ANY investigation dealing with the U wave has to surmount many difficulties: the wave is small and appears often only as a slight undulation of the 0 line; it varies in size and shape with respiration, it fuses with the following P at a heart rate over 100 and with the preceding T at a rate of 40 to 50.1 The T-U fusion seems to be rather related to the Q-T duration than to the rate itself.

Fig. 1. (Top) U wave in patient with anteroseptal infarction. It is present during the acute stage, disappears during deep T negativity, reappears after healing.

Fig. 2. (Bottom) Absence of U during septal involvement of posterior infarction (nodal rhythm with retrograde P); its reappearance when septal involvement subsides.

There are, however, other U changes that are exceptional in normal records such as (2) diphasic U waves in left precordial leads, and (3) U of a delayed appearance (eT-aU greater than 0.12 sec.).4 Minor abnormalities include (4) T:U ratio less than 1:1 or 1:2. Under normal conditions T in V2 or V3 is 5 to 20 times taller than its

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XV. U Wave in Coronary Disease

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U IN ACUTE CARDIAC INFARCTION

During the acute stage of anterior infarction (high S-T take-off) U is upright; when terminal T inversion develops U disappears because it is buried in the prolonged Q-T period; when the Q-T period shortens U may reappear again and then follows the direction of T. At the healing stage, when T becomes upright, U also returns to normal (fig. 1).

Posterior infarction does not influence the U wave of the anterior chest leads except when through septal invasion ectopic bradycardia develops, when U is obliterated by the prolonged Q-T period. Here U reappears when the septal invasion subsides and the rate becomes normal (fig. 2).

Admittedly this is a schematic version that has many exceptions; however, the U-wave changes in acute infarction are of secondary importance as other abnormal features are present. It is in chronic coronary disease, where the S-T changes are of a lesser degree, that the presence of abnormal U waves may help in the diagnosis.

U WAVE IN ANGINA AND CORONARY INSUFFICIENCY

In angina pectoris inverted U waves never appear in V2 or V3; at the most U is isoelectric in these leads. Inversion is seen in V4, V5, and V6 and it develops gradually from V2 onwards. The T-aU distance lengthens in V2. U becomes diphasic in V3 and V4 and inverted in V5, V6, and V4 (fig. 3A). U inversion at times is conditioned by S-T depression and not by T inversion (fig. 3B). An inverted U wave may be the only sign of coronary disease. It may be more prominent in lead III and aVF when the heart is vertical (fig. 4).

During the effort test in coronary disease an upright U or an isoelectric U may become inverted while the S-T interval remains isoelectric (fig. 5B); a negative effort test increases the voltage of U (fig. 5A). It is however, exceptional that this should be the only abnormality in the electrocardiogram. Under effort an abnormally inverted T may become upright (paradoxical reversal); at the same time U returns to normal through a diphasic pattern
U WAVE IN CORONARY DISEASE

Fig. 5. (Top) A. U increases after negative effort test. B. U inversion after positive effort test; anterior infarction 2 months later.

Fig. 6. (Bottom) U inversion on effort associated with paradoxical reversal of T in V2.

More often a pathologic S-T depression is associated with the inversion of U (fig. 7).

U wave inversion is not necessarily abnormal; it is seen in advanced age without heart disease though it is difficult to be certain whether the coronary circulation is normal at this age even if there is no angina (fig. 8).

In many patients with U inversion after effort, there is no U wave to be seen in the resting record; in fact the complete absence of U waves, exceptional in normal records, was found in 6 per cent of patients with angina (fig. 9A, B). In figure 9C, U waves failed to appear after effort.

An abnormal T:U ratio may depend on a diminished voltage of T, on an increased voltage of U, or on both. In figure 10A and B, left ventricular strain was present as well, which diminished T and may have increased U in right ventricular leads; in figure 10C the disproportion was due to the small voltage of T only. All 3 had moderately severe angina. Digitalis also realizes identical conditions by the R-T changes it produces and by the absolute increase in the voltage of U (fig. 11). However, this feature may not be necessarily abnormal. Occasionally neurocirculatory asthenia may show taller U than T waves in leads II and III (fig. 12).

In presence of an abnormal T:U ratio the U wave may be confused with the second peak of a bifid T wave. This can be easily avoided by
Fig. 7. U inversion on effort associated with R-T depression. A. Absent U waves in resting record; B. Regression of U inversion through its diphasic pattern.

Fig. 8. (Top) U inversion in old age. Heart normal, no angina.
Fig. 9. (Bottom) A and B. Absent U waves in patients with severe angina. C. No U wave on effort.
Fig. 10. Abnormal T:U ratio and discordant U in V₁ due to increase of U in left ventricular hypertrophy in A and B, and to decrease of T in C. All 3 with angina.

Fig. 11. Abnormal T:U ratio due to increase of U through digitalis, in patients with hypertension, angina, and atrial fibrillation.

Fig. 12. Abnormal T:U ratio in patient with vertical heart and neurocirculatory asthenia.
measuring the Q-aU distance in V₂ or any other lead with an upright U wave and reporting it to the lead in question; alternately if the position of U is doubtful in all leads, the Q-T distance should be determined; the addition of 0.10 sec. to this will show the approximate position of a U if the rate is between 60 and 80.

What is the diagnostic importance of abnormal U wave in coronary disease? Viewed through critical eyes, I do not believe it is great. In acute cardiac infarction, with so many diagnostic signs, the U-wave changes are unimportant.

In 30 per cent of patients with chronic coronary disease the U waves are normal. In cases with abnormal T:U ratio (28 per cent) or upright U with inverted T (20 per cent) the same factors that influence the R-T changes—left ventricular hypertrophy and digitalis—will make the assessment of the abnormal U wave difficult. Of the remaining 22 per cent, the absence of such an elusive wave as U in 6 per cent can hardly constitute an important diagnostic sign though it calls for an effort test. The U-wave inversion or delay without T-wave changes in the remaining 16 per cent could be regarded as diagnostic if it could not be seen at an advanced age without clinical coronary disease. On the other hand, its appearance during effort test with anginal pain is a pointer toward its coronary origin. The most that can be said is that in the presence of equivocal S-T and T changes or atypical chest pain, an inverted U wave is a useful confirmatory sign of coronary disease.

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