II. Various Types of Fusion Between T and U Waves

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The study of the U wave and of its clinical significance depends upon its accurate recognition. Accurate identification of the U wave is also needed for a correct determination of the Q-T duration. This identification is sometimes difficult because of superposition and fusion of the T and U waves. The term “superposition” is applied to patterns in which these waves are only partially merged and a notch is present by which the 2 components can be separated. The term “fusion” implies uniform monophasic or diphasic waves that show no landmarks for the differentiation of their components. Although the partially merged T and U waves have also frequently been misinterpreted as T waves, the greatest difficulty lies in the recognition of TU-fusion patterns.

The appearance of TU-fusion waves depends on certain peculiarities of the component waves, which are not the same for monophasic and diphasic patterns. The conditions under which upright TU-fusion waves appear will be investigated first. On analysis of such patterns, 3 types of monophasic TU-fusion waves can be differentiated.1

Figure 1A demonstrates sinus arrhythmia after mild exercise in a 29-year-old man with neurocirculatory dystonia. At the beginning and at the end of the tracing the U waves are rather high, their summits are drawn near the T waves (T-aU = 0.05 sec.), and the junctions between T and U are elevated. These are typical superposition TU waves. In the middle part of the tracing, on the other hand, during a phase of bradycardia, there is a uniform broad and high wave in lead II. The apex of this wave is situated between the summits of the T and U waves in lead I, where it can be distinguished well. To understand the mechanism of fusion of the 2 waves in lead II, we must consider that the apex of the T wave occurs later due to the slowing of the rate, that the U wave becomes higher for the same reason, and that the apex of the U wave remains fixed or is even drawn nearer to the T wave. Consideration of lead I shows that both T and U are involved in the formation of the fusion wave in lead II.

In figure 1B the 2 forms of merging of T and U in this case are analyzed. For the double TU wave (complex 2 of lead II) we must assume that the U wave begins before the T wave ends but after the apex of T. The fusion TU wave (complex 4 of lead II), on the other hand, is determined by the U wave beginning not only before the end of the T wave, but also at or before the apex of T. This fusion pattern with a new and higher summit may be called a “TU-fusion wave with a summation apex” or with “equidominant T and U components,” in which the T and U components hardly differ in their amplitude and the U-wave upstroke is steeper than the T-wave downstroke.

Figure 2A shows a normal resting electrocardiogram of a 44-year-old man with silicosis. The Q-T duration, the interval between Q and the second heart sound, and the relation between the summits of U and T are normal. Immediately after ascending 6 flights of stairs (fig. 2B) the Q-T interval in the leads I-III and the interval from Q to the second heart sound are shortened more than would be expected at the resulting heart rate. In the precordial leads, however, a higher upright wave appears, the termination of which produces an interval about as long as the expected Q-U interval. Therefore we must assume that this wave is a TU-fusion wave. This mechanism becomes obvious in succeeding tracings. One half minute later (B2) a notch appears that coincides with the notch separating the U wave from the T wave in the limb leads. The emerging U wave is much lower than the T wave. The interval T-aU as well as the interval Q-U appear to be shortened.

Figure 3 from the same case shows in dotted lines the resting pattern in leads II and V₃, with the second heart sound designated by an arrow, and in solid lines the findings after exercise. The tall waves in V₃ cannot be ex-
Fig. 1. Analysis (B) of an example (A) of superposition (complex 2) and complete fusion (complex 4) of equidominant T and U waves during respiratory arrhythmia. In this and in the following figures, QT indicates the distance (in hundredths of a second) from the beginning of QRS to the end of T (Q-T), QcT is a similar distance to the apex or "culmination" of T (Q-aT), QU is the distance to the end of U (Q-U), and QcU is the distance to the apex of U (Q-aU). The time lines in all figures are 0.02 and 0.10 sec. apart.

Fig. 2. Development and regression of TU fusion with predominance of T. A. At rest. B1-B4. After exercise. C. Comparison of the values, as in figure 1, plotted against the heart rate ("frequency"), with the normal average and extreme values of Q-T (3 curved lines). Q2HS = distance to the beginning of the second heart sound; QFW = distance to the end of fusion wave.
plained as the result of fusion of T and U waves of similar height (fig. 3b), as was done in the preceding case. This explanation is contradicted by two facts: the U wave would then begin before the second heart sound, which is not in agreement with our experience; and the U wave, which can be seen in some of the tracings, is much lower than the T wave. This fusion wave must therefore be attributed to superposition of a high T and a small U wave. Considering the onset of the second heart sound, one must suppose that the U wave begins after the peak of the T wave (fig. 3c). The descending branch of the T wave crosses the apex or the descending limb of the U wave, and the apex of this fusion wave is identical with the peak of the T wave. This type can be called a "TU-fusion wave with predominance of T." From a similar analysis of the limb leads we must conclude that the Q-T interval is much longer in precordial leads V₂ and V₃ than in the limb leads, but that the Q-T interval is not so prolonged as it would seem if the fusion wave were considered simply a T wave. We can conclude that there may be a difference in length of the Q-T interval be-

between the sagittal and the frontal planes. Such an abnormal Q-T prolongation in the sagittal leads is not surprising, since, in our case, it can be attributed to the effect on the right ventricle of impairment of the pulmonary circulation.

Figure 4 was obtained from a patient in diabetic coma under insulin treatment. The broad upright waves seem to be T waves but last much longer than the interval from the Q to the second heart sound, the interval from Q to the carotid notch, or the Q-T interval predicted according to the heart rate. An almost invisible notch in lead V₅ strongly suggests that in this case also we are dealing with TU-fusion waves. The summit of this broad wave occurs very late. If we assume that the U wave does not begin before the second heart sound but begins with the apex of T, that the ascending branch of U is steeper than the descending branch of T (this is plausible since the U wave is much higher than the T wave), and that the T wave ends before the apex of U (see lower diagram of figure 4), the analysis reveals a new type of TU-fusion wave. It may
be called "TU-fusion wave with predominance of U." The apex of this wave is identical with the apex of U. Hypopotassemia is the most frequent condition favoring such a pattern.

Lepeschkin and Suraicz\(^3\) have demonstrated in a very clear manner 16 possible combinations produced by partial merging of upright, diphasic, and inverted T and U waves. Figure 5 shows the 48 theoretical patterns that can result from different degrees and types of merging of T waves of different heights with different forms of U waves. These patterns are constructed on the basis of the assumption that the U wave begins with the second heart sound and with the apex of the T wave. In the case of a marked predominance of T and prolongation of Q-T, the same patterns can appear also if U begins later. When the resulting wave is triphasic or shows a notch, the composition of the wave from 2 separate components is obvious; this is indicated by an arrow. In the first column, (a) positive U waves, the TU-fusion waves with a predominant T component appear twice (1a, 2a). The type

**Fig. 5.** Patterns caused by superposition or fusion of different types of T waves with different types of U waves.
with a summation apex appears once (3a). The type with a predominant U wave is represented only by the superposition of an isoelectric T wave upon the U wave (4a–d). In this case the pattern could be mistaken for a T wave with a prolonged Q-T interval and a long isoelectric S-T segment. The occurrence of the second heart sound before this wave, however, is a valuable aid in the correct interpretation. Unless the patterns are compared with the patterns in other leads, all of the complexes in row 4 could be interpreted as showing flattened T waves followed by U waves.

The superposition of upright T waves and diphasic U waves (fig. 5, b and c) does not tend to produce uniform smooth fusion waves. In most instances a notch is present between the waves. But there also may appear a diphasic wave with a flat-topped upright component resembling the configuration shown in pattern 2b. The superposition of an upright T wave upon an inverted U wave (d) may produce a plus-minus fusion wave. If T is high, the pattern may be mistaken for a diphasic T wave with a peaked terminal negative component (1d). Such examples occur during transient myocardial ischemia with anginal pain. In the 3 upper examples of the last column of figure 5 are conditions that can produce uniform diphasic fusion waves without any overlapping or notching. On the other hand, if T is isoelectric (4b–d), the U wave negativity is easily mistaken for T wave negativity. The patterns in row 4 may be called “pseudofusion” waves because there is no T wave component in the respective leads. Only through comparison with simultaneously registered leads that present distinct T waves is produced and the Q-T interval extended.

The superposition of diphasic T waves and various types of U waves (fig. 5, rows 5 through 8) often produces polyphasic patterns that are not difficult to recognize. In some instances, however, diphasic fusion waves may appear. In the case of plus-minus diphasic T waves, a diphasic fusion wave is produced by a superposition of an inverted U wave (5d). Such fusion waves can be seen in lead V3 of figure 6, situated in the transitional zone between the upright and the inverted T waves. Another example (fig. 5, 6b) is produced by the combination of a diphasic “coronary” T wave together with a plus-minus diphasic U wave. In these cases the typical aspect of the T wave is abolished. In the presence of a minus-plus diphasic T wave a fusion wave may result with the aid of a U wave of the same type (fig. 5, 7c and 8c). If the upright component of the T wave is high, the resulting upright deflection is expanded. If T is chiefly directed downward and its upright component very short, the resulting fusion wave becomes a short positive terminal deflection (8c). A similar pattern results from superposition of this latter type of T with an upright U wave (fig. 5, 8a). A great number of the very striking diphasic patterns in hypopotassemia may be due to this kind of superposition because of a high upright U wave component (fig. 7). If the overlapping of T and U is shorter and the U wave lower, there results a pattern similar to that shown in figure 5, 8a and 8b, and frequently seen in leads I, V5, and V6 in the presence of left ventricular hypertrophy. For practical convenience the typical diphasic pattern with initial negativity (fig. 5, 7c, 8a, 8c) may be compared to the letter S placed on its side or a “recumbent S,” the patterns with initial positivity with a “recumbent mirror-image S.” The frequent pattern resulting from superposition of a minus-plus T wave having a low upright component with a minus-plus or inverted U wave (fig. 5, 8d) resembles an “ascending W” in which the second trough of the letter does not descend as low as the first.

With simply inverted T waves, the possibilities for superposition are principally the same as with upright T waves, but in the form of a mirror image. Diphasic fusion waves of the recumbent S type (fig. 5, 9a, 9c, 12c) or broad monophasic negative fusion waves (fig. 5, 9–11d) can appear in this way. The minus-plus patterns resemble the types shown in figure 5, 7a, 7c, 8a and 8c, and appear under the same clinical conditions as in the case of diphasic T waves. Uniform diphasic waves may appear when inverted T waves are followed by upright U waves and the terminal branch of T shows the same inclination as the initial branch of
U without overlapping. But this condition seems not to be a frequent one. The uniform downward patterns are mirror images of the patterns produced by upright T waves. They may be found in similar conditions. The type with the very important predominance of a negative T wave in a case of pulmonary embolism was recognized in 1952 and presented with Ramer4 (fig. 6). This characteristic sign of acute cor pulmonale is confined to precordial

**Fig. 6.** Deeply inverted U waves causing TU-fusion waves in leads V1 through V3 of a case of pulmonary embolism.

**Fig. 7.** TU-fusion waves in a case of hypopotassemia. The expected Q-T duration for the heart rate and the actual Q-T duration (Q-T eff.), as well as the Q-U duration, are indicated in the upper right hand corner.
leads V_1 through V_4, and is usually best seen in leads V_2 and V_3.

Uniform diphasic waves consisting of T and U waves can appear also without any fusion or overlapping of these waves, if the terminal branch of the T wave is followed immediately by a monophasic U wave of opposite polarity. Such waves may be called "continuation waves."

To recognize TU fusion waves, as well as uniform diphasic TU continuation waves, the following attributes are valuable. 1. Agreement of the interval from Q to the end of the wave with the predicted duration of Q-U interval based on the heart rate. 2. The presence, in other complexes of the same lead, of a notch at the site where the T-U junction is expected according to the heart rate. 3. The recognition of a separation of T and U in other simultaneously recorded leads, at a point where these waves are expected in relation to the heart rate. 4. Observation of separated T and U waves in the same lead in previous or subsequent tracings. 5. Demonstration of the extension of the wave in question beyond the second heart sound by more than 0.04 sec.; if this sound is premature, then beyond the theoretically predicted second sound, according to the heart rate. 6. Demonstration of the components of TU-fusion waves, as Surawicz and Lepeschkin have suggested, by shortening the Q-T interval with calcium chloride or by altering the size of the T and U waves with potassium.

Although the relationship between the second heart sound and the U wave requires further study, it is obvious that the U wave closely follows this sound. In the light of this relationship the circumstances that can produce TU-fusion waves become predictable. These include all factors that lengthen the Q-T interval more than the interval between Q and the second heart sound and that amplify the U waves. These results are brought about by acute overloading of the ventricles and also, as Surawicz and Lepeschkin have pointed out, by the combination of hypocalcemia with hypopotassemia. Another important factor is the so-called energeto-dynamic heart failure studied and developed by Hegglin. Because the second heart sound in this condition appears earlier than predicted in relation of the heart rate or, in other words, because hemodynamic systole is shortened, the U wave also begins earlier. This favors the merging of U and T waves. It must be pointed out, that in this condition prematurity of the second heart sound is essential and the so-called dissociation between the Q-T interval and hemodynamic systole is either an illusion or much smaller than it appears to be. What has been measured in these cases equals actually the Q-U interval.

The presumed close relationship between the second heart sound and the U wave throws some light on the origin of the latter. This relationship fits very well into the concept that the U wave is related to the distention of the ventricular walls during early diastole; however, it does not furnish absolute proof of this concept.

The concepts developed in this paper are essentially in agreement with the statements published by Lepeschkin and Surawicz. In view of these findings it may be necessary for many of us to correct some interpretations of electrocardiograms published in previous papers. To make these concepts familiar to all who may have to deal with the U wave in the future is one of the purposes of this symposium.

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