CLINICAL RESEARCH

Clinical characteristics and management of paroxysmal junctional tachycardia in the elderly

Caractéristiques cliniques et prise en charge des tachycardies jonctionnelles paroxystiques chez le sujet âgé

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Summary

Background. — The proportion of elderly subjects is in progress. While atrial fibrillation is the most frequent arrhythmia after the age of 70 years, other tachycardias also occur in the elderly.

Aims. — The aim of this study is to assess the clinical and electrophysiological characteristics of paroxysmal junctional tachycardia (PJT) in patients older than 70 years.

Methods. — Eight hundred sixteen patients aged from 8 to 93 years have been consecutively recruited for PJT. Among them, 141 (17%) were older than 70 years. The clinical, electrophysiology and therapeutic data were studied.

Results. — Forty-eight men and 93 women with an age range from 70 to 93 years (mean 76±5) were admitted for recurrent PJT. They were associated to cardiac decompensation in 10 cases, syncope in 26 cases, acute coronary syndrome in 14 cases and unexplained acute vascular event in 5 cases. The electro-physiological mechanism of the PJT was similar to the younger patients with a majority of nodal reentrant tachycardia (73%). Atypical nodal tachycardias were more frequent than in the youth (15 versus 4%). PJT ablation was indicated more frequently in elderly patients than in younger patients (79 versus 57%), but complications (7% versus 2.5%) and failures, especially related to atrial fibrillation induction (19% versus 5%) were more frequent in elderly patients.

Conclusion. — Junctional tachycardias are not rare in the elderly and should not be missed. A cautious medical treatment can be impeded by the presence of conduction troubles or comorbidities. If this approach is not efficacious, these subjects might take benefit from curative ablation, with still a 10% failure rate.

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KEYWORDS
Junctional tachycardia; Geriatrics.

MOTS CLÉS
Tachycardie jonctionnelle ; Gériatrie.

Résumé

Justification. — La population âgée augmente. Si la fibrillation auriculaire (FA) est l’arythmie la plus fréquente après 70 ans, d’autres tachycardies surviennent chez le sujet âgé.

Objectifs. — Le but de cette étude a été d’évaluer les caractéristiques cliniques et électrophysiologiques de sujets de plus de 70 ans ayant une tachycardie jonctionnelle paroxystique (TJP).

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Introduction

The auriculo-ventricular junction tachycardias or paroxysmal junctional tachycardias (PJT) use in their total or partial trajectory the Tawara node and/or the His bundle down to its bifurcation.

They are mostly paroxysmal and rarely chronic. The PJT are mainly due to nodal reentry (typical or atypical) and sometimes related to a latent Bundle of Kent with exclusive retrograde conduction. Junctional tachycardias can also complicate the evolution of a patent Wolff-Parkinson-White, but this affection is more infrequent and is presented in another study and this as far as other more severe tachycardias may also complicate this affection.

In the United States, 89,000 new cases are reported yearly, and a total of 570,000 patients are affected by paroxysmal supraventricular tachycardia. The prevalence is estimated at 2,25/1000 subjects, but these data involve all types of supraventricular tachycardia as a whole [1].

Classically benign in the youth, junction tachycardia is often poorly tolerated in the elderly, because it is rapid or unfelt but prolonged or due to an underlying cardiac disease [2].

Despite, this condition is more known in young subjects and infrequently reported in the elderly. Above all paroxysmal and sustained atrial fibrillation are reported after the age of 70, even though PJT is not rare in the elderly.

The aim of this study is to assess the clinical and electrophysiological characteristics and the therapeutic consequences of PJT noted in the elderly.

Population and methods

Patients

The study population includes 141 patients (48 men and 93 women) aging from 70 to 93 years (mean 76±5 y) who presented several episodes of PJT despite drug therapy. They are selected from a series of 816 patients studied between 1985 and 2006 for the assessment of probable or documented tachycardia. Non-critical ECG recorded sinus rhythm without pre-excitation.

The diagnosis has been confirmed by ECG during tachycardia showing regular tachycardia with sudden onset and termination, with narrow QRS complexes similar to those during sinus rhythm. They occurred spontaneously or after being triggered during esophageal and/or intracardiac electrophysiology (EP) exploration.

Methods

Electrophysiology study protocol

The electrophysiology (EP) study was performed through esophageal exploration if the tachycardia was not documented or intra-cardiac exploration when the tachycardia was already documented, in order to attempt ablation. The protocol was similar for the two approaches although the means have been different.

The esophageal auricular pacing was performed during outpatient consultation. We used a bipolar pacing catheter (Fiab, ELA/Sorin, Prothia SP 8). Pacing was first performed using a standard EP pacer (Explorer 2000 Ela), connected to an impulse amplifier (Ela medical), which can deliver 20 ms width and 29 V amplitude pulses. In practice, 16 ms width impulses were actually used, with mean amplitude varying from 15 to 25 V [3]. Since 3 years ago, we have used the intra-cardiac biphasic pacer (Micropace, Bard France). This system can deliver impulses of 10 ms width, and 8 to 25 mv amplitude.

The protocol, common to both intra-cardiac and esophageal pacing has already been reported elsewhere [3]:

- basal state: we performed increasing frequencies auricular pacing up to the Wenckebach point, and then used a programmed auricular pacing using 1 and 2 extrastimulus delivered in two imposed cycles of 600 and 400 ms;
- if tachycardia was not triggered: this protocol was repeated after isoproterenol infusion at the dosage of 0.02 to 1 µg/mn, adjusted for heart rate, with mean heart rate ranging from 130 to 150 bpm;
Definitions

Once the tachycardia triggered, the esophageal or intra-cardiac ECG was recorded. To determine the reentry site, we used the classical EP parameters. Specifically we noted the triggering mode of the tachycardia by looking for a sudden jump of AV delay as a sign of presence of a double nodal pathway. We also looked for the effects of a bundle branch block on the tachycardia cycle, and examined the P wave morphology in leads V1 and I, and above all noted the position of the auricular complex compared to the ventricular complex.

- Junctional tachycardia (T) was attributed to atrioventricular nodal reentry (AVNRT) if it was started after a sudden jump of the AV delay, if the VA interval was below 70 ms, and if there was an AV dissociation during the episodes of tachycardia;
- the tachycardia was attributed to a reentry through an accessory pathway (AVRT) if the atrioventricular complex followed the ventricular one after a delay exceeding 70 ms or if the functional right or left bundle branch block increased the tachycardia cycle, if P wave was negative at lead I and if the P wave at V1 lead occurred after esophageal atrial activity. The VA delay was constant;
- the mechanism remained undetermined if these criteria were not noted, if during intra-cardiac exploration a retrograde conduction through the normal or accessory pathways occurred which was not present under the spontaneous tachycardia frequency even under isoproterenol and finally if the clinical tachycardia was not induced.

Radiofrequency ablation of the reentry circuit

In case of indication of radiofrequency (RF) ablation of the reentry circuit, this protocol also included increasing frequency ventricular pacing, in order to study the retrograde conduction.

In a second step, the RF ablation of the reentry circuit (ablation of the slow pathway in case of nodal reentry or of a latent accessory bundle) was performed using the “Stocksst” device (Cordis, USA) with a power delivery limited at 50W and temperature limited at 65°C during 60 seconds [2].

After ablation, the initial pacing protocol is repeated during 30 minutes after the shot, and if the tachycardia is triggered again, the same procedure was repeated. The procedure was stopped when no retrograde conduction through the bundle of Kent was found or when it was impossible to trigger the junctional tachycardia related to nodal reentry. In this case, the procedure was stopped either in case of complete ablation of the slow pathway, or when it was only possible to induce only 1 to 3 echoes during the programmed pacing technique.

Statistics

The results are presented by the mean and standard deviation. The differences in the clinical and EP data were tested using the Chi-2 and Student’s t-test as appropriate.

Results

Clinical characteristics

A majority of patients presented palpitation, sometimes for years, but being more frequent and refractory to anti-arrhythmic drugs. Three patients have experienced these episodes for 20 years. An 88 years-old patient has presented these episodes since the age of 20, and another patient of 73 years has experienced these episodes since the age of 4.

The tachycardias were sometimes slowed by anti-arrhythmic drugs or were spontaneously slow due to underlying conduction abnormalities.

The tachycardia could have been prolonged during several hours and even days, turning to be ceaseless or chronic in 8 patients. Palpitations could have been increasingly disabling and poorly tolerated. Conversely, in some patients, the tachycardias remained asymptomatic.

Twenty-six patients (18%) presented presyncope (n=3) and/or syncope (n=23). During tachycardia episodes, 14 patients (10%) experienced angina, sometimes very disabling. Five patients presented an unexplained stroke and AVNRT was the only arrhythmia induced during esophageal EP exploration. Ten patients had a heart failure episode due to tachycardia.

Associated conditions

Heart disease was associated to the tachycardia in 35 patients (25%):

- a 76 year-old female patient presented an atypical AVNRT complicated by acute pulmonary edema, with one episode managed with mechanical ventilation. She presented a non-obstructive hypertrophic cardiomyopathy (HCM). Two additional cases also had HCM;
- sixteen patients suffered from ischemic heart disease, with one case complicated by dilated cardiomyopathy. The patient had triple-vessel disease;
- eight other cases had dilated cardiomyopathy, appearing as primitive in 4 cases, following the use of anthracyclines in one patient and related to hypertension in another case. In 2 other cases with almost permanent tachycardia due to a slow accessory pathway and admitted for NYHA stage IV heart failure, the left ventricular function has been normalized a few weeks after the ablation of the accessory bundle;
- three other cases presented hypertensive heart disease;
- two patients had chronic pulmonary heart;
- four patients presented a progressed valvular disease (mitral regurgitation in 2 cases, mixed aortic stenosis and regurgitation in 2 cases);
- three patients had an associated blood disease and 3 other cases had chronic respiratory failure.

Associated arrhythmias (n=59,42%)

In 15 patients (11%) a substantial bradycardia or sinus node dysfunction was present;
- three patients had a first-degree AV block, including three cases with RBBB and another case with left anterior fascicular block;
- five other patients had a RBBB;
- three patients had a LBBB and three others presented a left anterior fascicular block;
- one patient had a pacemaker;
- nineteen patients presented other types of supraventricular tachycardia (atrial fibrillation or atrial tachycardia);
- one patient presented also episodes of ventricular tachycardia.
Tachycardia characteristics

The PJT was due to a typical AVNRT in 101 patients (72%), and was related to an atypical AVNRT in 21 patients (15%). In this latter case the interval between the QRS complex and retrograde atrial activity was very long and the retrograde conduction used the AV node. The PJT was due to a reentry through a latent bundle of Kent in 11 patients (8%). We found both typical and atypical tachycardia in 2 patients. One patient had a typical AVNRT as well as a latent bundle of Kent.

In one patient the nature of tachycardia was difficult to characterize: it was a “slow-slow” atypical AVNRT or a latent right posteroseptal bundle of Kent.

In two cases the tachycardia was connected to a focal automatic activity in the nodal area.

Other electrophsiologic data

A sinus node dysfunction was found in 13 cases.

One patient had an intra-His block and another a prolonged HV interval at 75 ms.

Two patients who had a LBBB presented a traumatic and regressive complete AV block during the manipulation of intra-cardiac catheters.

In 27 patients (19%) an atrial fibrillation was induced, including two with a history of stroke.

Treatment

In 29 patients PJT was controlled with low doses of beta-blockers.

The indication of RF ablation was decided in case of poorly tolerated treatment or in case of recurring PJT in 112 patients. The ablation of the slow pathway or the latent bundle of Kent was attempted in 102 and 10 cases, respectively.

A high rate of 11 failure cases was noted due to the failure to approach of the retrograde bundle of Kent due to severe atherosclerotic lesions, or in case of induction of irreducible tachycardia or atrial fibrillation in 7 cases. Other causes of failures were the very proximal situation of the slow pathway next to the His bundle (2 cases), the occurrence of traumatic regressive complete AV block only after the catheters withdrawal in one case. Small Koch triangle was noted in 12 patients.

Seven patients presented a regressive second-degree AV block.

Two deaths are directly related to cardiac catheterization. The first was due to the occurrence of a huge femoral hematoma in a female patient with Kahler disease followed by intravascular disseminated coagulation and consecutive cerebral hemorrhage. In the second case a cardiovascular collapse occurred when the patient got up the day after the procedure. She had a mild aortic stenosis. The telemetric surveillance during death revealed no arrhythmia.

Follow-up

The follow-up period ranged from 6 months to 7 years.

Two patients presented unexplained stroke despite the ablation of the slow pathway. Patients who experienced stroke prior to the procedure did not present any recurrence but the occurrence of paroxysmal AF in two cases led to the prescription of anticoagulants.

A PJT recurrence was noted in 10 patients with a new ablation procedure. In 8 cases PJT was difficult to trigger in the beginning of the procedure, with the induction of atypical tachycardia in 5 cases. In these 8 patients PJT was finally impossible to trigger again and a block of the slow pathway was apparently obtained for all of them, without induction of echoes. In two other cases an accelerated junctional rhythm (>100/min) remained inducible after isoproterenol infusion, while initially a more rapid junctional tachycardia (>150/min) was induced.

Two out of 10 patients who initially had a reversible second-degree AV block finally needed a pacemaker several months later.

An irreducible atrial fibrillation was noted in 12 patients after 1 month to 5 years after ablation.

Two patients deceased during follow-up: In one case sudden death occurred 3 months after ablation of an AVNRT. The other case was related to cardiac decompensation due to a rapid atrial fibrillation, 8 months after the procedure.

Conversely, the patients who experienced an acute coronary syndrome or heart failure during an episode of PJT have not been readmitted in hospital during follow-up and remained in stages I or II of their conditions.

Comparison with subjects <70 years of age

Table 1 shows higher rates of concomitant cardiac diseases after the age of 69. During cardiac catheterization, syncope and risk of atrial fibrillation induction was also more frequent in the older group. The indication of ablation has been more frequent in the older patients, with a higher rate of complications. The mechanisms of tachycardia were similar in both groups, however atypical AVNRT occurred more frequently in the older patients.

Discussion

Compared to the adult patients, several points differ in the elderly population.

| Table 1 Comparisons of clinical, EP and therapeutic data between our study population versus patients younger than 70 years of age. |
|---|---|---|---|
| | Age <70 | Age >69 |
| Cardiac disease | 11 (1.6%) | 35 (25%) | P<0.001 |
| Syncope | 11 (1.6%) | 26 (18%) | P<0.05 |
| AVNRT | 76 (16%) | 101 (72%) | NS |
| Latent Bundle of Kent | 499 (74%) | 11 (8%) | NS |
| Atypical Tachycardia | 59 (9%) | 21 (15%) | P<0.05 |
| Ablation | 26 (4%) | 112 (79%) | P<0.01 |
| RF complications | 383 (57%) | 10 (7%) | P<0.05 |
| Induced AF | 17 (2.5%) | 27 (19%) | P<0.01 |

RF: radiofrequency.
Clinical characteristics and management of paroxysmal junctional tachycardia in the elderly

The clinical signs were often more critical with syncope in 26% of cases, cardiac decompensation in 7% of cases and acute coronary syndromes in 10% of cases. This poor tolerance is probably explained by a higher rate of cardiac comorbidities (25%) whereas typically, in a young patient, PJTs occur on a healthy heart and are manifested by palpitations with favorable prognosis [4]. A majority of patients are seen between the ages of 50 and 60 [3], with frequently a long history of initially well-tolerated tachycardia, turning to be less and less well tolerated with aging.

In the elderly, recurrence is more frequent [5], usually with atypical clinical presentation, poor tolerance signs, which can involve the vital prognosis [6]. The tachycardia may lead to discomfort or syncope episodes, related to an excessively high heart rate, in a patient with progressed heart disease or presenting concomitantly carotid lesions, or more often due to a vagal reaction [7].

Tachycardia can induce a coronary syndrome with episodes of angina, sometimes functional, in patients free of significant coronary lesions. This can be at origin of frequent hospital admissions in the emergency room or in cardiology units for chest pain, since these older subjects are at risk of atherosclerotic disease. Junctional tachycardia can also destabilize an authentic, even severe, coronary artery disease.

When relatively slow, this tachycardia can be undiagnosed. When prolonged, it may induce overt heart failure as well as arrhythmic cardiac disease [8], which can be totally regressive after the arrhythmia treatment.

Some unexplained strokes may be due to the transformation of PJT to atrial fibrillation [9]. The relationship between these two entities is well known [10]; 12% of patients with junctional tachycardia develop atrial fibrillation during the first year [11], with an increasing risk with aging. The risk of atrial flutter also exists [12]. These risks are not decreased after PJT ablation [13]. We report 13% incidence of atrial fibrillation (11% after ablation).

The PJT has exceptionally been associated to sudden death [6, 8], if it occurs in the context of hypertrophic and obstructive cardiomyopathy, it can be responsible for blood pressure fall, which can lead to ventricular fibrillation. The preventive treatment is therefore mandatory. Drug therapy is often difficult to use since sino-atrial and AV conduction troubles are frequent (28% in our series). In the elderly patient, the choice of anti-arrhythmic drug is often limited, and despite a close surveillance, side effects are more frequent, due to comorbidities and drug interactions [14]. The risk of drug over dosage is more frequent and a correct therapeutic adherence requires adapted entourage, especially in case of cognitive troubles. These patients have frequently another cardiac disease or conduction abnormalities affecting the sinus function or AV conduction, which are susceptible to worsen with these anti-arrhythmic drugs.

In these conditions, RF ablation, routinely used since several years [15-19], has been even more frequently used in the elderly than in the younger patients in our center, although the concerns for complications might have interfered with our indications limited to poorly tolerated episode or drugs used. The NASPE registry included 1197 patients who had an ablation for AVNRT, with success rate at 96.1% and 1% incidence of second- and third-degree AV block. The recurrence rate varied from 2 to 10% during a follow-up period ranging from a few days to several years. In case of PJT ablation with a latent bundle of Kent, the primary success approached the 95% in most series, with a 5% recurrence rate. In the 1995 NASPE registry, the incidence of complete AV block varied from 0.17% to 1% and the rates of tamponade varied from 0.13% to 1.1% after RF ablation of an accessory pathway [20, 21].

The complications are now well described, whenever they are simply related to catheterization as in our series or to the ablation, with especially the risk of AV block in case of ablation of the slow pathway [22] and tamponade, arterial embolism or hematoma in case of ablation of a latent bundle of Kent. In the elderly, these procedures are more difficult, due to a small Koch triangle in case of ablation of NRE tachycardia, or atherosclerotic lesions complicating the arterial access for left bundles of Kent. Nevertheless, the technical feasibility has already been demonstrated [2, 23], with even a larger quality of life improvement in the elderly patients than in the younger ones [24, 25]. However, complications and recurrences are more frequent in the elderly, and the junctional tachycardia ablation does not seem to be protective of occurrence of atrial fibrillation [13].

Conclusion

In conclusion, junctional tachycardia should not be missed in the elderly. They are not rare in the elderly and are often presented by another complication. A cautious medical treatment can be impeded by the presence of conduction troubles or comorbidities. If this approach is not efficacious, these subjects might take benefit from curative ablation. The complications are frequent but overall the success rate remains high.

References


