Editorial

The J wave: why so many contradictions and confusion in interpretation of its diagnostic value?

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There has been a significant interest in recent times in discussing how to properly interpret the diagnostic value of the point J on ECG. At first sight, it might appear to be a rather simple issue, but till now no consensus in its interpretation has been reached. Volume 46 of Journal of Electrocardiology No. 5, September/October 2013 is fully devoted to the above mentioned topics. A lot of publications in various journals and conference reports supply us with a great variety of concepts treating this problem [1-4].

Referring to our studies in the field of the cardiac cycle phase analysis, we would like to suggest our opinion on this issue.

There is a paradox that up to the present the phase-structured cardiac cycle is not understood in a proper manner. After the comprehensive research, we have identified that the J point corresponds to the end of the rapid ejection phase [5,6]. In this connection, a question arises: where is the beginning of this phase? It should correspond to the time of the beginning of the aortic valve opening that is recorded as the onset of a rheogram upslope segment. This issue has not been described in scientific literature, and therefore we have introduced a new point on ECG: it is point L that corresponds to the beginning of the rapid ejection phase. These topics were considered in details in a number of our papers and monographs. According to our concept, the cardiac cycle phase structure appears as follows (Fig. 1)[5].

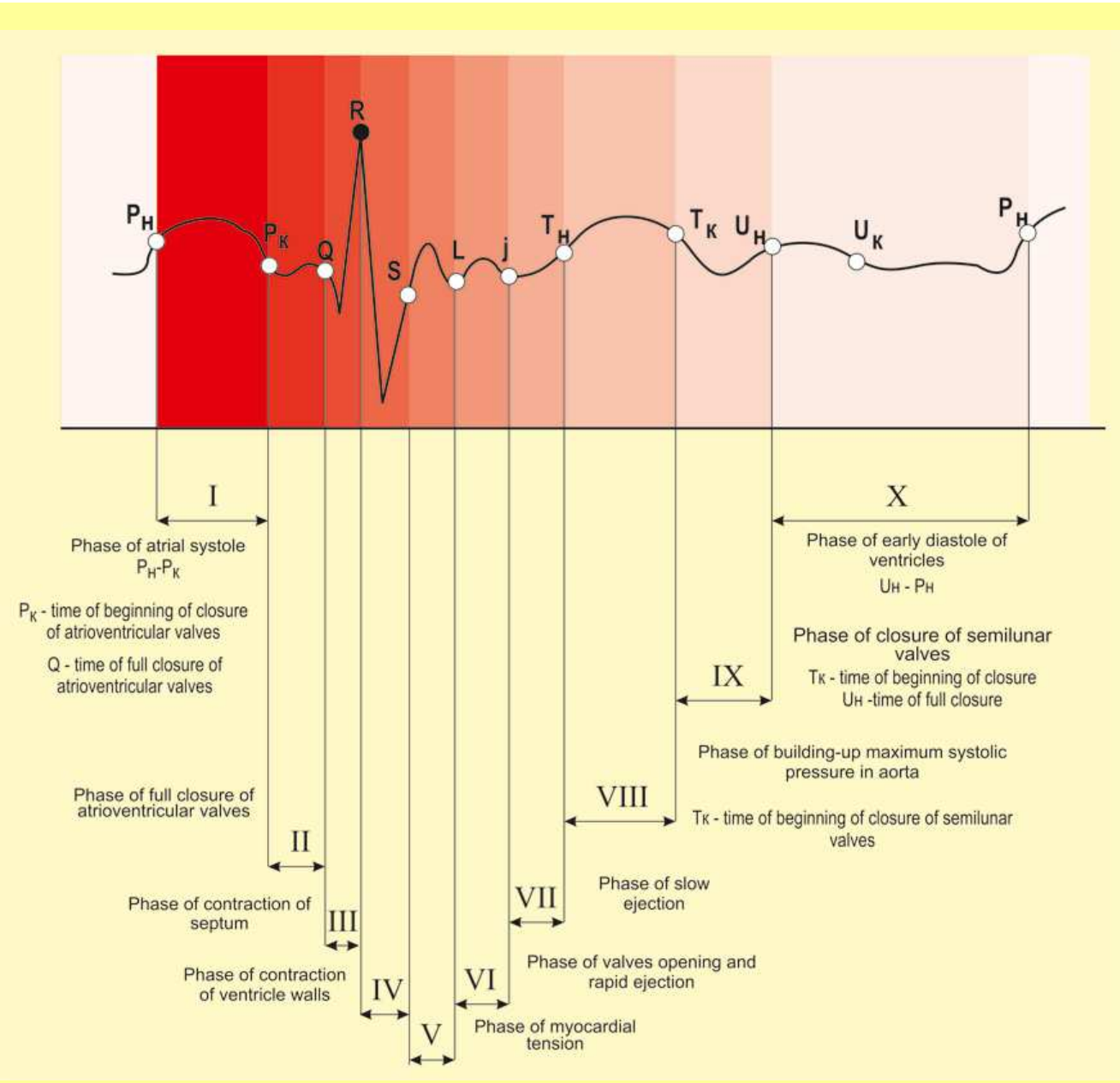

Figure 1. A new concept developed by us: cardiac cycle phase structure.

Firstly, let us note that the above mentioned phase cannot shift and overlap the preloading S-L phase. It is just the matter that seems confusing for many authors and many minds and that makes them think that it is exactly the shifting of the phase that is the pathological onset of the early repolarization (ER). The repolarization can be initiated if and only if the blood pressure at the AV node is released, i.e., in case of depressurization at the AV node. Therefore, it is always the rule without exception that the repolarization begins with the end of the T wave [7].

Answering the question about the diagnostic value of the ECG segment in the phase being studied, it should be noted that the L-j phase reflects the amplitude of the third repeated septal and myocardial muscle contraction in the background of the general tension of all muscles that occurs in the S-L phase. Actually, we deal with the third QRS complex within the same cardiac cycle. This case has been already detailed by us in our papers [5].

Let us consider some real records to better illustrate our approaches. Fig. 2 below exhibits two synchronously recorded curves: an ECG and a Rheo of the ascending aorta, when changing the muscle diaphragm position caused by a mechanical force generated by an expansion in the abdominal volume. The specific feature of these curves is that the first part of the recordings corresponds to the starting of the abdominal volumetric expansion that leads to shifting of the heart electrical axis, and the second portion of the records reflects traveling of the heart electrical axis without such volumetric expansion. Fig.3 shows the respective responsiveness in detail.

Figure 2. ECG and Rheo recordings of the ascending aorta.

Cardiac cycles are numbered below from 9 to 31. Both the shift of the heart's electrical axis and the associated alteration in the S wave amplitude from its normal value up to its full disappearing are caused by an abdominal expansion in the volume that reduces the pressure on the diaphragm and contributes to the S wave formation.

Figure 3a. Cycles 11-15.

Figure 3b. Cycles 27-31.

Fig. 3. Detailed representation of the various parts of ECG and Rheo shown in Fig.2. Cardiac cycles from 11 to 15 (a) and from 27 to 31 (b).

The measured basic parameters of hemodynamics given in Fig.4 below unambiguously indicate the stability of all processes in each cardiac cycle regardless of the ECG shape. Such is the case for the stroke volume (SV) that remains stable and reaches 55,1 ml in cycles from 11 to 15. When assessing the stroke volume in the other cycles, it is evident that this parameter is not significantly larger in cycles 27-31 and accounts for 55,3 ml. The same is the case with the rest of the phase volumes. This proves the fact that the j point does not change its position, and therefore there are no grounds to speak about the early polarization.

	SV	MV	PV1	PV2	PV3	PV4	PV5	HR
Средн.	53.7	4.3	31.8	22.0	31.9	21.8	8.3	79.4
1	55.1	4.6	31.2	23.9	32.7	22.4	8.4	83.5
2	47.2	3.9	26.3	20.9	28.0	19.2	7.5	82.6
3	55.1	4.5	33.0	22.1	32.7	22.4	8.4	81.7
4	55.1	4.4	33.1	22.0	32.7	22.4	8.4	80.3
5	55.1	4.4	32.9	22.3	32.7	22.4	8.4	79.5
6	55.3	4.4	32.5	22.8	32.8	22.5	8.5	79.9
7	55.3	4.4	32.3	23.0	32.8	22.5	8.5	80.3
8	47.2	3.8	27.2	20.0	28.0	19.2	7.5	80.8
9	55.1	4.4	32.0	23.1	32.7	22.4	8.4	79.9
10	55.3	4.4	32.3	23.0	32.8	22.5	8.5	80.3
11	55.1	4.5	32.3	22.8	32.7	22.4	8.4	81.7
12	55.1	4.5	31.5	23.6	32.7	22.4	8.4	80.8
13	55.1	4.4	31.8	23.3	32.7	22.4	8.4	80.3
14	55.3	4.4	32.3	23.0	32.8	22.5	8.5	80.3
15	55.1	4.5	32.6	22.5	32.7	22.4	8.4	81.2
16	55.1	4.4	33.2	22.0	32.7	22.4	8.4	80.3
17	55.6	4.3	33.9	21.7	33.0	22.6	8.6	77.4
18	55.6	4.4	32.8	22.9	33.0	22.6	8.6	78.2
19	55.6	4.5	31.9	23.7	33.0	22.6	8.6	80.8
20	54.9	4.5	30.6	24.3	32.6	22.3	8.3	82.6
21	47.6	3.8	27.1	20.5	28.2	19.4	7.7	79.5
22	55.1	4.4	31.1	24.0	32.7	22.4	8.4	80.3
23	55.6	4.4	31.4	24.3	33.0	22.6	8.6	79.5
24	54.9	4.4	31.7	23.3	32.6	22.3	8.3	80.8
25	56.0	4.4	32.5	23.5	33.2	22.8	8.7	78.6
26	55.1	4.5	31.0	24.2	32.7	22.4	8.4	81.7
27	55.3	4.4	31.5	23.8	32.8	22.5	8.5	79.5
28	55.6	4.4	33.0	22.7	33.0	22.6	8.6	79.0
29	55.3	4.4	31.4	23.8	32.8	22.5	8.5	79.5
30	55.3	4.3	32.0	23.3	32.8	22.5	8.5	78.6
31	55.3	4.4	32.5	22.7	32.8	22.5	8.5	79.9
32	55.3	4.4	31.6	23.6	32.8	22.5	8.5	80.3

Figure 4. Hemodynamic parameters derived from the ECG shown in fig. 2. The basic hemodynamic parameters SV- stroke volume, MV-minute volume, PV1, PV2, PV3, PV4, PV5 and HR are indicated herein.

Another example is an ECG curve given in Fig.5 below. It was recorded in a patient with a lower body mass index. The so-called j-wave is marked clearer on this ECG. It is obvious that the S-L and L-j phases on the resting ECG demonstrate no changes in their durations. A more detailed examination of 2 cardiac cycles shows that we deal with a variation in the ECG shapes. Fig.6 below illustrates that a sudden change in the stroke blood volume (SV) is recorded during the sharp ECG shape alteration that corresponds to the time of the j wave disappearance. The SV parameter in cycle 13 is 45,3 ml, while the same parameter accounts for 38 ml in the previous and the next cycle. It is only evidence of the responsiveness of the cardiac cycle phases which are included as variables in the hemodynamic equations by Poyedintsev- Voronova used for the blood phase volume calculation [5-10].

Figure 5 The cyclically changing j wave on the curve for vertical patient body position. No symptoms of disease are reported. No patient's complaints are available.

	SV	MV	PV1	PV2	PV3	PV4	PV5	HR
Средн.	40.0	2.9	26.6	13.4	23.8	16.3	6.3	72.8
1	37.8	2.8	24.3	13.5	22.4	15.4	6.0	74.4
2	38.2	2.8	25.5	12.7	22.7	15.6	6.1	72.2
3	38.0	2.8	25.0	13.1	22.6	15.5	6.0	72.9
4	45.1	3.2	30.2	14.9	26.7	18.3	6.8	71.1
5	38.2	2.6	25.6	12.6	22.7	15.6	6.1	69.2
6	45.1	3.2	29.3	15.8	26.7	18.3	6.8	71.5
7	38.0	2.7	25.8	12.2	22.6	15.5	6.0	70.8
8	37.8	2.7	25.0	12.8	22.4	15.4	6.0	70.1
9	38.2	2.6	26.0	12.2	22.7	15.6	6.1	68.8
10	38.0	2.7	24.9	13.1	22.6	15.5	6.0	70.5
11	38.0	2.7	25.3	12.8	22.6	15.5	6.0	71.8
12	37.8	2.7	25.4	12.4	22.4	15.4	6.0	72.5
13	45.3	3.2	32.0	13.3	26.9	18.4	6.9	70.5
14	38.0	2.7	25.8	12.2	22.6	15.5	6.0	70.8
15	45.3	3.1	30.7	14.6	26.9	18.4	6.9	69.2
16	38.0	2.7	25.7	12.3	22.6	15.5	6.0	71.5
17	45.3	3.2	31.7	13.6	26.9	18.4	6.9	71.5
18	38.0	2.7	25.5	12.5	22.6	15.5	6.0	70.8
19	38.2	2.7	26.3	11.9	22.7	15.6	6.1	70.5

Figure 6. Hemodynamic parameters derived from the ECG given in fig. 5.

In this case, the following common feature should be highlighted: the j wave appears only in the event that a significant decrease in the QRS amplitude in general is observed. Nowadays it is still impossible to exactly define what processes may cause a drop in the potential on an ECG. In our research, we have detected sudden sharp changes in the electrical potential in one cycle in a patient due to his emotional stress. But today there are still no definitive answers to this question. Another issue of significance is to investigate how a high level protein input can influence the electrical potential of the heart muscle. We believe we are capable of solving all these issues within the nearest future.

In summary it may be said the following:

1. The recorded j wave cannot be considered as a criterion of pathological changes in the heart performance.

2. Hereby we confirm that we fully accept the statements by Prof. Victor Froelicher on the necessity to develop more profound methods for further investigations of the j wave phenomenon in

accordance with the considerations given by us herein that should be supported by the utilization of the cardiac cycle phase analysis.

3. It should be noted that we share Prof. Macfarlane's opinion on the complexity of computerization in studying the j wave phenomenon.

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