Preexcitation with different patterns in the same patient: which is the mechanism?

Español: Reporte de caso

Hombre de 30 años con historia de síndrome de WPW. ECGs registrados en dos ocasiones diferentes muestra diferentes patrones de pre-excitación. Ambos trazados muestra ritmo sinusal con intervalo PR de 100ms. El ECG-1 muestra onda delta positivas en la pared inferior y en las precordiales con delta negativa en aVL.

El ECG-2 sugiere una posible segunda vía accesoria con onda delta negativa en las derivaciones inferiores y positiva en aVL.

Pregunta:

¿Cual es la mas plausible explicación y por que para esta variación de patrón de pre-excitación mostrada en ambos ECG en un mismo paciente?

English: Case report

30-year-old man with a history of the Wolff-Parkinson-White syndrome. ECGs recorded on two separate occasions showed different patterns of preexcitation.

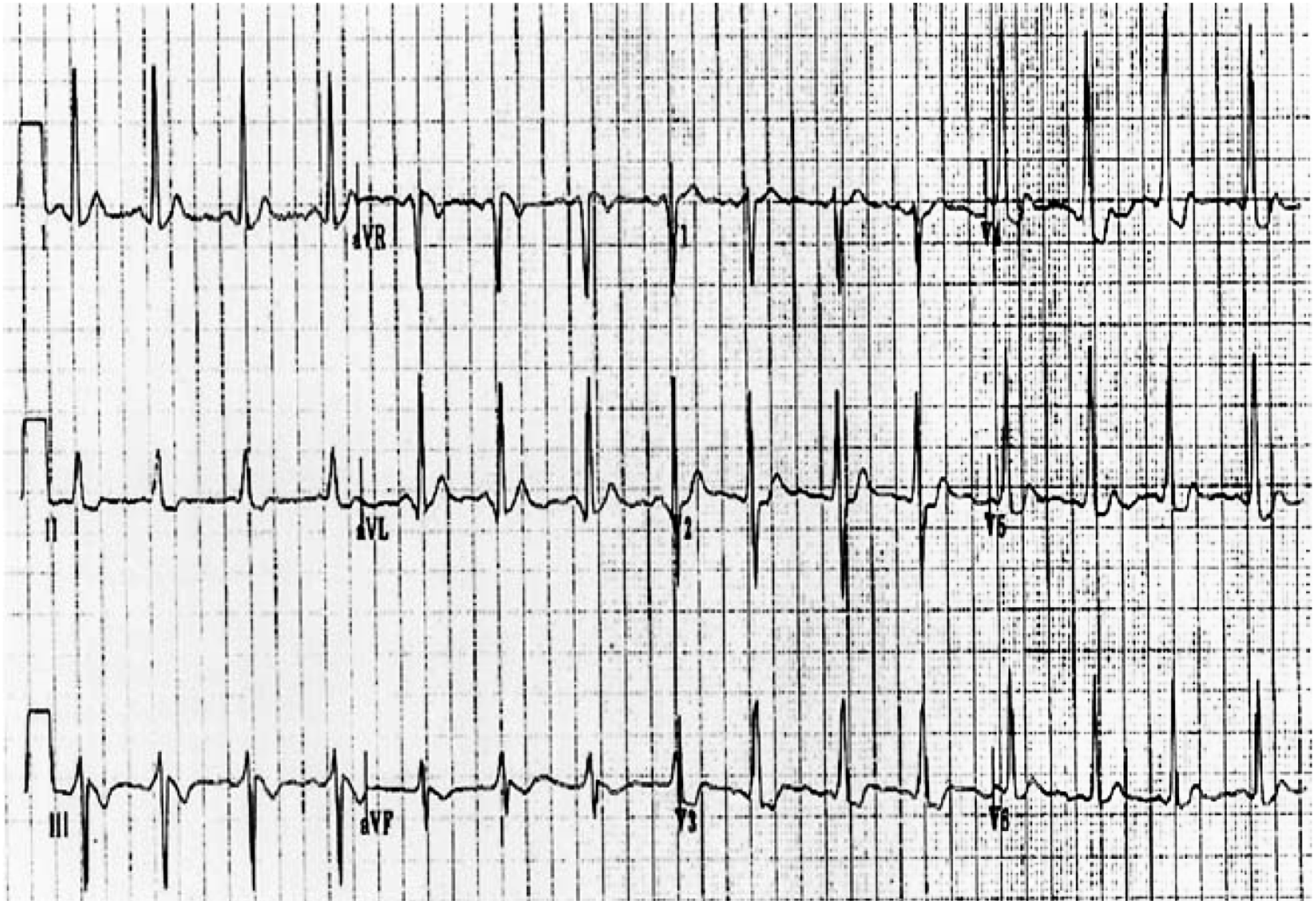
Both ECGs (ECG-1 and ECG-2) show sinus rhythm, short PR interval (110 ms). The ECG-1 has positive delta waves in inferior and precordial leads, and concomitant negative delta wave in aVL.

The ECG-2 suggests a second accessory pathway because delta waves are negative in inferior leads and positive in aVL.

Question:

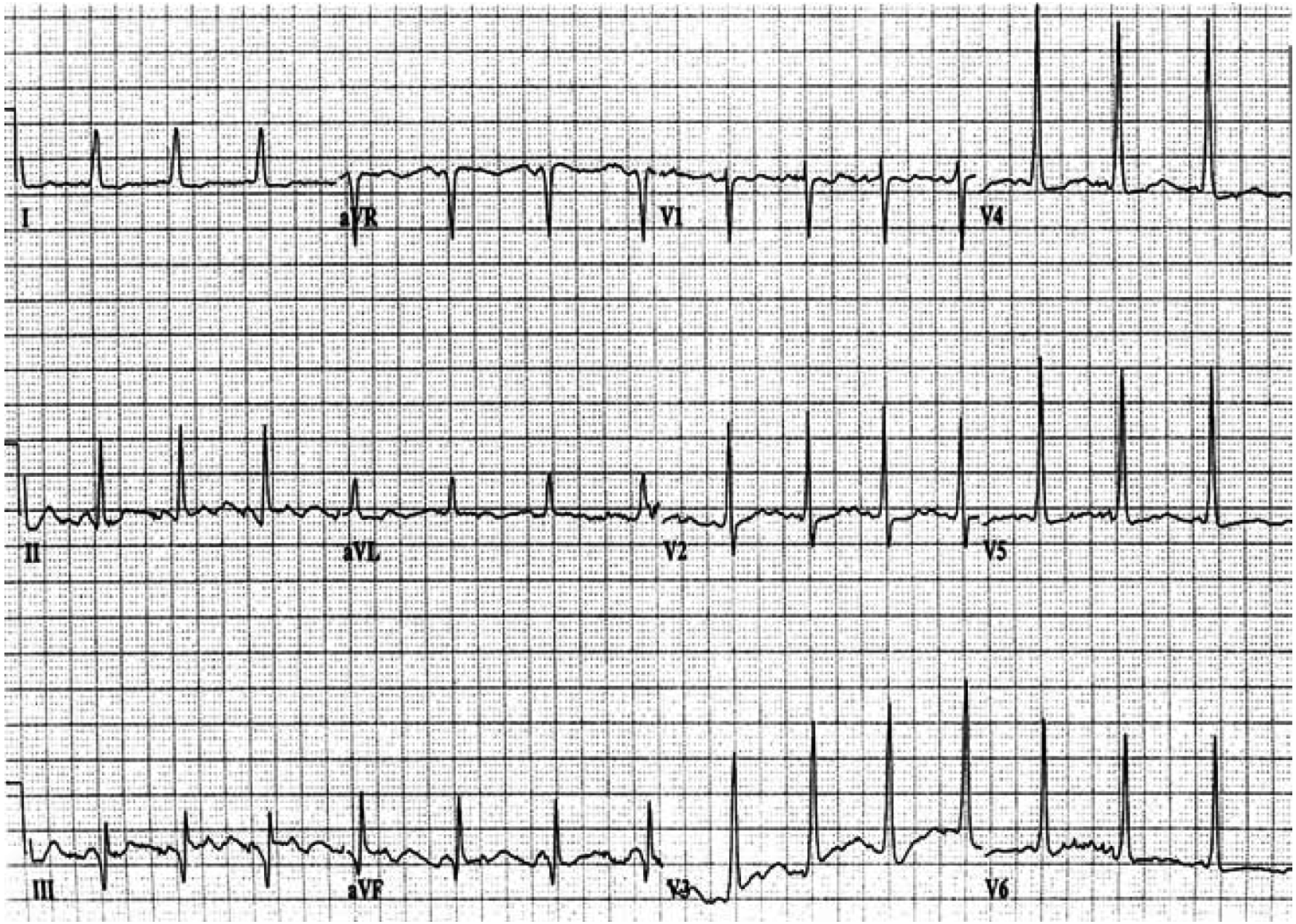
What is the most likely explanation and why for the different patterns between ECG-1 and ECG-2 in the same patient?

ECG-1



ECG demonstrating with positive delta wave in II, III and aVF

ECG-2



ECG demonstrating with negative delta wave in II, III and aVF

Spanish
Hola Andres,

Es bastante frecuente que un paciente tenga dos vías accesorias y que se manifiesten en diferentes ECGs.

Esto es bastante común en la anomalía de Ebstein pro ejemplo.

Saludos,

Mario D. Gonzalez

English
Hello Andres,

It is quite common for a patient to have two accessory pathways and to manifest themselves in different ECGs.

This is quite common in Ebstein's anomaly for example.

Thanks,

Mario D. Gonzalez

Buenas tardes! Además de lo que opina Mario, también son más frecuentes más de una vía en la transposición de grandes arterias. La presencia de estas vías predisponen a la aparición de taquicardias antidrómicas por su menor intervalo PR anterógrado y ocasionan mayor riesgo de muerte súbita. Se asocian con frecuencia a taquicardias por re-entrada Nodal AV.
Me resulta difícil determinar con claridad en el 2° trazado el ritmo sinusal con PR de 100 ms.
Saludos cordiales, esperemos las opiniones de los otros Maestros.
Juan Manzzardo

Good afternoon! In addition to what Mario thinks, more than one way is also more frequent in the transposition of great arteries. The presence of these pathways predisposes to the appearance of antidromic tachycardias due to their lower anterograde PR interval and cause an increased risk of sudden death. They are frequently associated with Nodal VA re-entry tachycardias.
I find it difficult to clearly determine the sinus rhythm with PR of 100 ms in the 2nd course.
Kind regards, let us wait for the opinions of the other Masters.
Juan Manzzardo

English

ECG #1 looks like anteroseptal AP while ECG #2 looks like posteroseptal AP. It maybe that the patient may have 2 distinct pathways alternatively have seen septal pathway phenotype in patients with both left lateral and right lateral pathways which summate to look like septal pathway. Would love to see results of EP study.

Spanish

ECG # 1 parece AP antero-septal mientras que el ECG # 2 parece póstero-septal AP Puede ser que el paciente pueda tener 2 vías distintas alternativas o haber visto el fenotipo de la vía septal en pacientes con ambas vías lateral y lateral izquierda las cuales sumadas parecem via septal. Me encantaría ver los resultados del estudio del estudio electrofisiológico.

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Estimado Potro: la respuesta más sensata el la del Dr. Gonzalez. Pero hay algunas cosas que al observar los ECGs no encuentro concordancia.
La onda P en DI y aVF entre ambos electrocardiogramas son diferentes en las derivaciones de los miembros e iguales en las precordiales. En el ECG-1 esta desviada a la izquierda lo que va en contra de la posibilidad de la anomalía de Ebstein.
En el segundo electro el eje electrico de la onda P es normal.
Ambas vias solo varían en las derivaciones de los miembros y no en la progresión en precordiales.
Perfectamente puede tener 2 vias anómalas pero encuentro poco probable solo varien en las derivaciones de los miembros y no en las derivaciones precordiales.
Por lo que mi presunción es la inadecuada colocación de las derivaciones de los miembros, que crea el artefacto de diferente conducción de 2 vías anómalas. El segundo ECG es el correctamente obtenido.
Un cordial saludo
Martín Ibarrola

Dear Foal: the most sensible answer is that of Dr. Gonzalez. But there are some things that when I observe the ECGs I do not find agreement.
The P wave in I and aVF between the two ECGs are different in the derivations of the limbs and equal in the precordial ones. On the ECG-1, this is shifted to the left which is against the possibility of Ebstein's anomaly.
In the ECG-2, the electric axis of the P wave is normal.
Both pathways only vary in limb and not in precordial progression.
It can perfectly have 2 anomalous pathways but I find it unlikely to only vary in limb leads and not in precordial leads.
So my presumption is the **inadequate placement of the members' leads**, which creates the artifact of different conduction of 2 anomalous pathways.
The second ECG is the one correctly obtained.
A cordial greeting
Martin Ibarrola MD Buenos Aires Argentina

Dear Martín,

I think Dr. Mario did not mean a diagnosis of Ebstein; he just mentions it as a congenital disease in which multiple accessory pathways are associated.

My opinion is akin, in terms of the association with great vessels transposition. We did not say they are diagnostic, just associations. Having clarified this point, I continue with another. Your reflection in terms of a probable exchange of electrodes in the frontal plane is very interesting.

I do agree with Sergio's opinion.

There is an exchange of the left arm lead by the left leg lead:

1) The P wave is of greater width in I than in II
2) Thus, leads I and II are exchanged
3) aVR does not change
4) aVL and aVF are exchanged
5) III is a mirror image
6) The changes in precordial leads are null or minimum (there should be changes in width or voltage if the "exchanged" electrode was the indifferent one, in the right leg).

This mistake in the placement of electrodes is hard to diagnose if no previous ECG is available.

In this case, the previous ECG is number 2.

In ECG1 P wave of DI > DII (with this being the ECG with a recording mistake).

Sorry for the length.

Best regards,

Juan Carlos **Manzzardo MD** Mendoza Argentine

Note: Dear Mazzardo,

When you say that ventricular pre-excitation is associated to transposition of the great vessels, I suggest you to be clearer, because the one associated to pre-excitation is the congenitally corrected transposition of the great arteries. If you don't clarify it, the colleagues may think you mean the transposition of the great vessels.

In the congenitally corrected transposition with pre-excitation, the substrate is the presence of Ebstein's anomaly of the left atrioventricular (mitral) valve apparatus, located between the morphologically left atrium and the morphologically right ventricle[1]. On the other hand, more than 75% of the patients with congenitally corrected transposition have some degree of AV block, from first-degree block, going through the second-degree one (generally 2:1), to complete AV block with narrow QRS. The latter is observed in 30% of T cases, and the degree of block typically varies once in a while in the same patient.

The transposition of great vessels IS NOT ASSOCIATED to ventricular pre-excitation. The corrected one is unfrequently associated, and always in an Ebstein scenario.

1. Bharati S, Rosen K, Steinfield L, Miller RA, Lev M.The anatomic substrate for preexcitation in corrected transposition. Circulation. 1980 Oct;62(4):831-42.

Andrés Ricardo **Pérez-Riera**

One of the 2 tracings has reversal of left arm and left leg cables.

Sergio Pinski MD

Sent from my mobile device

Dr. Sergio Pinski is a cardiologist in Fort Lauderdale, Florida and is affiliated with Cleveland Clinic Florida. He received his medical degree from University of Buenos Aires and has been in practice for more than 20 years. He is one of 25 doctors at Cleveland Clinic Florida who specialize in Cardiovascular Disease. He also speaks multiple languages, including Spanish.

This is the most appropriate observation. The lead exchange, which is shown by the discrepancy between the peripheral and precordial leads.
JOSE ANIBAL COELHO FREIRE

I cannot give definite advice for 2 reasons...1. The atrial rhythm seems very different on the 2 traces. Is this due to different rhythms or merely to misplacement of the standard electrodes..2. The degree of preexcitation in both tracings is small that renders discussion difficult..i have the feeling we are dealing with tracings belonging to a pediatric patient.

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Have a nice week from the undivided Jerusalem where I spent Shabbat.

Bernard Belhassen
Head of the Electrophysiology Laboratory, Department of Cardiology,
Tel-Aviv Medical Center, Tel-Aviv

Some years ago we published this paper, that gave us plenty of gratification, including an acronym to remember the change of electrodes, “REVERSE”: Crit Care Nurse.<https://www.ncbi.nlm.nih.gov/pubmed/?term=baranchuk+AND+commandements#> 2009 Feb;29(1):67-73. doi: 10.4037/ccn2009607.

Dr Adrian Baranchuk, MD FACC FRCPC FCCS
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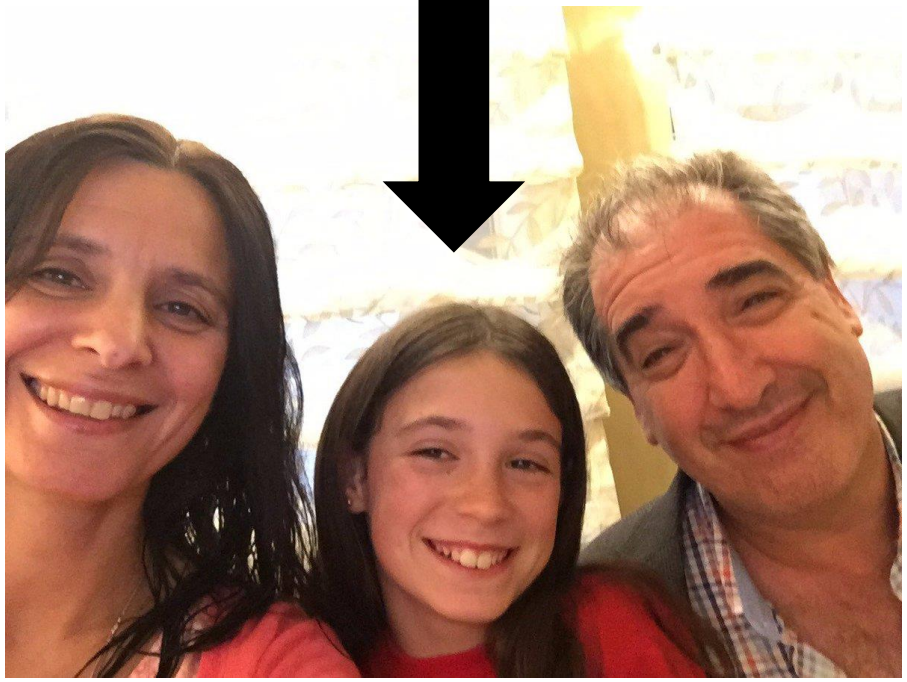
Gala



Final Conclusion by Andrés Pérez-Riera

Although multiple accessory pathways have been reported in as many as 10% of cases (**Bardy 1984**) and should make one think of commonly associated conditions such as Ebstein's anomaly, careful scrutiny of the two ECGs reveal a subtle and often unrecognized lead reversal of the left arm and left leg (LA/LL) electrodes cable reversal. Lead reversal has been found to be present in 0.4 a 4% of all ECGs preformed (**Kross 2001**). Arm lead reversal demonstrates a characteristic pattern easily recognized by most electrocardiografers and computer programs. Right arm with right leg (ground electrode) reversal will result in a straight line in lead II along with the unlikely similarity of aVR and aVF. With reversal of left arm/left leg, as in this case, leads aVL/aVF and standard leads I/II are interchanged, whereas lead III is upside down. In the absence of an available ECG with appropriate lead placement, these changes may not arouse the suspicion of lead reversal.

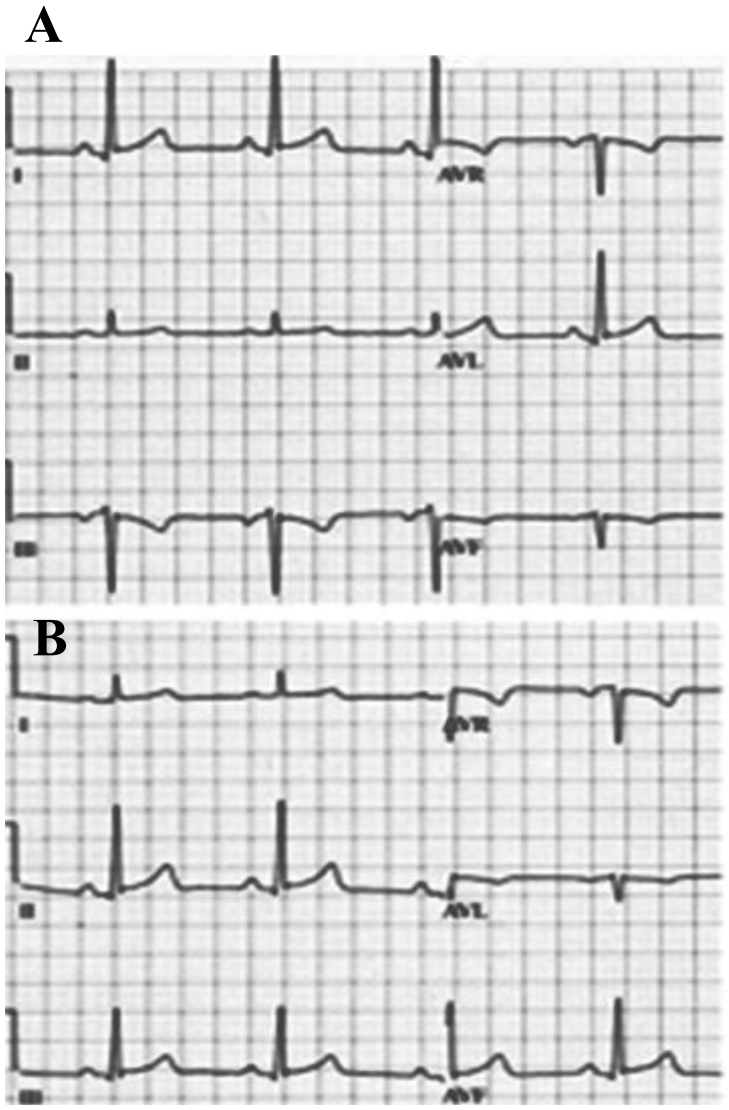
The criteria for diagnosis of LA/LL lead reversal P wave voltage have been used (**Abdollah 1997; Ho 2001**). The P wave in normal sinus rhythm should have greater amplitude in lead II than in lead I. If these leads are interchanged, as in this case, this can be a clue to incorrect lead placement. The P wave in lead III should never have a terminal positive component; the presence of one also is a clue to incorrect placement. Artificial neural networks based on > 10,000 ECGs have been shown to be 100% specific in the diagnosis of LA/LL lead reversal with a sensitivity of 58%, but they are not widely available clinically (**Hedén 1996**).

In this case, the patient underwent electrophysiologic study and was easily inducible into an orthodromic AV reentrant tachycardia. Ablation was performed successfully at a single site in the right posteroseptal region with confirmation of the lack of anterograde or retrograde AV conduction.

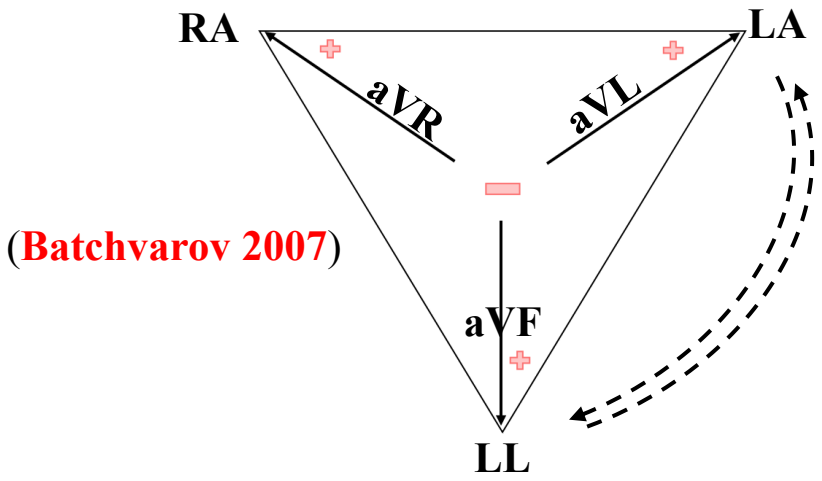
In summary, one should always be aware of the potential for limb lead reversal when considering the preexcited rhythm. If a previous ECG demonstrating preexcitation is not available, the only clue may be the P wave morphology.

As the surface ECG often is used to predict the location of the accessory pathway, this usually subtle electrode reversal can lead to a perplexing clinical enigma.

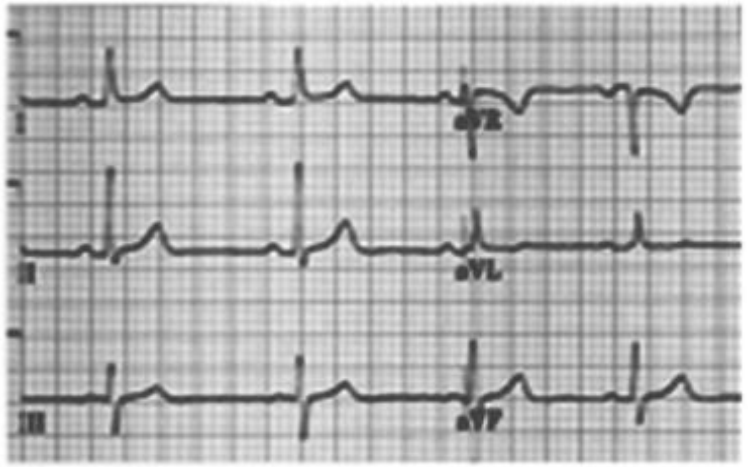
Left arm (LA)/Left leg (LL) lead reversal

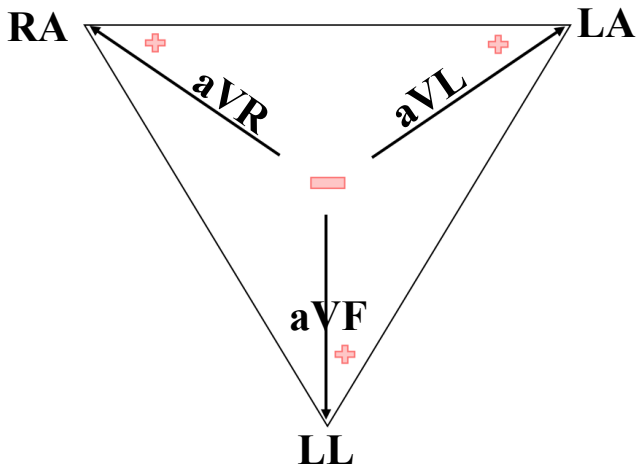


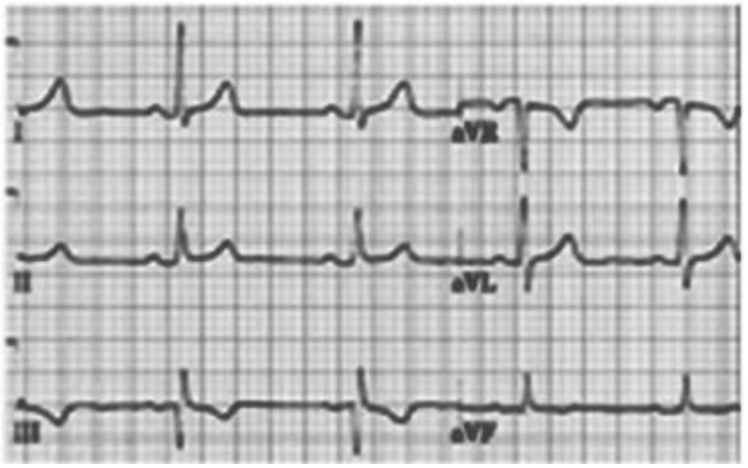
Middle panels



(Batchvarov 2007)

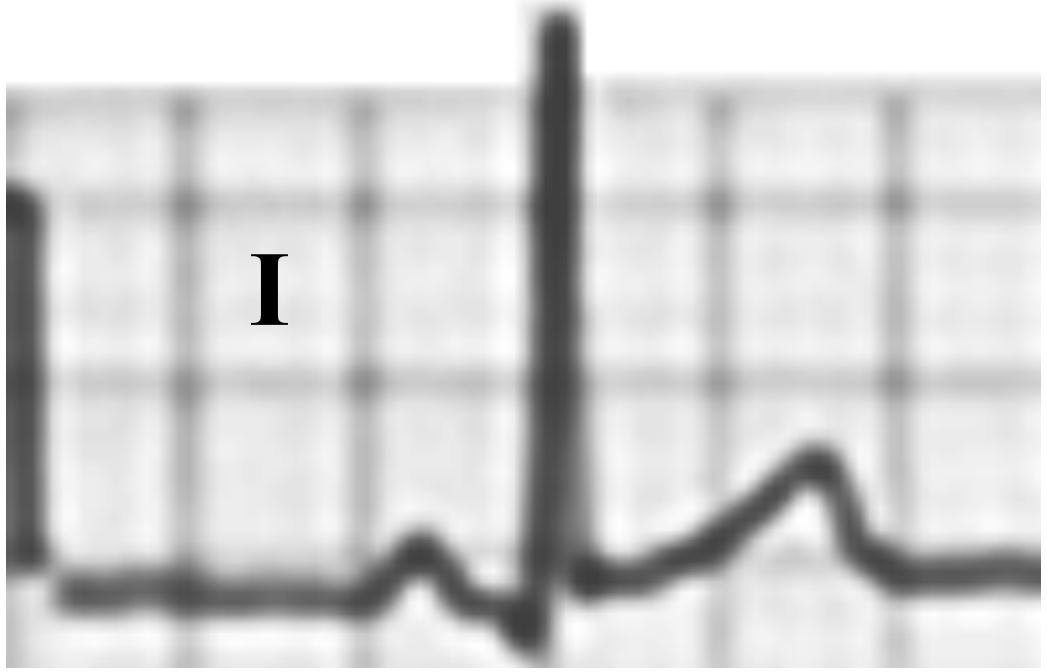


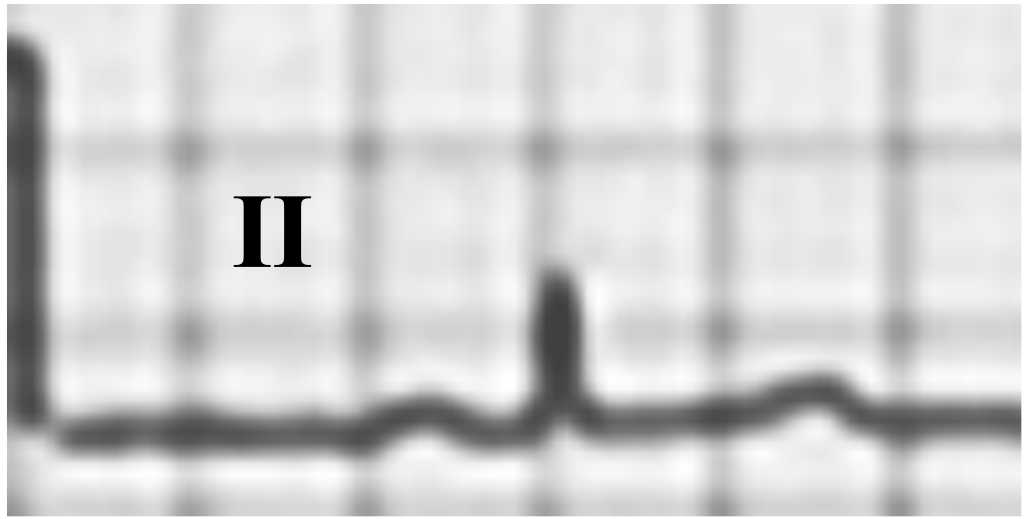




Changes in the I/II P wave voltage is the clue to detect LA/LL cable reversal. The figure shows frontal plane leads of ECGs recorded in a healthy individual (**A**) and in a man with mild hypertension (**B**). The ECGs with LA/LL cable reversal are in the top panels, and those with correct cable connections are in the bottom panels. In the middle panels are the incorrect (top) and correct connection (bottom). In both cases, the ECGs recorded with cable reversal look normal. In **A**, **the clue to detect the mistake is the higher amplitude of the P wave in lead I related lead II (abnormal)** (Abdollah 1997; Ho 2001).

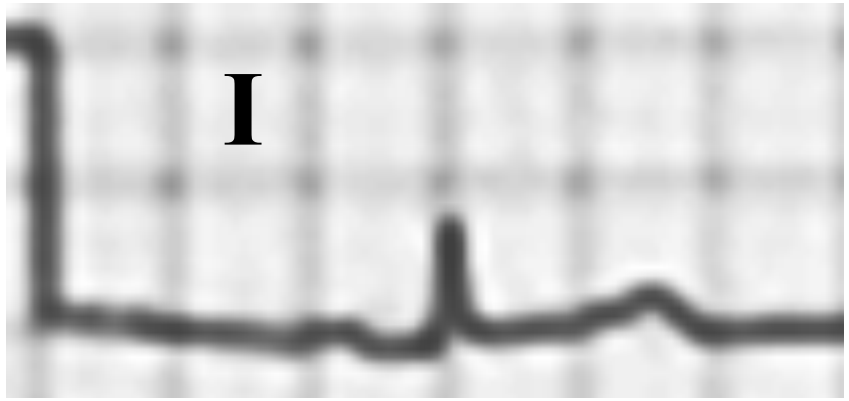
The cable reversal is also suspected if a biphasic minus-plus P wave is registered in III (abnormal) (Goldberger 1942).

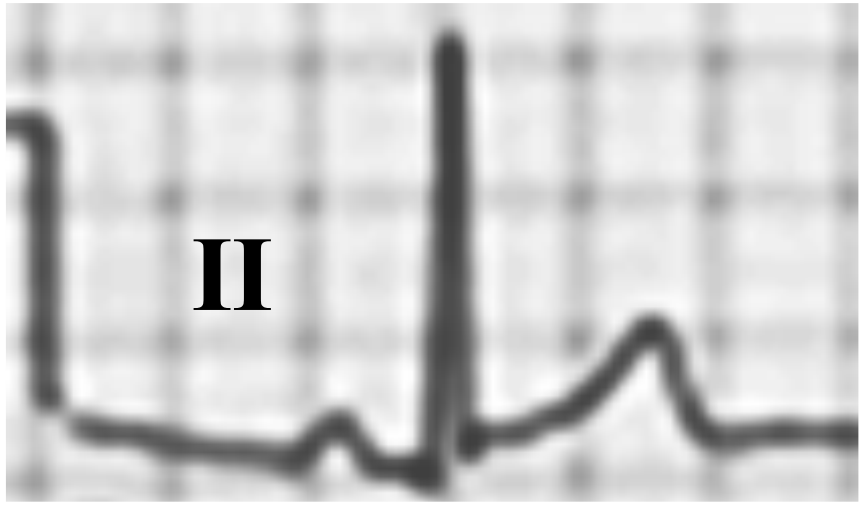




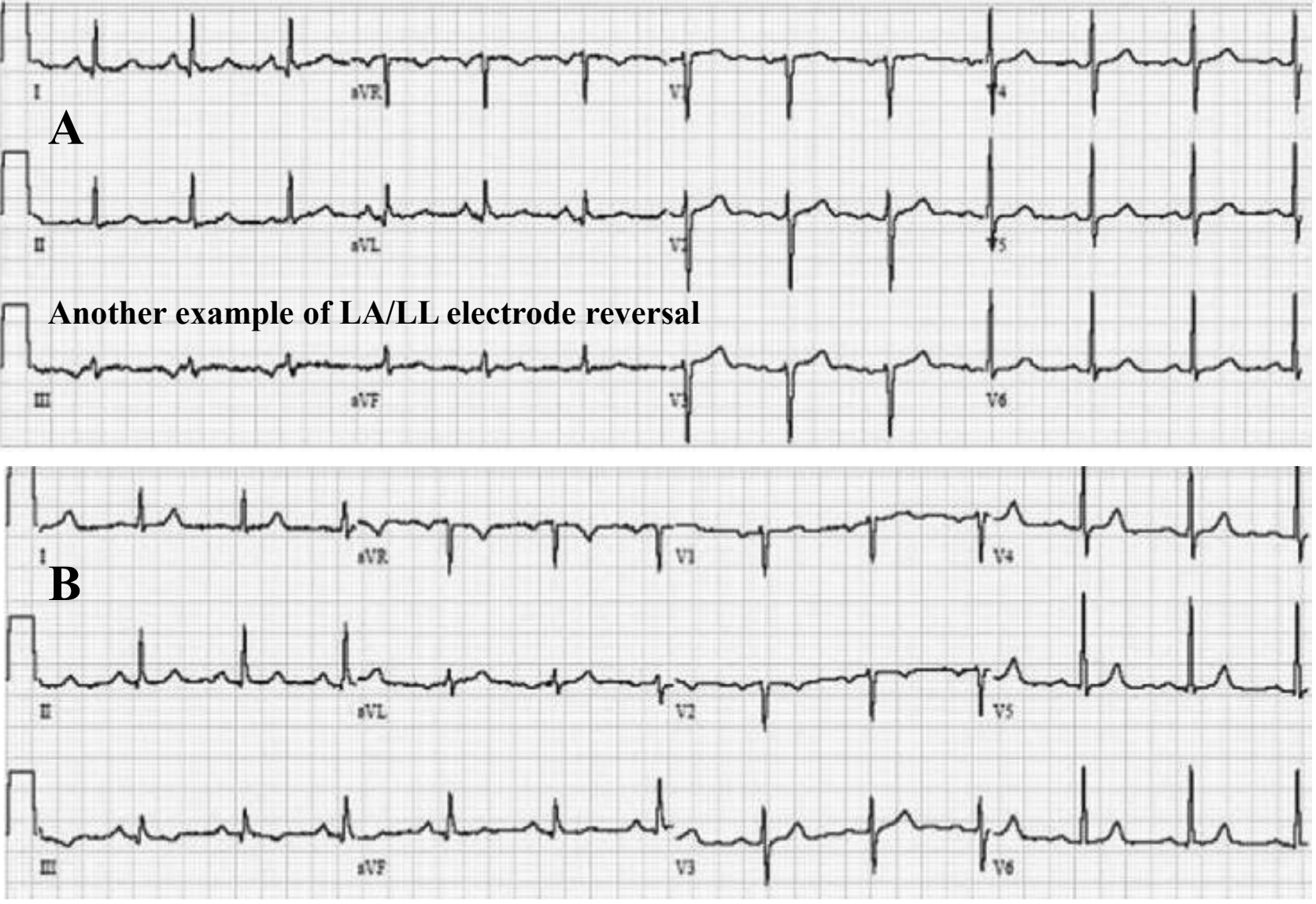
The clue to detect LA/LL cable reversal is the higher amplitude of the P wave in lead I related lead II. This is abnormal.

(**Goldberger 1942**)





Normal position of LA/LL cable. In this case, the PII amplitude > PI amplitude. This is normal.



LA/LL electrode reversal. **ECG-A** tracing does not appear abnormal, but it does appear different than **ECG-B**, which was recorded moments later in the same patient. ECG-A was recorded with the inadvertent reversal of the LA/LL electrodes; ECG-B demonstrates proper electrode positioning. Closer inspection reveals that lead I in one tracing is lead II in the other; similarly, lead aVF in one tracing is lead aVL in the other. Lead III is the inverted image of the comparison tracing, but either lead III appears grossly acceptable. The key to discovering that the tracing in (A) is a LA/LL reversal lies in noticing that the P wave lead I voltage is higher than P wave lead II—this is unusual in most people and should trigger consideration that left-sided electrode reversal has occurred. Additionally, the QRS axis has shifted: it is closer to +30° in A yet closer to +60° in B. Such an axis shift should prompt consideration of electrode reversal as well as body position changes and other pathologic entities.

Finally, in A there is the appearance of poor R-wave progression across the chest leads; indeed, the computer read this as anterior infarction, age indeterminate. B reflects the proper placement of the precordial electrodes, the growth of an R wave in lead V3, and smooth R-wave progression across the precordium—no longer giving the appearance of an indeterminate-age anterior infarction. This tracing was typical of the patient's prior ECGs. This is an example of precordial LA/LL electrode reversal.

So how does one detect LA/LL reversal? One key is analysis of the P wave in leads I, II, and III. In a case series of 70 sinus rhythm tracings, finding a P wave voltage that was smaller in lead II than in lead I, or an upward terminal phase in a biphasic minus-plus, lead III P wave (i.e., P-wave deflection that is down/up, rather than up/down as would be expected in this relatively right-sided lead) correctly predicted reversal of arm and leg electrodes on the left side in 90% of cases (**Knight 2001**). This is the key clue to detecting LA/LL reversal without a comparison tracing. One should also look for unexpected shifts in the QRS axis (and P-wave axis); although this is not specific for limb electrode reversal, it is one cause to be considered when an axis change is evident during comparison of old and new ECGs.

In the presence of typical atrial flutter, LA/LL cable interchange can easily be detected by the appearance of the sawtooth flutter waves in leads I, III, and aVL, but not in lead II (**Surawicz 2001**). The observant ECG reader may even notice that the flutter waves in lead III are inverted (e.g. with the acute angles pointing upwards instead of downwards in case of typical (counter-clockwise, CCW) atrial flutter) (**Abdollah 1997**). In such case, if the typical pattern was not observed previously, it would not be possible to exclude atypical flutter patterns. The inversion of lead III and interchange of leads aVF and aVL (in which the T wave can normally be inverted) can produce negative T waves in leads III and aVF, simulating inferior myocardial ischemia (**Surawicz 2001**). In a patient with inferior myocardial infarction, this cable reversal can cause the disappearance of the negative Q waves and T waves in the inferior leads (**Surawicz 2001**). ECG changes resulting from electrode misplacement or misconnection may simulate clinical disease and lead to misattribution of pathology, and thus affect clinical decision-making and treatment. Patterns of electrode reversal or misplacement can be recognized if the Emergency Physician is aware of the characteristic findings associated with the respective electrode placement errors. Electrode misplacement and misconnection should be in the differential diagnosis of ECG changes when the Emergency Physician interprets the ECG.

References

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