Disociación de ritmos auriculares - 2018

Dr. Gustavo Adamowicz

Estimados colegas del foro,

Les quiero acercar una pregunta...

¿Existe la posibilidad, práctica o teórica, de que al mismo tiempo, un paciente presente una aurícula en taquicardia por reentrada y la otra aurícula en ritmo sinusal?

Igual pregunta para aleteo + ritmo sinusal.

Gracias

Gustavo Adamowicz

OPINIONES DE COLEGAS

Si, por supuesto. Lo vemos en aurículas muy enfermas.

Saludos,

Mario D. Gonzalez

São famosos os traçados com Dissociação Inter átrios

Cada um bate um ritmo independente

Abraços

Fábio Sândoli de Brito

Diretor da Soc. Brasileira de Cardiologia 2010 a 2015

Diretor da Central Brasileira de Holter

Coordenador dos Serviços de Holter e Looper do Hosp. Sírio Libanês

Buena día!!

Dr Gustavo ¿tienes el registro? Interesante la respuesta del Dr Mario González ! nunca tuve la posibilidad de verlo. ¿Se observa en ECG de superficie o endocavitario?

Dr Juan Carlos Manzzardo

Prezado colega Dr. Fabio Sandoli

Alegria imensa em te-lo por aqui e certamente grandes contribuições teremos

Assisti-o na SOCESP no coloquio da reabilitação se não me engano

Abraços

Adail Paixão Almeida

Hola a todos! Sabemos de la posibilidad de ritmos auriculares independientes, pero si alguno de los maestros nos pudiera ilustrar con ejemplos de ECG sería fantástico!

Desde ya muchas gracias! Saludos cordiales,

Patricia Ortiz

Hola

No tengo registro en este momento.

Elevo el tema al foro pues muchas veces un ECG de superficie genera dudas en cuanto a si se trata de una FA o una taquicardia auricular, especialmente al verse actividad auricular organizada en algunas derivaciones.

Por ello las inquietudes que elevo,

- ¿una taquicardia auricular captura siempre las 2 aurículas?

- ¿puede un ECG de superficie predecir 2 ritmos diferentes en cada aurícula?

- ¿puede tener un rol el ritmo independiente de la orejuela?

Gustavo Adamowicz

Buen día estimados integrantes del foro!

La verdad que la pregunta del Dr Adamowicz es más que interesante, sobre todo para los que hacemos Cardiología clínica, y sólo miramos las arritmias por "la ventana" porque nos parece un capítulo fascinante de la Cardiología. Y nos animamos a informar, a veces, "fibriloaleteo auricular" en algunos ECG.

Desde mi ignorancia, les comparto un antiguo PDF publicado por el Dr. Jorge González Videla en Revista de la Sociedad Argentina de Cardiología, donde dice que los primeros registros de "disociación interauricular" se describieron y registraron a principios del siglo XX ! Y major mérito merecen todavía cuando se observa la calidad de los registros de esa época, unos verdaderos genios!

http://www.old2.sac.org.ar/wp-content/uploads/2015/03/PDFs201503/2183.pdf

Quizás algunos de los Maestros del Foro quiera ilustrarnos un poco en este tema propuesto por el Dr Gustavo.

Me despido cordialmente.

Dr. Juan Carlos Manzzardo

Los casos que he visto fueron con registros intracavitarios. Recuerdo en este momento un caso que hicimos con el Dr. Alejandro Cordero en Guadalajara. Teníaaleteo en aurícula derecha y macroreentrada auricular peri-mitral en aurícula izquierda. Desde un comienzo vimos que las aurículas tenían diferentes longitudes de ciclo. Esto no se veía en el ECG.

Luego de la ablacion, comprobamos el bloqueo interauricular.

Saludos,

Mario D. Gonzalez

Queridos colegas del foro: Este manuscrito abajo de ingenioso diseño nos explica con clareza el porqué de la posibilidad de dos ritmos auriculares puedan coexistir en un mismo paciente, aunque el objetivo es otro, es decir aleteo y FA en forma secuencial y no concomitante. Los autores están proponiendo que al tratar el AFL deba también realizarconcomitantemente el aislamiento de las venas pulmonaresLa respuesta está implícita en la inteligente discusión que los autores elaboraron (lean abajo).

Andrés R. Pérez Riera

Steinberg JS, Romanov A, Musat D, Preminger M, Bayramova S, Artyomenko S, Shabanov V, Losik D, Karaskov A, Shaw RE, Pokushalov E. *Prophylactic pulmonary vein isolation during isthmus ablation for atrial flutter: the PReVENT AF Study I*. Heart Rhythm. 2014 Sep;11(9):1567-72.

Abstract

BACKGROUND:

Although catheter ablation of isthmus-dependent atrial flutter (AFL) is successful at eliminating the target arrhythmia, many patients subsequently experience new-onset atrial fibrillation (AF).

OBJECTIVE:

The aim of this study was to determine whether AF can be prevented by prophylactic pulmonary vein ablation in patients with AFL.

METHODS:

A prospective, single-blind, randomized clinical trial in patients whose sole arrhythmia was AFL without AF was conducted. Patients were randomized to cavotricuspid isthmus ablation alone or with concomitant pulmonary vein isolation. All patients received an implantable cardiac monitor.

RESULTS:

Fifty patients completed the trial, and patients were well matched. Isthmus ablation was successful in all patients; pulmonary vein isolation was successful in 25 (100%) randomized patients. Procedure ($P < .0001$) and fluoroscopy ($P < .0001$) times were longer in the combined ablation group. More patients in the isthmus ablation-only group experienced new-onset AF during follow-up (52% vs. 12%; $P = .003$). The 1-year AF burden also favored the combined ablation group compared with the isthmus ablation-only group (8.3% vs. 4.0%; $P = .034$). In the isthmus ablation-only group, 8 (32%) patients subsequently underwent another ablation for AF. The performance of pulmonary vein isolation and male sex were independent predictors of freedom from AF.

CONCLUSION:

In the PREVENT-AF Study I randomized clinical trial of patients in whom only typical AFL had been observed clinically, the addition of pulmonary vein isolation to cavotricuspid isthmus ablation resulted in a marked reduction of new-onset AF during clinical follow-up as assessed with a continuous implantable cardiac monitor.

Discussion

In this prospective randomized single−blind clinical trial, we have demonstratedthat in patients whose sole clinical arrhythmia had been typical AFL, the prophylactic

performance of pulmonary vein isolation (PVI) in conjunction with ablation of the cavo−tricuspid isthmus (CTI) resulted in a marked reduction of new onset AF and need for subsequent AF ablation during follow−up over one year as assessed carefully and comprehensively by an ICM and clinical follow−up. The addition of PVI to the CTI was strongly and independently predictive of freedom from AF after ablation. PVI was performed safely without complications although there was an increment in procedure and fluoroscopy times at the primary ablation procedure. In planning this trial, we believed that PVI in this setting was potentially clinically valuable based on the following premises:

1.Left atrial pathology is common in patients with right AFL, thus facilitating AF;

2.AF and AFL may share common arrhythmic initiation mechanisms, presumably arising in the pvs;

3.The incidence of symptomatic AF after AFL ablation is very high;

4.The presence of AF after AFL ablation can cause continued symptoms and stroke risk; (5) PVI is effective for the elimination of AF, particularly before advanced remodeling has ensued; and

5.The performance of PVI has become efficient and safe in experienced laboratories.

The association of AFL with AF is strong. Mechanistically, Waldo and Feld have proposed that in the vast majority of patients with AFL, antecedent AF is a triggering prerequisite for the establishment of the line of block that generates right atrial macroreentrant typical AFL (**Waldo AL, Feld GK: Inter−relationships of atrial fibrillation and atrial flutter.Mechanisms and clinical implications. J Am Coll Cardiol 2008; 51: 779−786**.). Ablation studies have confirmed that both AFL and AF are likely triggered by PV firing (**Wazni O, Marrouche NF, Martin DO, et al: Randomized study comparingcombined pulmonary vein−left atrial junction disconnection and cavotricuspidisthmus ablation versus pulmonary vein −left atrial disconnection alone inpatients presenting with typical atrial flutter and atrial fibrillation. Circulation2003; 108: 2479−2483**.). If so, once AFL is eliminated, AF vulnerability is exposed and almost inevitable and indeed, clinical observations have borne this out **Da Costa A, Romeyer C, Mourot M, Messier A, Cerisier A, Faure E, Isaaz K: Factorsassociated with early atrial fibrillation after ablation of common atrial flutter. EurHeart J 2002; 23: 498−506**.). The incidence of AF after AFL ablation has been reported to be as high as 82%. In our recent study that was advantaged by continuous ECG acquisition using an ICM, we also observed a very high short−term incidence of new onset AF of 55% (**Mittal S, Pokushalov E, Romanov A, Ferrara M, Arshad A, Musat D, Preminger M,Sichrovsky T, Steinberg JS: Heart Rhythm 2013; 10: 1598−1604**.). If AFL is very frequently initiated and accompanied by AF and if successful elimination of AFL by ablation exposes the patient to AF and its consequences, it stands to reason that therapy of AF may be an inevitable necessity in many or most of these patients. At present, there is no standardized management protocol or formal treatment guideline for this common scenario. PVI has, however, become a common intervention for patients with documented AF, and has a Class I indication for patients who have drug−refractory symptomatic paroxysmal AF (**Calkins H, Kuck KH, Cappato R, et al: 2012 HRS / EHRA / ECAS Expert consensusstatement on catheter and surgical ablation of atrial fibrillation:recommendations for patient selection, procedural techniques, patientmanagement. Heart Rhyth 2012; 9: 632−696**.).

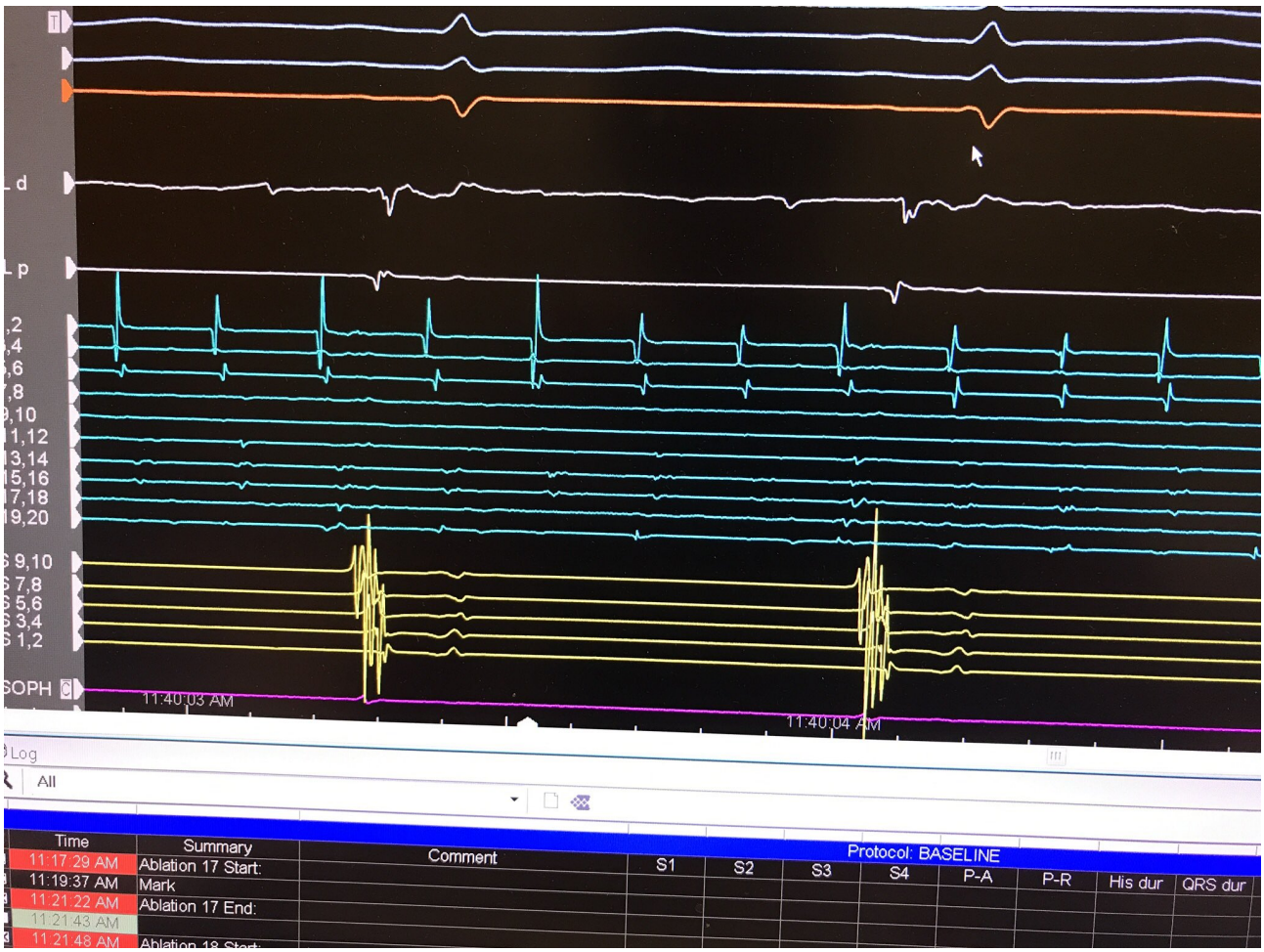
PVI is associated with good AF suppression, particularly when paroxysmal, and can be performed safely in the vast majority of patients. Importantly, the most common complications are related to vascular access, which is already part of the AFL ablation procedure. In experienced laboratories, PVI can be accomplished expeditiously with minimal radiation exposure. Given these justifications, a study to test whether prophylactic PVI could reduce AF that follows the AFL ablation would be of clinical interest. Navarette et al (**Navarrete A, Conte F, Moran M, Ali I, Milikan N: Ablation of atrial fibrillation atthe time of cavotricuspid isthmus ablation in patients with atrial flutter**

withoutdocumented atrial fibrillation derives a better long−term benefit. J CardiovascElectrophysiol 2011; 22: 34−38.) suggested in a study of 48 patients that PVI in combination with additional LA linear or electrogram−guided ablation (if AF was inducible) reduced the incidence of AF from 56% to 13% after AFL ablation, similar to our findings although AF detection after a 2 month blanking period was by 48 hr Holter recording every 2 mos. This design feature may have reduced AF detection. In our study, by protocol design, prophylactic PVI alone was used without any additional LA ablation and specifically selected to make the feasibility of this approach potentially more generalizable, efficient and presumably safer. Recent contemporary data in a very large observational study suggest that the fatal and very serious irreversible complications are extremely rare (**Schmidt M, Dorwath U, Andresen D, Brachmann J, Kuck K−H, Kuniss M, LewalterT, Spitzer S, Willems S, Senges J, Junger C, San M, Hoffmann E. Cryoballon versusRF ablation in paroxysmal atrial fibrillation: Results from the German AblationRegistry. J Cardiovasc Electrophysiol 2014; 25: 1−7**.). In addition, our study was greatly advantaged by continuous ECG monitoring using an ICM so that asymptomatic events would not be lost and genuine AF burden could be quantified. Only 12% of patients who underwent PVI with CTI developed AF, much less than the 52% who had only CTI, among whom almost one third required subsequent PVI as a second ablation procedure. In the combined CTI + PVI group, the AF burden did not increase after 3 mos in contradistinction to the CTI only group where the AF burden progressively increased. The small AF burden early post−ablation in the CTI + PVI group may reflect the effects of remodeling resulting from AFL and thus may be self−limited and temporary. Prophylactic PVI is a new treatment concept and must be well validated before it is introduced into clinical practice. Although we have demonstrated the feasibility of this approach and have succeeded in reducing AF incidence, more robust clinical outcomes should be examined in larger data sets to establish the ultimate net clinical value of prophylactic PVI. The targeting of patients with known AFL was advantageous because these patients were in the pipeline to undergo catheter ablation and thus already exposed to a portion of the risks of an interventional approach, and because of the well-established and very high risk of subsequent AF. For those who go on to experience AF, there will be a toll on quality of life, and most will require therapy to control symptoms and require chronic anticoagulation. Assuming AF will need to be addressed at some point in the not too distant future, it may be reasonable to consider eliminating or limiting its prevalence before it starts if indeed it is inevitable. If there was no incremental cost or risk, the answer would clearly be affirmative. But of course, there is an increment in cost (mainly the catheters needed for PVI) and*small*risk. One could argue that there is not greatly increased risk as the most common complications for both AFL and AF ablation are access−related, and thus shared. There is no question that there are infrequent complications unique to PVI that would not occur with CTI ablation but in high volume experienced labs using contemporary techniques, these risks are now very small. Finally, costs and risks will be later borne by those who need PVI if it were not performed with CTI, and would by definition be greater because a second

hospitalization, lab procedure and access for ablation would be required in a proportion of the original patient group. So the argument is plausible, but unproven, that prophylactic PVI is potentially cost−effective and associated with improved quality of life and reduction in health care expenditures. Like all prophylactic interventions (most notably, ICD therapy), patients may be treated who would never need the intervention. Whether patients will have tangible benefits or will be exposed to an unfavorable balance of risk from the procedure, must be the subject of a larger randomized trial that includes a whole range of relevant endpoints. Finally, there may be other populations of high−risk patients in whom this prophylactic strategy may be desirable, but clearly additional prospective investigation will be necessary.

Trazado sacado de Twitter: fluter en AI (celestes), sinusal en AD (amarillo), probable después de múltiples líneas de bloqueo postablación
Abrazo!

Oswaldo Gutiérrez



Estimado Gustavo,

Si bien existe la posibilidad real de disociación eléctrica interauricular, la misma es muy rara.

No hay forma de hacer diagnóstico por ECG. Si bien hay algunas fichas que plantean el diagnóstico mediante ECG de superficie, eso no es real. Todos los casos que conozco son diagnosticados por registros invasivos intracavitarios.

Creo que esto es importante tener presente estas limitaciones a la hora de informar un ECG o un HOLTER. No hay que hacer diagnóstico de fibriloflutter. Una causa muy común de confusión es que muchas fibrilaciones auriculares son organizadas en V1, presentando ondas auriculares a 300 ms, positivas, que generan le generan la duda al médico respecto al diagnóstico, y se suele plantear flutter cuando en realidad es FA.

Un abrazo,

Daniel Banina

Interesantes los comentarios.

Y para el caso de Oswaldo, con AD sinusal y AI aleteada...el ECG de superficie fue aleteo?

Gustavo Adamowicz

Generalmente la actividad en la aurícula izquierda es la que se expresa en el ECG por tener más masa muscular.

Mario **D. González**

Estimados compañeros del foro:
Muy interesantes los comentarios expuestos. Me resultaría muy difícil realizar un diagnóstico de una arritmia ya sea una FA o taquicardia auricular en la AI y ritmo sinusal en la AD. Como bien dice Mario la AI posee más masa muscular y a pesar de que en las derivaciones precordiales la que mejor observamos es la AD no en las frontales, la irregularidad de la línea de base hace imposible tanto en el ECG como en el VCG su diagnóstico.
Yo en mis años de médica he observado un solo caso de FA en la AI y supongo que un ritmo sinusal en la AD y esto fue diagnosticado por el ecocardiograma.
Afectuosamente

Isabel Konopka