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Supplement-1 CARDIOLOGY

Difficult Interpretation of ECG: Small Clues May Make the Difference. The Role of the P Wave

Alessandro Maolo, Daniele Contadini

Case Report

A 24-year-old woman suspected of scleroderma was referred to the clinic of internal medicine for clinical evaluation.

The patient presented with asthenia and mild dyspnea for moderate physical activity. She reported sleeping with just one pillow, and she had no episodes of nocturnal dyspnea. She had no syncope or presyncopal symptoms.

Physical Examination

- *General:* no fatigue, no acute distress, alert, awake, and oriented; well developed and well nourished
- *Neck:* supple, no jugular venous distention, no lymphadenopathy, and no carotid bruit
- *Cardiovascular:* regular rate and rhythm, S1 normal, paradoxical splitting of the S2, and reduction of intensity at the apex. Mild systolic crescendo-decrescendo murmur at the second right intercostal space with no radiation, early low-pitched diastolic decrescendo murmur, and no pansystolic plateau murmur attributable to ventricular septal defect. Point of maximal intensity displaced to the center of the chest. No hepatojugular reflux and capillary refill less than 2s.
- *Lungs:* rales at auscultation at the bases bilaterally, no rhonchi or wheezes, no egophony, no alterations in tactile fremitus, and normal percussion sounds
- *Abdomen:* mild overweight, no pulsatile masses, normal bowel sounds in all four quadrants, no high-pitched or tinkling sounds, resonant to percussion, soft, nondistended/nontender, no rebound or guarding, no costovertebral angle tenderness, and no hepatosplenomegaly
- *Extremities:* no cyanosis or clubbing and no peripheral edema

A. Maolo, D. Contadini(✉)
Clinica di Cardiologia e Aritmologia, Università Politecnica delle Marche, Via Conca 71, Ancona 60126, Italy
e-mail: alessandro.maolo@alice.it; daniele.contadini@gmail.com

Electrocardiogram

A routine ECG was performed and is described as follows (Fig. 1a,b):

Regular RR interval with a heart rate of 75 bpm.

P wave is apparently increased in amplitude and duration; however, watching carefully, we can notice that the amplitude of the P wave doesn't reach a pathologic value. In fact the maximum amplitude is 0.20 mV in II lead. Also the duration is mildly-prolonged, with the maximum P wave duration of 120 milliseconds (ms) reached in II lead. P wave morphology can be considered pathological: if we pay attention on the P wave in II, III, and aVF leads, we can notice that the first part is high and narrow and it's followed by a second part that is smaller and larger than the first one. This is a likely sign of biatrial enlargement. P wave axis is 90°, being negative in aVR and aVL leads, biphasic in I lead, and positive in II, II, and aVF leads. A negative P wave in aVL is something to think about.

PQ interval is normal (160 ms).

QRS complex is clearly enlarged (160 ms) with a normal amplitude. However, it decreases progressively from V1 to V6. Looking at precordial leads, the morphology is typical for right bundle branch block. In the limb leads, the

QRS complex has a strange morphology. A deep S wave, typical for RBBB, in I and aVL leads is missing, and the intraventricular and interventricular conduction disturbance seems to be more complex than in a simple RBBB. Also the "rS" morphology in aVR lead and the "rSR" in aVL leads are not typical for RBBB. Axis is indeterminable.

Repolarization is not abnormal because this is secondary to the conduction disturbance. The QT interval measures 400 ms and the QTc 447 ms.

Conclusions

ECG shows sinus rhythm, normal heart rate, right and left atrial enlargement, normal atrioventricular conduction, right bundle branch block, and secondary repolarization's abnormalities.

Looking at a previous chest X-ray, something unexpected was found (Fig. 2).

The patient had a situs viscerum inversus with dextrocardia (please note the cardiac apex and the gastric bubble placed on the right side). Moreover, the patient had tetralogy of Fallot and underwent a surgical correction when she was a child.

Taking a new look to the previous ECG, we can now relate some findings with the anatomic abnormalities.

First of all, the axis of the P wave is not normal. The notch on the apex of the P wave is not a sign of an atrial enlargement. In fact in young people, the P wave duration can be normal up to 130 ms. The notch is a sign of interatrial conduction disturbance compatible also with the previous surgical intervention.

The previously-diagnosed RBBB is a left bundle branch block. In dextrocardia, the morphological left bundle branch is placed on the right, and its conduction disturbance could be due to the interventricular septal defect that is always present in tetralogy of Fallot. The other intraventricular and interventricular conduction disturbances could be linked to the previous surgical intervention.

The atypical morphology of QRS complex in aVR and aVL leads can be related to dextrocardia too. In fact, in RBBB, "rSR" and "qR" morphologies in aVR lead and a deep or large S wave in aVL lead are common findings, and in this case, the morphologies are inverted between the two leads.

By comparing the previous ECG with the following, it is visible that in the typical RBBB (ECG from another patient below), the QRS morphologies in aVR and aVL are switched (Fig. 3a,b).

A sign that could suggest the presence of dextrocardia is the decreasing amplitude of the QRS complex from V1 to V6 together with the negative P wave in aVL. This happens because the electrodes are placed on the left part on the chest and the electric signal from the heart is progressively going farther from the precordial leads, in particular from the left ones.

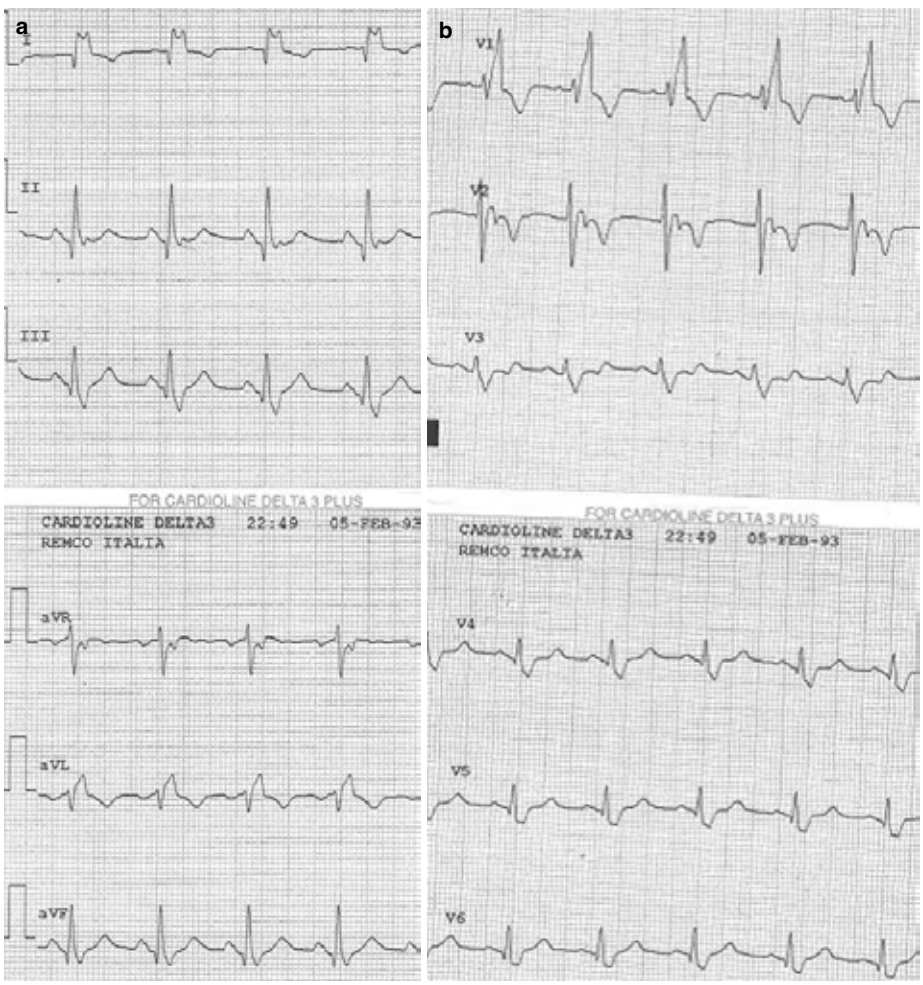

Fig. 1: (**a**, **b**) Standard 12-lead ECG at rest. See the chapter for the description.

The Normal P Wave

P wave comes from the electrocardiogram sign of atrial depolarization. The impulse starts from the sinoatrial (SA) node and spreads to the right atrium first and then through the interatrial septum to the left atrium.

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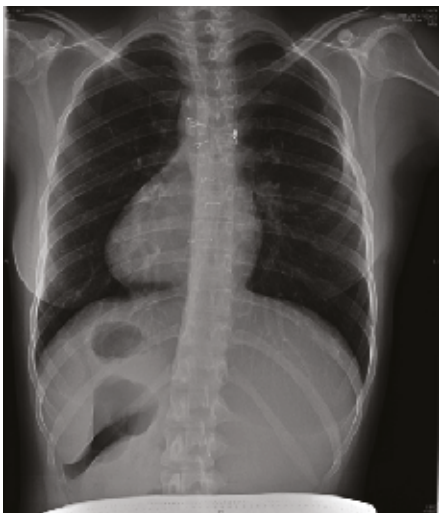

Fig. 2: Chest X-ray. Please notice the cardiac apex and the gastric bubble placed to the *right side*.

Its normal duration varies from 70 to 140 ms even in normal subjects [1]. A P wave lasting more than 110 ms can be considered abnormal.

The P wave axis is the result of the electrical activation of the right atrium (directed anteriorly first and then posteriorly when involving the inferior wall) and of the left atrium (directed posteriorly). Therefore, on the limb leads, the normal P wave range is 0° to +75°. That's why the normal P wave is positive in I and II leads and always negative in aVR lead. Moreover, also in the precordial leads from V3 to V6, the P wave is generally positive. Conversely in V1 and V2, normal P wave is often diphasic and sometimes even negative.

Talking about the normal amplitude of the P wave, we have to say that it's affected by many factors such as the distance of electrodes from the heart and the level of atrial fibrosis. In the limb leads, a P wave inferior or equal to 0.25 mV or 25% of the following R wave is considered as normal amplitude. In the precordial leads, the normal amplitude is a bit lower and doesn't exceed 0.15 mV [1].

Interatrial Conduction Delay

Atrial depolarization begins in the sinus node; from here, the electrical impulse goes through the internodal fascicles (fibers that connect sinus with the AV node). There are three bundles: the Bachman fascicle (anterior), the Thorel fascicle (in an intermediate position), and the Wenckebach fascicle (posterior). The last one conducts the impulse from the right to the left atrium too. So, the atria activation begins from the right atrium and then reaches the left one. The normal duration of P wave is from 50 to 80 ms. A P wave duration superior to 110 ms is pathological and related to interatrial conduction delay. This condition is caused by a complete or partial interruption of the Bachman fascicle or other connecting atria fibers. Usually the interatrial conduction delay is associated with a modification of the axis of the terminal part of P wave. It is more negative (from −30° to −60°) with a caudal-cranial activation of the left atrium. This pattern of activation is visible as a biphasic P wave (positive-negative, with terminal negative component evident in inferior leads) lasting more than or equal to 100 ms. The interatrial conduction delay is highly specific of left atrium enlargement [1, 2].

P Wave Other Than That from the Sinus Node

The P wave is defined not to be sinusal when it originates from an ectopic focus

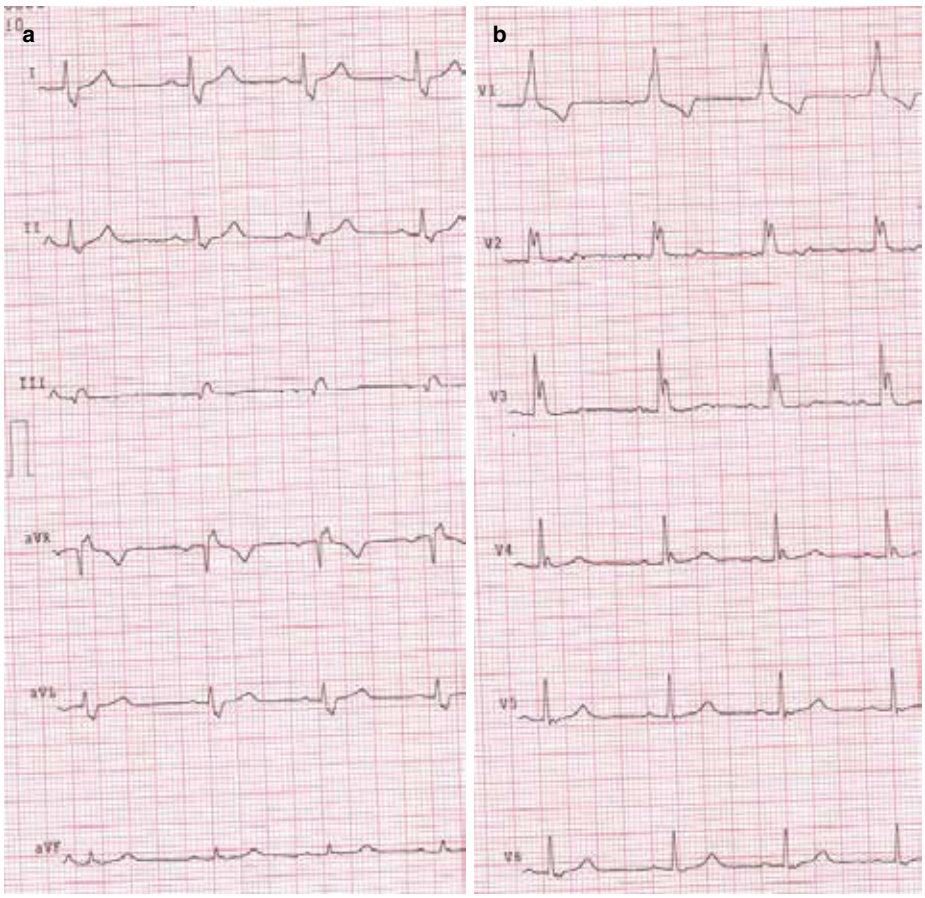

Fig. 3: (**a**, **b**) Standard 12-lead ECG at rest from another patient having a situs solitus and an RBBB. Please compare with the ECG in Fig. 1.

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that can be located in variable parts of the atria. A non-sinus P wave can be present as a premature atrial impulse (isolated or in pairs), as an ectopic atrial tachycardia (heart rate ≥ 100 bpm), or as an ectopic atrial rhythm (heart rate < 100 bpm).

In all these cases, the P wave has a different morphology and axis compared with the sinusal P wave. Analyzing its characteristics on the surface ECG can help us to identify the location of the ectopic focus, but it isn't always accurate. This variability depends on the possible presence of ectopic focus close to the sinus node and therefore originating P wave very similar to that present in normal sinus rhythm. Moreover, a single ectopic focus can cause P waves of different morphology related to the presence of intra-atrial or interatrial conduction disturbances. Likewise, ectopic P waves originating from different foci can have similar or the same morphology [1, 3].

Despite this wide variability, some ectopic foci are identifiable with fairly good precision. For example, if the ectopic impulse originates from a focus close to the AV node, a negative P wave can be visible in II, III, and aVF leads, while it is positive in aVL and aVR leads. On the contrary, if the ectopic focus is located in the left atrium, the P wave is negative in aVL and positive in aVR (similarly to the P wave in dextrocardia), while in the inferior leads, it can be positive or negative depending on the part of the left atrium in which the focus is placed.

Right Atrial Enlargement

Right atrial enlargement (RAE) is a common consequence of various cardiopulmonary diseases. As we already said, the atrial depolarization firstly involves the right atrium, so it is easy to understand that RAE alterations interest the initial portion of P wave with voltage increase but without modification in terms of P wave duration (Fig. 4a, b). Some typical P wave modifications can help to recognize RAE: P wave (or initial positive P wave deflection) > 0.15 mV (more specific ≥ 0.20 mV) in V1 and V2 leads; an initial positive part > 40 ms in V1 when P wave is biphasic in this lead; high and sharp P wave in inferior leads (II, III, aVF) ≥ 0.25 mV; and P wave axis ≥ +75° (it can reach +90° too). The last modification is very strictly associated with COPD. Sometimes the RAE correlated P wave modifications can be associated with QRS modification as increased voltages of right ventricle electrical vectors or delayed right ventricle depolarization [1, 2].

The P pulmonale pattern isn't strictly correlated with the increase of right atrial volume, and it can be present even in healthy subjects. In these cases, usually a sympathetic stimulation, resulting in a stronger and synchronous atrial contraction, or the vertical position of the heart can explain abnormalities in P wave axis and morphology similar to those present in the P pulmonale pattern.

P Pulmonale

A P wave is defined "pulmonale" when it appears as a tall and peaked P wave in the inferior limb leads (II, III, and aVF) and consequently having an axis superior to 70° [1]. This finding on the ECG has different correlations, but it's usually associated with pulmonary hypertension. The P pulmonale is most frequently associated with congenital heart disease such as tetralogy of Fallot, pulmonic stenosis, tricuspid atresia, or interatrial septum defects and interventricular septal defects in the presence of Eisenmenger syndrome. Furthermore, in patients with chronic cor pulmonale, this ECG pattern has an incidence of about 20 % [1]. As expected, the P pulmonale is a marker of chronic lung disease, but it is mostly associated with COPD and then pulmonary fibrosis. Moreover, severe COPD is responsible for alterations of the P wave axis more than its morphology. This axis deviation over 70° is due to the hyperinflation of lungs, always present in these kinds of patients.

Despite all, the P pulmonale pattern isn't strictly correlated with the increase of right atrial volume, and it can be present even in healthy subjects. In these cases, usually a sympathetic stimulation, resulting in a stronger and synchronous atrial contraction, or the vertical position of the heart can explain abnormalities in P wave axis and morphology similar to those present in the P pulmonale pattern [1].

Left Atrial Enlargement

Left atrial enlargement (LAE) is a common consequence of left ventricular or mitral valve diseases. The left atrial depolarization is the last part of atrial activation, so the LAE modifications involve the final component of P wave with duration's increase but without significant voltage alterations (Fig. 4a, c). Some typical P wave modifications can help to recognize LAE: P wave duration more than or equal to 120 ms in limb leads; left deviation of P wave axis more negative than +30°; and biphasic P wave

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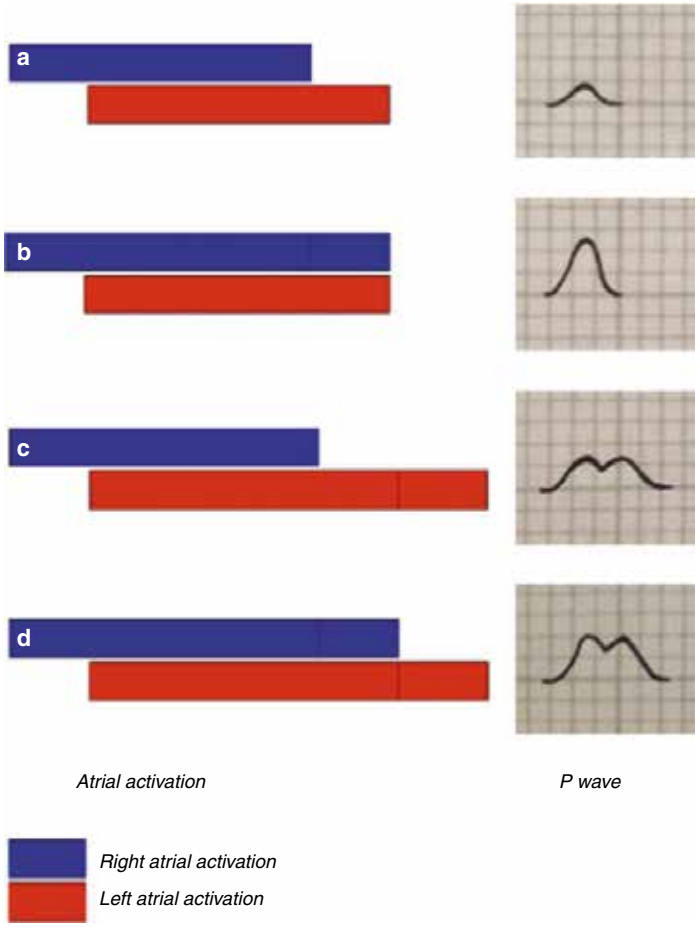

Fig. 4: Atrial activation and P wave morphology in normal atria (**a**), in right (**b**), left (**c**) and biatrial enlargement (**d**).

in V1 with negative terminal component more than or equal to 40 ms and with a minimum of 1 mV bifid P wave (usually in dII, dI, aVL, V3, V4, V5, or V6), with an interval between the two peaks longer than 40 ms. A more peak-to-peak prolongation could be the sign of a mitral disease, often a severe stenosis. This pattern is also defined as P mitrale [1, 2].

Biatrial Enlargement

The P wave abnormality during biatrial enlargement can be simply considered as the summary of the RAE and LAE alterations (Fig. 4a, d). ECG findings could be a P wave duration more than or equal to 120 ms associated with an amplitude more than or equal to 2.5 mV; a diphasic P wave (positive-negative) in V1 with the positive component of at least 1.5 mV and a negative component longer than 40 ms with an amplitude of 1 mV or more; and the presence of notched P wave in left precordial leads and high P wave in right precordial leads [1, 2].

P Wave and Arrhythmias

Some P wave abnormalities during sinus rhythm can be useful as atrial fibrillation predictor. That concept will be discussed below.

P Wave Duration

A prolonged P wave can be related to echocardiographic findings of left or biatrial enlargement and consequently help us to identify patients with a high risk of developing atrial fibrillation. In a clinical trial of 660 patients, who underwent dual-chamber pacemaker implantation, P wave duration was measured using the standard 12-lead ECG (50 mm/s velocity). A P wave duration superior to 100 ms was identified in a group of patients with a higher risk for developing persistent atrial fibrillation and atrial fibrillation-related hospitalization [4].

The P wave duration analysis can be more accurate using signal-averaged ECG (SAECG). SAECG is a particular ECG technique using cardiac electric signals from many surface electrodes. The values measured with all the leads are averaged to minimize interference and to see even the smallest alterations. A prolonged signal-averaged P wave duration compared to the standard 12-lead ECG was found to be a more precise marker for the development of atrial fibrillation [5].

Other important findings detectable using SAECG are atrial late potentials. Late potentials are very-low amplitude electric signals not visible with the standard 12-lead ECG. The QRS late potentials originally were studied to estimate the ventricular arrhythmia risk. Similarly, the P wave (or atrial) late potential can be useful to stratify the risk for paroxysmal atrial fibrillation. Budeus *et al.* hypothesized that atrial late potentials found on P wave SAECG could have a role in the development of paroxysmal atrial fibrillation [6]. However, the predictive value of atrial late potentials has not been demonstrated univocally yet.

P Wave Dispersion

P wave dispersion is defined as the difference between the maximum and minimum durations of P wave measured on the standard 12-lead ECG. A significant high value of P wave dispersion could be considered as the sign of abnormal

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atrial electrical impulse propagation. In the literature, the most used cutoff value for high P wave dispersion is considered to be 40 ms [7]. This value has been demonstrated to have a sensitivity of 83 % and a specificity of 85 % with a positive predictive value of 89 % in detecting subjects who had a history of paroxysmal lone atrial fibrillation [8]. Calculating the P wave dispersion on the standard 12-lead ECG in 100 patients, it was found to be higher in patients who had a history of paroxysmal atrial fibrillation [8]. Moreover, other studies demonstrated that the P wave dispersion calculated on the standard surface ECG could be an effective predictor of atrial fibrillation development in postoperative period (after CABG) and in patients with hypertension [9, 10].

For a more accurate evaluation of P wave dispersion, SAECG was also used and some more precise markers were studied. In particular, P wave dispersion index (P wave duration standard deviation/P wave duration mean value × 100) calculated in 40 subjects was demonstrated to have an 83 % sensitivity and 81 % specificity values in the identification of patients with a history of paroxysmal atrial fibrillation [11].

The rationale for using P wave dispersion as a predictor of atrial fibrillation is that a high dispersion could be considered the electric sign of nonhomogeneous atrial conduction and consequently may reflect an electrical instability of the atria.

Nevertheless, the techniques used for ECG recording and P wave duration and dispersion measurement were not standardized in the different trials. Finally, we have to consider an interobserver and intraobserver variability that may affect accuracy and reliability of P wave dispersion as a noninvasive predictor of paroxysmal atrial fibrillation.

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