times the normal, provided the lumen of the arterial system remains the same and the rigidity of the venous system remains normal and, therefore, much lower than the rigidity of the arterial. 2. The minute volume of blood flow in the systemic arterial system is not decreased more than 5% when the "effective" coefficient of volume elasticity of the arterial system is increased 10 times the normal, provided the lumen of the arterial system remains the same and the rigidity of the venous system remains normal and, therefore, much lower than the rigidity of the normal arterial system. 3. Apparently at times the systolic ejection time is lengthened and the diastolic filling time of the left ventricle decreased when the rigidity of the arterial system is increased greatly, thus causing slightly decreased diastolic volume of the ventricles and decreased stroke volume. The coronary flow is slightly decreased by the increased length of systole. The external work of the heart is slightly decreased also. These conclusions must be confirmed by optical recording and more accurate stroke volume measurements.

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Presence of the Morning Alkaline Tide in a Case of Achlorhydria.

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In previous communications from this clinic it has been stated that the urinary alkaline tide is primarily due to the secretion of hydrochloric acid by the stomach, for the tide was absent in most cases of achlorhydria.[1] This observation has been confirmed by Ackman[2] and Davies.[3] Further study showed that it was necessary to distinguish between alkalinity developing during the day-time and that shown immediately after awakening,[4] for this latter change is frequently seen in patients where hydrochloric acid is not found in the stomach, and is probably due to the respiratory adjustment of the subject to waking conditions.[5] Recently we have studied a patient with achlorhydria who showed a tide exactly resembling

[1] Munford, S. A., and Hubbard, R. S., *J. Am. Med. Assn.*, 1926, lxxxvii, 922.

[2] Ackman, F. D., *Canadian Med. Assn. J.*, 1925, xv, 1099.

[3] Davies, D. T., *Brit. J. Exp. Pathol.*, 1929, x, 1.

[4] Hubbard, R. S., *J. Biol. Chem.*, 1929, lxxxiv, 191.

[5] Leathes, J. B., *Brit. Med. J.*, 1919, ii, 165.

those found in normal subjects. It seems desirable for us to report this case because the absence of hydrochloric acid was found after histamine had been injected—a method giving as complete proof of functional disability of the stomach as can be obtained. In many cases where a urinary tide has been present, but hydrochloric acid apparently was absent from the gastric juice, further gastric studies have failed to confirm the abnormal finding of the first one,[6] and it has been claimed by some that the presence of a satisfactory alkaline tide furnishes definite proof that the stomach secretes hydrochloric acid.

The patient was a man 46 years old who was a mechanical engineer by profession. He had occasional periods of mild diarrhoea, and symptoms which were somewhat suggestive of the presence of a peptic ulcer. The stomach emptied rapidly, as frequently is the condition in cases of achlorhydria. Gastric analyses were carried out, and the reaction of successive samples of morning urine determined. A discrepancy between the results of the 2 tests was noted, and further studies were accordingly made.

Free hydrochloric acid was not found in the gastric contents, even when histamine in the maximal amounts used in such tests was given. The case therefore should be considered as one of achlorhydria.

TABLE I.

Studies of the alkalinity of the urine.

Period spent	Morning meal	Reaction of urine specimens.						
		Time.						
		to 7	7-8	8-9	9-10	10-11	11-12	12-1
		pH	pH	pH	pH	pH	pH	pH
up	taken	5.1	5.7	6.6	7.3	8.0	7.5	6.8
abed	taken	5.1	5.5	5.9	6.5	7.5	7.3	6.7
abed	omitted	4.9	5.1	5.3	6.6	6.9	7.6	6.8
					Time.			
			11-1	1-3	3-5	5-7	7-9	
			pH	pH	pH	pH	pH	
Afternoon tide			6.5	5.3	5.2	5.2	5.2	

The table shows the urinary reaction determinations made upon this patient. Atypical morning alkaline tide was present whether a meal was fed or omitted. Exactly similar results have been obtained on various normal subjects.[7] The tide was also demonstrated when the patient was in bed, thus probably reducing respiratory

[6] Hubbard, R. S., *Clifton Med. Bul.*, 1928, xiv, 7.

[7] Hubbard, R. S., and Steele, T. M., *J. Biol. Chem.*, 1929, lxxxiv, 199.

changes to a minimum, as well as when he was up and moderately active. The afternoon tide, which seems to be present in most normal subjects,[8] was lacking.

The morning tide observed was almost certainly not produced by the secretion of hydrochloric acid by the stomach, nor was it connected with the ingestion of a meal. The experiments also seem to show that it was not caused by respiratory adjustment to waking conditions. It is possible that in this subject there is an unusual persistence of the "habit" alkali-acid rhythm implied by the work of Muschat[9] and others. It is known that the rhythm sometimes persists when the stimulus to gastric secretion is absent, and here it may have persisted when acid secretion by the stomach had wholly ceased. Whether this explanation is or is not adequate, such a case as this, where an absence of hydrochloric acid is clearly demonstrated in a patient who shows a typical alkaline tide, makes necessary a somewhat conservative attitude towards the clinical interpretation of alkaline tide studies.

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Effect of Metals Purified by Electrolytic Deposition on Hemoglobin Regeneration in Anaemic White Rat.

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Recent work by Hart, Steenbock and others[1] and Myers and Beard[2] has given rise to opposing results as to the value of metals other than copper in the regeneration of hemoglobin in the anaemic white rat. The suggestion has been made[3] that these differing results were due to the fact that the work was carried out in 2 rather widely separated localities. It has seemed to us that the publication of our results obtained in a third and entirely different section of the country might be of value as bearing on this point.

[8] Hubbard, R. S., Munford, S. A., and Allen, E. G., *Am. J. Physiol.*, 1924, lxviii, 149.

[9] Muschat, M., *J. Clin. Invest.*, 1926, ii, 245.

[1] Waddell, J., Steenbock, H., and Hart, E. B., *J. Biol. Chem.*, 1929, lxxxiv, 115.

[2] Myers, V. C., and Beard, H. H., *J. Am. Med. Assn.*, 1929, xciii, 1210.

[3] Discussion by Drs. Hubbard, Steenbock and Myers, *J. Am. Med. Assn.*, 1929, xciii, 1212.