")	
"	13, "	38 " (1140 ")	2.14 "	24.62+ " (380.00 ")	
"	14, "	62 " (1860 ")	1.59 "	25.48+ " (391.46 ")	
"	15, "	44 " (1320 ")	2.52 "	33.59+ " (518.82 ")	
"	16, "	64 " (1920 ")	1.89 "	36.49+ " (563.12 ")	
"	17, "	42 " (1260 ")	1.64 "	20.81+ " (321.14 ")	
"	18, "	48 " (1440 ")	1.64 "	23.86+ " (368.10 ")	
"	19, "	48 " (1440 ")	1.64 "	23.86+ " (368.16 ")	
"	20, "	56 " (1680 ")	1.89 "	31.98+ " (494.48 ")	
"	21, "	68 " (2040 ")	1.51 "	31.11+ " (480.08 ")	
"	22, "	66 " (2080 ")	1.51 "	30.45+ " (465.96 ")	
"	23, "	30 " (900 ")	1.39+ "	12.63+ " (195.99 ")	
"	24, "	42 " (1260 ")	1.64 "	13.03+ " (201.00 ")	
"	25, "	70 " (2100 ")	1.51 "	29.87+ " (460.92 ")	
"	26, "	70 " (2100 ")	1.39 "	24.76+ " (382.15 ")	
"	27, "	76 " (2280 ")	1.51 "	32.11+ " (495.60 ")	
"	28, "	76 " (2280 ")	1.51 "	32.11+ " (495.60 ")	
		Albumin.		Albumin.	
December	29, 1896, a one-quarter bulk.			January	4, 1897, a one-tenth bulk.
"	30, " " " "			"	5, " a one-twelfth bulk.
"	31, " " " "			"	6, " a trace.
January	1, 1897, " " " "			"	7 to 19, " "
"	2, " one-fifth "			"	24, " None.
"	3, " " " "				

Thus it will be seen that so far from there being a suppression of uric acid during the baths the secretion was markedly increased, amounting to 1980 c.c. (66 + oz.) in the twenty-four hours on January 2d, when he was tubbed five times. In the corresponding twenty-four hours the urea reached 32.8 gms. (506.2 grains), and this, too, on a milk-diet, which affords a moderate percentage of proteids. On the 3d there were 1680 c.c. (56 ounces) and 31.98 gms. (494.4 grains) of urea; on the 4th,

1800 c.c. (60 ounces) with 32.16 gms. (496.2 grains). Thea followed a few days of smaller but still free secretion until the 11th, when 2040 c.c. (68 ounces) were again recorded. Throughout the remainder of the illness the secretion, as will be seen from the table, was liberal but fluctuating. He had his last bath, the fifty-third, on January 12th. On the 11th, when two baths were given, the urine rose to 2040 c.c. (68 ounces), with the urea at 44.07 gms. (680 grains), the maximum attained while the analyses were being made. On the twenty-fifth, twenty-sixth, twenty-seventh, and twenty-eighth days of the disease the quantities were 2100 c.c. (70 ounces), 2100 c.c. (70 ounces), 2280 c.c. (76 ounces), 2280 c.c. (76 ounces), with urea amounting to 29.87 gms. (460.9 grains), 24.76 gms. (382 grains), 32.11 gms. (495.6 grains), 32.11 gms. (495.6 grains).

The albumin remained at one-quarter bulk until January 2d, when it fell to one-fifth. On January 5th it was one-twelfth bulk. After that date it disappeared and with it the casts.

No treatment was ordered for the cough, and on January 4th the notes inform us that it continued, though showing some abatement, the expectoration being still copious and mucopurulent, sometimes bloody. On the 7th, we noted that his cough was less, but expectoration about the same. On this day the sputum was examined for tubercle-bacilli with negative results. By the 18th the cough had abated greatly, though the expectoration still contained some blood. At the date of his discharge, February 12th, the cough and expectoration had completely disappeared. A physical examination on the day previous failed to discover anything abnormal.

On January 12th we noted "the patient has developed an otitis media," and on the 18th "both ears are noted as discharging;" on the 20th "that he is deaf," but in other respects is doing well. There appeared no defect in his hearing at the date of discharge.

This is the history of a case of typhoid fever strikingly complicated with conditions which superficial observation would expect to increase any risk to which the patient is subjected by cold bathing, viz., nephritis and deep-seated bronchitis, possibly bronchopneumonia. So far from aggravating either of these two conditions, both abated and early disappeared. The urine, so far from falling off, increased in quantity, and the urea-elimination increased *pari passu*.

The nephritis was, of course, mild, and it is not likely that the kidney-lesion amounted to more than cloudy swelling of the cells. Whether a more active inflammatory condition of the kidney would justify continuing the baths is not so simple a question. Our associate, Dr. Charles G. Stockton, in his part of the splendid symposium held at the annual meeting of the Medical Society of the State of New York, at Albany, on January 28, 1897, "On the Relation of Impure Water to Disease, and the Cure and Prevention of the Latter," names renal complications and intestinal hemorrhage as the only two conditions which contraindicate the full method of Brand. By renal complications he specifically states he means actual inflammatory involvement of the renal epithelium, and

probably did not intend the simpler condition of cloudy swelling present in my case. In point of fact, such a positive degree of inflammation is rarely met with in typhoid fever; but I am inclined to think I would not be deterred from using the tub-treatment by the presence of any degree of nephritis, provided the conditions otherwise calling for it were present. For, after all, in inflammation of the kidney it is not the rapid movement of the blood through the organ which constitutes the inflammation, but rather its stagnation. Now, the direct effect of cold is to increase the force of the heart's contraction, to impel the blood onward and carry it and its toxic ingredients through the kidney, in the course of which a large proportion of these toxic matters are excreted with the urine. E. Ausset¹ has shown conclusively that the toxic properties of the urine secreted during the baths is increased, as contrasted with cases of typhoid not tubbed. Indeed, he assigns almost the entire efficiency of the Brand treatment to this active elimination of toxic agencies.

Vogel's experience supports my own notion as to the harmlessness of the baths, even if active nephritis is present. He refers to an epidemic of scarlet fever among soldiers, a part of whom, on account of circumstances, were treated on the expectant plan and a part with cold baths at 68° to 72° F. every three hours, when the rectal temperature reached 102.2° F. Of the 69 patients subjected to the cold-bath treatment, 5 had nephritis, of whom none died. Of 56 treated by the expectant plan 9 had nephritis and 5 died.

The effect of cold baths in exciting secretion of urine has long been recognized, and almost every person who is in the habit of taking a frequent cold bath has experienced such effect in his own person. It is not remarkable, therefore, that diuresis should be a constant event of the Brand method of treating typhoid fever. The earliest observers of the Brand method called attention to this fact, and every one who has watched the effect of the treatment confirms this. As many as 4000 and 5000 cubic centimetres have been noted.

I have not as yet met the results of systematic urea-estimations daily carried out in the study of the effects of this treatment. The sources of urea in the urine in fever are two: first, the nitrogen in the proteid food ingested; secondly, the oxidation of tissue, which is much increased during the fever-process. It is comparatively easy to estimate the nitrogen ingested by my patient. Being on a milk-diet throughout the whole period of observation, six ounces every two hours, or 72 ounces in the twenty-four hours, the daily amount of milk may be put down at 2232 grammes. Allowing 600 milligrammes of nitrogen for each 100 grammes of milk, the approximate daily amount of nitrogen would be 13.382

¹ E. Ausset: *Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris*, 1894, Tome II., 3e Serie, 1894, p. 385.

grammes. Of the total nitrogen eliminated, 95 per cent. may be approximately assigned to the urine, and 5 per cent. to feces, whence 12.7 grammes would be eliminated in the urine. Of this, however, 10 per cent. is uric-acid nitrogen, and 85 per cent. or 11 grammes urea nitrogen. Finally, since the ratio of urea to nitrogen is as 60:80, the proper amount of urea derived from the food ingested would be about 24 grammes if all the milk ingested was absorbed. In point of fact, the actual amount absorbed is probably considerably less than is ingested, so that the urea should be correspondingly less. The urea arising from the tissue-metamorphosis is, of course, disregarded; but allowing for this, the results of my analyses show that the kidneys are quite equal to the work demanded of them. It would be more satisfactory to compare these results with analyses of the urine of a case of typhoid fever on a like diet not treated by the baths. At present, my confidence in the hydratic treatment is such that I would not feel comfortable to treat a case in any other way unless such treatment was insisted upon by the patient.

ANGINA PECTORIS:

ITS RELATION TO DILATATION OF THE HEART.

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THE purport of the following paper is the consideration of angina pectoris inasmuch as it bears relationship to dilatation of the heart. It includes an estimate of the influence of this relationship in the prognosis and treatment of the subjective symptoms (pain), on the one hand, and of the organic condition (dilatation) on the other. The paper is entirely clinical, and is a record of the writer's observations, not of angina pectoris, but of such of his cases of this affection which manifestly exhibited the relationship above mentioned.

It will be appreciated, the writer trusts, that he is fully cognizant of the difficulties that attend a diagnosis of *angina pectoris*; that he is alive to the fact that, in spite of consummate care, we may err in the diagnosis; that he is aware that the occurrence of cardiac pain is not alone sufficient to admit of the diagnosis.

The presence of the symptom-complex so skilfully portrayed by the earliest historians of this affection, Heberden and Jeaner, and more fully elaborated by Latham and Gardiner, is essential to the formation of a diagnosis, although we cannot fail to agree with the dictum of the latest author, our distinguished colleague, Professor Osler, that "pain about the heart of an agonizing character, occurring in paroxysms, is the dominant feature."

We will all admit also that in angina pectoris we are confronted with the facts that it usually occurs in males; that it is an affection of the latter decade of life; that the patients are of the better class, usually well fed, and that they are often the victims of other affections (gout, diabetes, etc.), indicating a more or less sedentary life, with an abundance of food which contributed to the physiological hypertrophy that proceeds with advancing years.

Before proceeding further it may be well to state that we can reasonably infer the presence of *dilatation of the heart* by the physical signs of displaced apex-beat, gallop-rhythm, a soft, regurgitant murmur in the tricuspid or mitral area, by venous phenomena, and by the congestions, cyanosis, and dropsy that attend this affection. The results of cardiac percussion may be confirmatory, but are not looked upon as essential in the diagnosis of cardiac dilatation.

Bearing in mind these fundamental propositions, the writer begs to submit cases (it is to be regretted they are too few) to illustrate the following points:

1. When dilatation of the heart supervenes in a patient the subject of an attack or attacks of angina pectoris the subjective symptoms may subside. At the same time the physical type of the individual changes. (Cases I., II., and III.)
2. Angina pectoris may occur in a patient who has had dilatation of the heart when the organic condition (dilatation) is removed by treatment. (Cases IV. and V.)
3. True angina, when it occurs in dilatation of the heart, admits of a prognosis more favorable than when it occurs with other mural conditions, as myocarditis or hypertrophy, without dilatation. (Case VI.)
4. Grave cases of dilatation of the heart, conversely to the above, may be looked upon as amenable to successful treatment if the patient should have paroxysms of true angina pectoris.
5. In the treatment of angina pectoris digitalis is of doubtful value, not to be given unless there is an excess of dilatation.
6. The pain of angina appears to be due to increased intraventricular pressure, although other causes are no doubt operative.

CASE I. Angina pectoris; dilatation of heart; relief to angina; death from angina sine dolore.—A. W. R., aged sixty-seven years, merchant, consulted me for dyspnoea and pain April 3, 1894. Smoked to excess, but did not use alcohol. He had been subjected to severe mental strain. He took but little physical exercise. He had inflammatory rheumatism in 1874; otherwise previous health good.

The patient had the first attack of severe pain after ascending a steep incline. He had had moderate dyspnoea and oppression on exertion previously, but the attack just referred to was such as to compel him to discontinue his efforts. It was noticeable, too, because it was attended by a cold perspiration and the patient was compelled to assume a fixed