

Case Report

The U wave and papillary muscle variants: revisiting an old association

JAMES KER

Summary

One of the earliest hypotheses on the origin of the U wave was that these waves represent repolarisation of the papillary muscles and their neighboring structures. Various U-wave and TU-segment abnormalities have been described and ascribed to certain cardiac pathological conditions.

In this case report it is hypothesised that prominent U waves in the inferior leads are caused by an accessory papillary muscle. Any possible long-term consequences are not known.

Department of Physiology, University of Pretoria, South Africa

JAMES KER, MRCP, PhD, FESC, FACC, jker@wol.co.za

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The U wave, first described by Einthoven,¹ is still an electrocardiographic deflection of enigmatic origin. It is an additional low-amplitude wave that is sometimes visible after the T wave. The U wave is usually less than 0.1 mV in amplitude and normally has the same polarity as the preceding T wave. It is usually best seen in the mid-precordial leads at slow heart rates.

The electrophysiological basis for U-wave generation is not certain. Any one (or more) of the following may be the cause of the U wave: repolarisation of the papillary muscles,² repolarisation of the Purkinje fibers outlasting that of the contracting myocardium,³ prolonged repolarisation in cells of the mid-myocardium – the ‘M cells’,⁴ or after-potentials, caused by mechanical forces in the ventricular wall with termination of mechanical systole – the ‘mechano-electrical feedback’ hypothesis.⁵

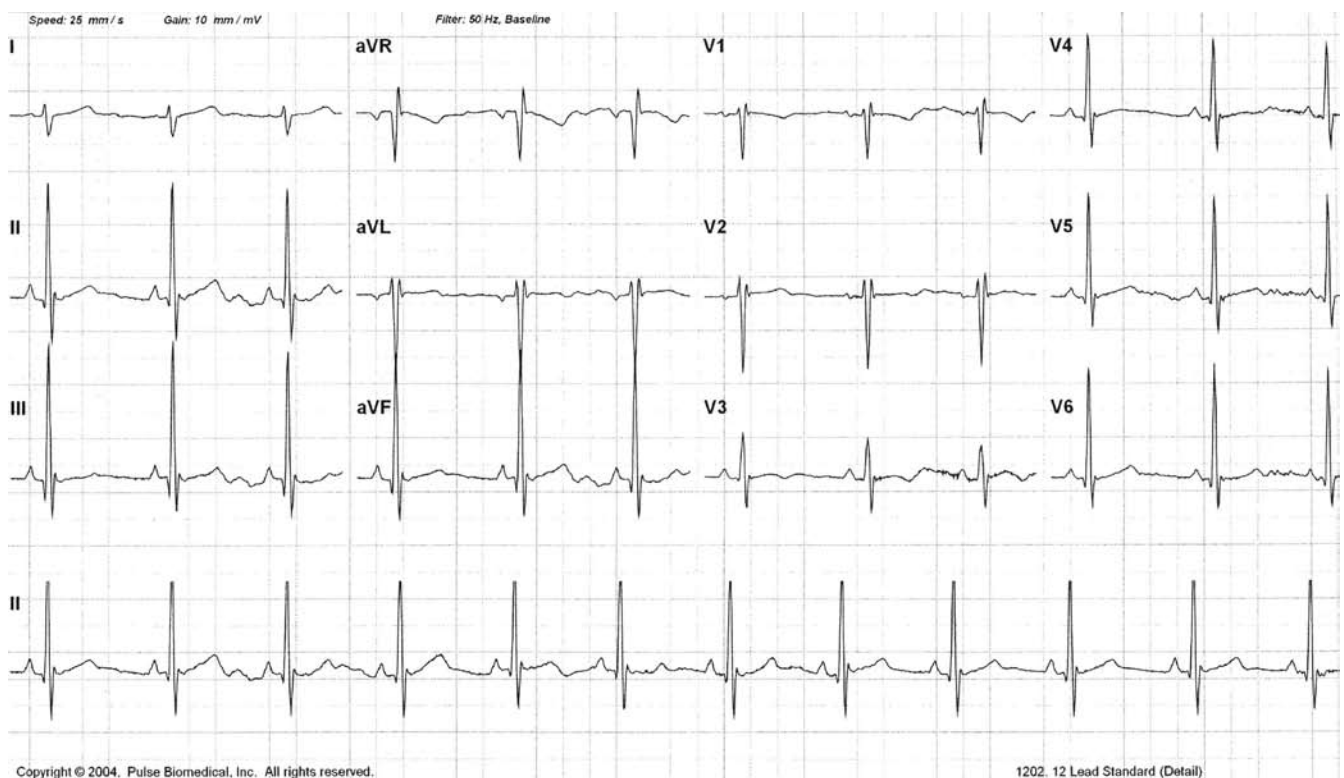


Fig. 1. Standard 12-lead electrocardiogram. Note the prominent U waves in leads II, III and aVF. Interestingly, they are absent in the preceding beat with a longer R-R interval. A possible explanation for this phenomenon is the longer diastolic time with resultant increase in end-diastolic volume, leading to a greater degree of papillary muscle stretch with a possible augmentation of the U wave of the following beat.

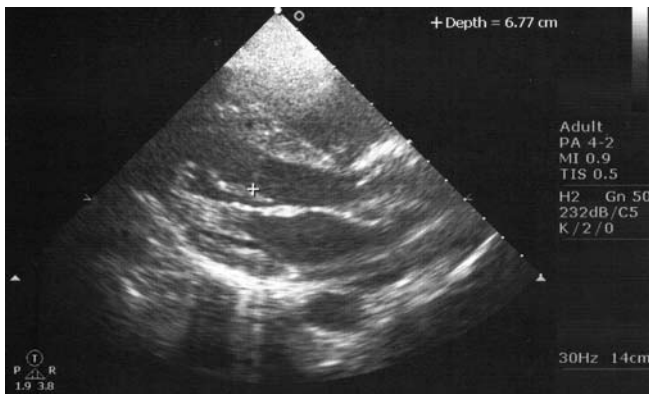


Fig. 2. The parasternal, long-axis view from a transthoracic echocardiogram; + marks the accessory papillary muscle. This accessory papillary muscle led to no obvious adverse sequelae. The mitral valve functioned normally and no intra-ventricular pressure gradient was present.

Recently, various primary abnormalities of the ventricular papillary muscles have been described.⁶⁻¹⁰ These abnormalities include: haemangiomas,⁶ solitary hypertrophy,⁷ endodermal heterotopia (inclusion cysts),⁸ papillary fibroelastoma,⁹ and an octopus-shaped papillary muscle causing mid-ventricular obstruction.¹⁰

Ker⁷ described a case of ST elevation and QRS notching with a prominent U wave in lead V4 in a patient with solitary papillary muscle hypertrophy. Although the papillary muscle origin of the U wave is only one of many theories (each one of them plausible but not conclusively proven), the current era in cardiology where echocardiography readily identifies variants (not necessarily pathological) of intra-ventricular structures is ideal for clinicians to try and correlate these variants with electrocardiographic changes.

Case report

A healthy 15-year-old girl was referred for a cardiovascular evaluation because of very prominent U waves in the inferior leads of a 12-lead electrocardiogram (Fig. 1). She had no previous medical or surgical problems, clinical examination was normal and a comprehensive biochemical evaluation revealed no abnormalities.

Transthoracic echocardiography revealed a prominent accessory papillary muscle (Fig. 2). This accessory papillary muscle had no apparent functional consequences; the mitral valve functioned normally and no intra-ventricular pressure gradient was present.

Discussion

The U wave is usually the most prominent in leads V2 or V3.¹¹ Two observations in this case merit discussion. Firstly, the prominent U waves are noted in the inferior leads (II, III, aVF). It is therefore possible (and proposed) that in cases of prominent U

waves, which might be caused by accessory papillary muscles, that these U waves will be noted only in the inferior leads. Therefore, it is proposed that the location of visible U waves may be a clue to underlying papillary muscle variants.

Secondly, as is clearly visible in Fig. 1, the U waves in leads II, III and aVF are seen in the beat following a beat with a longer R–R interval. It is proposed and hypothesised that this might be due to the following mechanism: the beat preceding the U wave has a longer R–R interval, which will cause a longer diastolic interval, thus leading to a greater left ventricular end-diastolic volume and a consequently larger ejection fraction (Frank-Starling mechanism). This may cause a greater amount of papillary muscle stretch and torsion with a possible effect on the observed U waves in the following beat. Therefore, this case report indirectly supports the mechano-electric feedback hypothesis for these visible U waves and not repolarisation of the papillary muscles.

Lastly, it is hoped that this case report focuses attention on the possible correlation between a growing number of anatomical variants of the papillary muscles and possible variants of the U wave, which might be identified on the standard 12-lead electrocardiogram.

References

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