gestion of glycine. Or, what is more likely, it may have resulted from the loss of glycine through the urine, with an exacerbation of the defect in the creatin metabolism.

The normal synthesis of glycine in these patients and the ready formation of creatin when they ingest glycine, tend to place the defect in creatin metabolism beyond these stages. The inference is that the disturbance is located in the muscle itself, probably in the enzyme system which controls the breakdown and building up of phosphocreatin. The thyroid hormone is apparently involved in this process.

7062 P

The "Q" Deflection in Normal and Abnormal Human Electrocardiograms.

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(Introduced by Harry Gold.)

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Recent literature[1, 2, 3, 4] contains much work on the significance of the Q-wave, and, more particularly of a large Q-3. Attempts have been made in some of these studies[1, 4] to explain the mechanism underlying the production of a large Q-wave. Our knowledge of the mechanism of the normal Q-wave (*e. g.,* Lewis' and Wilson's theories) has been used as a basis for discussions of the abnormal Q-waves. Conclusions have been drawn by one author[4] from pathological correlations that the large Q-wave is representative of changes in a well localized portion of the heart (the left half of the septum posteriorly). Insufficient emphasis has been placed on the fact that the names Q, R and S are entirely arbitrary and that the Q-R-S system of terminology frequently gives the same name to parts of the electrocardiogram in the 3 standard leads that do not correspond in time, and different names to parts that do so correspond. We attempted an analysis of the Q-wave in normal records and in records of the large Q-3 type (described by Pardee[1])

[1] Pardee, H. E. B., *Arch. Int. Med.*, 1930, **46**, 470.
[2] Willius, F. A., *Am. Heart J.*, 1931, **6**, 723.
[3] Carr, F. B., Hamilton, B. E., and Palmer, R. S., *Am. Heart J.*, 1933, **8**, 519.
[4] Fenichel, N. M., and Kugell, V. H., *Am. Heart J.*, 1931, **7**, 235.

to determine the relative time occupied by this deflection in the belief that this time data would throw light on the significance of the Q-wave in normal and abnormal electrocardiograms. We obtained our time data by using Einthoven's formula: *i. e.,* when on any and every vertical line the electrocardiographic curves can be superimposed so that the height in Lead I added algebraically to that in Lead III gives the height in Lead II, the time relations are identical.

Results. In each of 25 normal ventriculograms Q-1, Q-2 and Q-3 corresponded in time with reasonable accuracy, suggesting that the Q-wave represented a more or less fixed physiological phenomenon in these instances. In ventriculograms of the large Q-3 type (fulfilling Pardee's criteria[1]) Q-3 did *not* correspond in time with Q-1 or Q-2. The time represented by Q-3 in 50 of these tracings (taken from 1200 consecutive electrocardiograms) was in *no* case identical with that occupied by Q-1 and Q-2. In *most* of these tracings Q-3 corresponded in time with parts of R-1 and R-2. In many of these tracings Q-3 corresponded more or less accurately in time with R-1 or R-2.

The results given above indicate that the *large* Q-3 is not comparable with the normal Q-wave in that it represents a grossly varying time interval after the beginning of the Q-R-S and cannot, therefore, represent a fixed phenomenon in the heart.

In studying the electrocardiograms of the large Q-3 type it was apparent that the "R" and "S", and possibly "P", as well as "Q" waves, were altered in a characteristic way. As Q-3 got larger (*e. g.,* with respiratory changes) R-3 became smaller, S-3 disappeared and P-3 occasionally became inverted. These facts suggested some alteration of the electrical axis of the ventricle as a whole rather than of some limited portion of it, and at times of the electrical axis of the auricle as well. Meek and Wilson[5] showed that rotation of the dog's heart about a longitudinal axis produced a large Q-wave. This work suggested that a large "Q" in humans might be indicative of a rotation of the ventricular electrical axis about a longitudinal axis; rotation to the left producing a large Q-3, and rotation to the right a large Q-1. Such a thesis appeared to explain all the known facts about the large Q-wave—including its relative frequency in cases with coronary artery disease,[1] in pregnant women,[3] in normal infants[6]; and its occasional occurrence in normal adults.[7]

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[5] Meek, W. J., and Wilson, A., *Arch. Int. Med.*, 1925, **36**, 614.

[6] Seham, M., *Am. J. Dis. Child.*, 1921, **21**, 247.

[7] Ziskin, Thos., *Arch. Int. Med.*, 1932, **50**, 435.

Rotation of the ventricular electrical axis could be the result: (a) of mechanical factors that change the position of the ventricle; (b) of a change in the position of the leads that causes a relative rotation of the heart; (c) of damage to one part of the ventricular musculature with collapse, the contraction of scar tissue, or dilatation of the damaged area; (d) of thickening of one part of the ventricular musculature.

The work of Pardee,[1] Willius[2] and others suggests that most large Q-waves are caused by the mechanism described in "C" above, but in any particular electrocardiogram there is no more reason for considering a large Q-3 or a large Q-1 of itself indicative of coronary artery or myocardial disease than there is for regarding a deep S-3 as always indicative of hypertrophy of the left ventricle.

7063 P

Antistreptolysin Titre of the Serum in Acute Glomerular Nephritis.

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Todd[1] has demonstrated that the serums of animals immunized with *S. hemolyticus* develop a significant amount of an antibody which inhibits the activity of the streptococcal hemolysin. The serums of animals immunized to a number of other bacteria (pneumococcus, hemolytic staphylococcus, non-hemolytic streptococcus, and *B. diphtheriae*) do not contain appreciable quantities of this antihemolysin.

In subsequent studies Todd[2] working in conjunction with Coburn and Pauli[3] showed that there was a consistent increase in the antistreptohemolysin titre (termed antistreptolysin by Todd) in human serums *after* hemolytic streptococcus infections. This antibody did not appear in significant amounts in the serums of patients convalescing from such other infections as lobar pneumonia, hemolytic staphylococcus osteomyelitis, joint tuberculosis and measles. The presence of high antistreptolysin titres in the serums of patients with rheumatic fever was additional immunological evidence lead-

[1] Todd, E. W., *J. Exp. Med.*, 1932, **55**, 267.

[2] Todd, E. W., *Brit. J. Exp. Path.*, 1932, **13**, 248.

[3] Coburn, A. F., and Pauli, R. H., *J. Exp. Med.*, 1932, **56**, 609.