Friday, May 5, 2017 - 09:30 - 11:00

ROUND TABLE E | ΣΤΡΟΓΓΥΛΟ ΤΡΑΠΕΖΙ E’
Chairpersons: I. Goudevenos, Ant. Manolis

09:30 - 09:45 Atrial fibrillation: clinical types and outcome / Κολπική μαρμαρυγή: κλινικές μορφές και πρόγνωση
K. Papadopoulos

09:45 - 10:00 DC conversion of atrial fibrillation: Does appendage interrogation preclude embolism? / Ηλεκτρική ανάταξη κολπικής μαρμαρυγής: Διασφαλίζει η μελέτη του ωτίου τα εμβολικά επεισόδια?
G. Karatasakis

10:00 - 10:15 Left atrial appendage occlusion: Ready for use? / Αποκλεισμός του αριστερού κολπικού ωτίου: Ετοιμότητα προς χρήση?
E. Vavouranakis

10:15 - 10:30 Atrial fibrillation and coronary artery disease: strategies for combined anticoagulant-antiplatelet therapy / Κολπική μαρμαρυγή και στεφανιαία νόσος: στρατηγικές για συνδυασμένη αντιπηκτική αντιάιμοπεταλιακή αγωγή
K. Gatzoulis

10:30 - 11:00 Discussion / Σχολιασμός
Commentators: Athanasios Pipilis, P. Danias, M. Agelaki, I. Skiadas, M. Agelaki, Th. Maounis
“PRECLUDE”
prevent from happening, make impossible

• DC conversion of atrial fibrillation: Does appendage interrogation preclude embolism?

• Ηλεκτρική ανάταξη κολπικής μαρμαρυγής: Διασφαλίζει η μελέτη του ωτίου τα εμβολικά επεισόδια;

• G. Karatasakis. No Conflicts
PRECLUDE?

• NO

• INADEQUATE IMAGING MODALITIES FOR LAT

• POOR KNOWLEDGE OF LAT FORMATION TIMING AND NATURAL HISTORY (STUNNING)

• UNCERTAIN IDENTIFICATION OF EMBOLIC EVENTS
Resolution

The ability to discern as separate 2 objects along (axial) or perpendicular (lateral) to the echo beam.

Objects spaced closer than ½ SPL will not be resolved.
For echo imaging, each ultrasound pulse includes 3 cycles.

A 5 MHz transducer has a wavelength of 0.31 mm.

This means that each echo pulse is $3 \times 0.31 = 0.93$ mm long (SPL)

The axial resolution of a 5 MHz transducer is: $0.93/2 = 0.47$ mm

Important arteries are of smaller diameter
The typical lateral resolution for an unfocused transducer is approximately 2 to 5 mm.

- A focused transducer uses an acoustic lens (a curved acoustic material analogous to an optical lens) to decrease the beam diameter at a specified distance from the transducer.
ATRIAL FIBRILLATION – FLUTTER
Factors involved with LA-SEC and LA thrombus formation

- Atrial dimensions
- Valve disease (mitral, tricuspid)
- Atrial function- STUNNING
- Ventricular contractility
- Appendages function
<table>
<thead>
<tr>
<th>Event</th>
<th>Association with AF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>Increased mortality, especially cardiovascular mortality due to sudden death, heart failure or stroke.</td>
</tr>
<tr>
<td>Stroke</td>
<td>20–30% of all strokes are due to AF. A growing number of patients with stroke are diagnosed with ‘silent’, paroxysmal AF.</td>
</tr>
<tr>
<td>Hospitalizations</td>
<td>10–40% of AF patients are hospitalized every year.</td>
</tr>
<tr>
<td>Quality of life</td>
<td>Quality of life is impaired in AF patients independent of other cardiovascular conditions.</td>
</tr>
<tr>
<td>Left ventricular dysfunction and heart failure</td>
<td>Left ventricular dysfunction is found in 20–30% of all AF patients. AF causes or aggravates LV dysfunction in many AF patients, while others have completely preserved LV function despite long-standing AF.</td>
</tr>
<tr>
<td>Cognitive decline and vascular dementia</td>
<td>Cognitive decline and vascular dementia can develop even in anticoagulated AF patients. Brain white matter lesions are more common in AF patients than in patients without AF.</td>
</tr>
</tbody>
</table>

AF = atrial fibrillation; LV = left ventricular.

**Table 3** Clinical events (outcomes) affected by AF

<table>
<thead>
<tr>
<th>Outcome parameter</th>
<th>Relative change in AF patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>Death rate doubled.</td>
</tr>
<tr>
<td>Stroke (includes haemorrhagic stroke and cerebral bleeds)</td>
<td>Stroke risk increased; AF is associated with more severe stroke.</td>
</tr>
<tr>
<td>Hospitalizations</td>
<td>Hospitalizations are frequent in AF patients and may contribute to reduced quality of life.</td>
</tr>
<tr>
<td>Quality of life and exercise capacity</td>
<td>Wide variation, from no effect to major reduction.</td>
</tr>
<tr>
<td></td>
<td>AF can cause marked distress through palpitations and other AF-related symptoms.</td>
</tr>
<tr>
<td>Left ventricular function</td>
<td>Wide variation, from no change to tachycardiomypathy with acute heart failure.</td>
</tr>
</tbody>
</table>

(continued)
Atrial fibrillation

FACTS

- Prevalence of approximately 1% in the general population
- Increases with age: 6% in pts over 65 and 10% in pts over 75
- Annual risk of stroke in AF: 5% in untreated population
LAT was the only independent predictor of E-F survival
Variables included: Age, sex, HT, HF, ASA or warfarin

Stoddard et al Am Heart J 2003;145:676-82
2481 pts NO AC PRE and POST CV

**Figure 1** Incidence of TE After Cardioversion

Variation of the risk of definite thromboembolic complications (TE) after cardioversion of acute (duration <48 h) atrial fibrillation (AF), according to the chi-square automatic interaction detection analysis.
ACUTE study CV IN AF

“Can TEE replace 3 weeks of anticoagulation?”

- 1222 pts, AF>48h,
- 619 pts 1-5 days heparine or coumadin + TEE, **LAT 13.8%!!!**, excluded from CV, none embolized
- 603 pts 3 weeks coumadin (INR 2-3) **NO PT WHO HAD NO TEE WAS EXCLUDED**
- Cardioversion followed by 4 weeks coumadin for all pts
- Embolic events: 0.8% in TEE group vs 0.5% in coumadin group (p:NS)
- Bleeding: 2.9% in TEE group vs 5.5% in coumadin group (p=0.03)
TEE in AF before Cardioversion

*Ludwigshafen observational study*

- LAT was detected by TEE in 7.7% of anticoagulated pts with persistent AF. These pts were excluded.

- Post CV rate of embolism was not affected by the use of TEE (0.8% in TEE and non TEE group)

- All **1076** pts had pre CV 3 and post CV 4 weeks anticoagulation (INR 2-3)

*Seidl JACC 2002*
Spontaneous Echo Contrast

- Due to stasis of blood or a low-flow state enabling red blood cells to form aggregates.
- Most commonly seen in atria.
- Debate exists if SEC is an independent predictor of embolic events.
- Associated with:
  - Atrial fibrillation, mitral stenosis, left atrial enlargement, left atrial thrombus, prosthetic mitral valve, history of arterial embolization.
Thrombi and SEC

- Usually coincide
- LASEC more frequent
- Thrombi without LASEC: rare
- AC: reduce size and incidence of thrombi
- AC: no effect on LASEC
ATRIAL STUNNING: ΟΡΙΣΜΟΣ

• Μία παροδική, μηχανική δυσλειτουργία του αριστερού κόλπου, που συνδέεται με την ανάταξη της κολπικής μαρμαρυγής ή του κολπικού πτερυγισμού σε φλεβοκομβικό ρυθμό.
• Οδηγεί σε μία «παράδοξη» μείωση της ταχύτητας ροής του ωτίου και του κόλπου παρά την αποκατάσταση Φ/Κ ρυθμού
ΑΘΡΟΙΔΗ ΠΕΡΙΛΗΨΗ:

Η αρχική παρατήρηση, που οδήγησε στην αναγνώριση της νοσολογικής οντότητας AS ήταν κλινική. Συγκεκριμένα διαπιστώθηκε ότι η ανάταξη της κολπικής μαρμαρυγής, δημιουργούσε βραχυπρόθεσμα αύξηση των εμβολικών έπεισοδίων.

Θεωρήθηκε (λανθασμένα) ότι αυτό οφειλόταν στην ανάκτηση της κολπικής συστολής, που αποκολλούσε θρόμβους σχηματισμένους κατά την περίοδο της AF.

Αργότερα-με τη χρήση TTE και κυρίως TEE-διαπιστώθηκε ότι αμέσως μετά την ανάταξη η ταχύτητα ροής μειωνόταν, εμφανιζόταν SEC και θρόμβοι που δεν προπήρχαν.
• Εμφανίζεται (38-80%) ανεξαρτήτως μεθόδου ανάταξης υπό την προυπόθεση ότι αυτή είναι επιτυχής
• Διαθωρακική ηλεκτρική ανάταξη
• Φαρμακευτική ανάταξη
• Ενδοκαρδιακή ηλεκτρική ανάταξη
• RF ablation
• Αυτόματη ανάταξη
METHODS of ASSESSMENT of ATRIAL STUNNING

- Decreased LAA emptying velocities
- Decreased LAA emptying fraction
- Decreased transmitral A wave
- Decreased atrial contribution to the total transmitral flow (E/A)
- Appearance or worsening of LA SEC
LAA emptying velocities

- Normally (SR): $50 \pm 6\text{cm/sec} - 64 \pm 19\text{cm/sec}$
- AF+LASEC: $18 \pm 11\text{cm/sec}$
- AF: $45 \pm 23\text{cm/sec}$
- If $V < 20\text{cm/sec}$ 80% of pts have LASEC, and 17% LA Thrombus, 2.6 times greater probability of ischemic stroke
- Pre cardioversion $V: 31 \pm 15\text{cm/sec}$.
  Post: $V: 14 \pm 12\text{cm/sec}$  \cite{JACC1993}

\cite{JACC1993}
LAA emptying fraction and timing of stunning

- Pre cardioversion: 58±23%
- Post cardioversion: 30±22%
- Serious technical difficulties and low reproducibility in measuring the clotting rate of the atrial wall
- The measurement is preferred
- The maximum of the AS is observed immediately after the administration
- Starts to decrease after a few minutes, but the complete recovery of the ligation function requires 4-6 weeks
- DEN EINAİ GΝΩΣΤΟ ΠΟΤΕ ΕΜΦΑΝΙΖΟΝΤΑΙ ΟΥΤΕ ΠΟΤΕ ΑΠΟΚΟΛΛΩΝΤΑΙ ΟΙ ΘΡΟΜΒΟΙ
ΜΗΧΑΝΙΣΜΟΣ

• Ταχυ-μυοπάθεια του κόλπου. Δεν εξηγεί την σύνδεση με CV
• Επίδραση της ηλεκτρικής ενέργειας στην αιματική ροή. Δεν ισχύει.
• Ενδοκυττάρια συσσώρευση Ca λόγω των συχνών εκπολώσεων της AF. Δημιουργείται απευαισθητοποίηση των υποδοχέων Ca στους κόλπους. Μετά την ανάταξη, η επανευαισθητοποίηση συμπίπτει με την μείωση του ενδοκυτταρίου Ca και η διαδικασία παρατείνεται, δημιουργώντας ενδοκυττάριο έλλειμα Ca. Η χορήγηση αναστολέων του ασβεστίου πριν την ανάταξη μείωσε το AS.
ΜΗΧΑΝΙΣΜΟΣ

• Οι παθολογοανατομικές διαταραχές κατά την κολπική μαρμαρυγή (αύξηση του μεγέθους των μυοκυττάρων, συσσώρευση γλυκογόνου, κατάτμηση του σαρκοπλασματικού δικτύου, διάσπαση της πυρηνικής χρωματίνης) δημιουργεί ανισομέρεια στην κολπική συστολή μετά την ανάταξη.

• Ομάδες έντονα εκφυλισμένων κυττάρων ουσιαστικά ανταγωνίζονται με τα άθικτα κύτταρα που τείνουν να συσταλούν οργανωμένα
Drugs and atrial stunning

- Verapamil: Attenuates stunning when given IV prior to CV (6 pts, 16min AF) *Circ.1999*
- Isoproterenol: Given IV post CV (18 pts). LAAEV: 41cm/sec pre, 20cm/sec post cv, 27 post cv+isuprel *Int J Cardiol2002*
- Pretreatment with sotalol worsens atrial stunning *JACC1995*
- ACEI and ARB’s attenuate electrical and structural changes of atrial remodeling induced by AF
- Is atrial stunning affected by ARB administration prior to CV??
CONCLUSION:
Irbesartan significantly attenuates LA stunning after electrical cardioversion of AF. Therefore, ARBs may represent an important pharmacological supplementation in patients being prepared for cardioversion.
Management of patients presenting acutely with AF and heart failure

**Acute management**

- Cardiovert if unstable
- Anticoagulate according to stroke risk
- Normalise fluid balance with diuretics to improve symptoms
- Control rate: Initial rate target <110 bpm; stricter if persistent HF/AF symptoms
- Inhibit the renin–angiotensin–aldosterone system

**Chronic management**

- Early consideration of rhythm control
- Advanced HF therapies, including devices
- Treatment of other cardiovascular disease, especially ischaemia and hypertension

ACE = angiotensin-converting enzyme; AF = atrial fibrillation; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor neprilysin inhibition; bpm = beats per minute; HF = heart failure.

*In patients with heart failure and reduced ejection fraction. Also consider combined ARNI in patients able to tolerate an ACE Inhibitor or ARB with ongoing symptoms.*
GDL 2014 Prevention of embolism
Cardioversion for AF

**TABLE 11** Summary of Recommendations for Electrical and Pharmacological Cardioversion of AF and Atrial Flutter

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prevention of thromboembolism</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>With AF or atrial flutter for $\geq 48$ h, or unknown duration, anticoagulate with warfarin for at least 3 wk before and 4 wk after cardioversion</td>
<td>I</td>
<td></td>
<td>(320-323)</td>
</tr>
<tr>
<td>With AF or atrial flutter for $&gt;48$ h or unknown duration, requiring immediate cardioversion, anticoagulate as soon as possible and continue for at least 4 wk</td>
<td>I</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>With AF or atrial flutter $&lt;48$ h and high stroke risk, IV heparin or LMWH, or factor Xa or direct thrombin inhibitor, is recommended before or immediately after cardioversion, followed by long-term anticoagulation</td>
<td>I</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>Following cardioversion of AF, long-term anticoagulation should be based on thromboembolic risk</td>
<td>I</td>
<td></td>
<td>N/A</td>
</tr>
<tr>
<td>With AF or atrial flutter for $\geq 48$ h or unknown duration and no anticoagulation for preceding 3 wk, it is reasonable to perform TEE before cardioversion and then cardiovert if no LA thrombus is identified, provided anticoagulation is achieved before TEE and maintained after cardioversion for at least 4 wk</td>
<td>IIa</td>
<td>B</td>
<td>(164)</td>
</tr>
<tr>
<td>With AF or atrial flutter $\geq 48$ h or unknown duration, anticoagulation with dabigatran, rivaroxaban, or apixaban is reasonable for $\geq 3$ wk before and 4 wk after cardioversion</td>
<td>IIa</td>
<td>C</td>
<td>(230,324,325)</td>
</tr>
<tr>
<td>With AF or atrial flutter $&lt;48$ h and low thromboembolic risk, IV heparin, LMWH, a new oral anticoagulant, or no antithrombotic may be considered for cardioversion</td>
<td>IIb</td>
<td>C</td>
<td>(326)</td>
</tr>
</tbody>
</table>

TEE IIa ACUTE STUDY 2001 the only ref.
NOAC IIa

- In pts with AF > 48h duration or of unknown initiation oral anticoagulation should have been given for at least 3 weeks prior to C/V OR TEE should be performed to r/o LAT
- No prospective data exist about the safety of C/V under NOAC
- However in 3 large observational studies (ROCKET-AF, ARISTOTLE, and RE-LY) showed no difference in stroke and embolism rate between warfarin and NOAC
Cardioversion carries an inherent risk of stroke in non-anticoagulated patients, which is reduced substantially by the administration of anticoagulation.
Immediate initiation of anticoagulation is important in all patients scheduled for cardioversion. Patients who have been in AF for longer than 48 h should start OAC at least 3 weeks before cardioversion and continue it for 4 weeks afterwards (in patients without a need for long-term anticoagulation). OAC should be continued indefinitely in patients at risk of stroke. This practice has never been evaluated in controlled trials, but seemed safe in a large observational data set from Finland.
18th SYMPOSIUM on ECHOCARDIOLOGY

October 13-14, 2017

Under the Auspices of the Hellenic Cardiological Society

FIRST ANNOUNCEMENT
PLEASE NOTE

• NO CLINICAL PATHWAY, GUIDELINE OR ALGORITHM SUGGEST ANTICOAGULATION AND TEE

• TEE is a “IIa” alternative of 3 weeks of anticoagulation when there is no time
11.1.4 Anticoagulation in patients undergoing cardioversion

Cardioversion carries an inherent risk of stroke in non-anticoagulated patients,\textsuperscript{642} which is reduced substantially by the administration of anticoagulation.\textsuperscript{643} Immediate initiation of anticoagulation is important in all patients scheduled for cardioversion.\textsuperscript{644-646} Patients who have been in AF for longer than 48 h should start OAC at least 3 weeks before cardioversion and continue it for 4 weeks afterwards (in patients without a need for long-term anticoagulation). OAC should be continued indefinitely in patients at risk of stroke. This practice has never been evaluated in controlled trials, but seemed safe in a large observational data set from Finland.\textsuperscript{647} When early cardioversion is desired, TOE can exclude the majority of left atrial thrombi, allowing immediate cardioversion.\textsuperscript{648,649} Ongoing studies will inform about the safety and efficacy of newly initiated anticoagulation using NOACs in patients scheduled for cardioversion.
MR and LAT

- Patients without significant MR are at high risk for LA thrombus formation and subsequent embolization and represent a subgroup in whom careful anticoagulation is needed. Conversely, the presence of significant MR correlates with a lower incidence of spontaneous contrast, thrombi, and embolization.

THROMBII and LA SEC

- When LAT is present there is always SEC
- Prevalence of SEC greater
- Anticoagulation does not affect SEC
Embolic Events in Patients With Atrial Fibrillation and Effective Anticoagulation: Value of Transesophageal Echocardiography to Guide Direct-Current Cardioversion

Final Results of the Ludwigshafen Observational Cardioversion Study

Karlheinz Seidl, MD, Monika Rameken, MD, Axel Drögemüller, MD, Margit Vater, MD, Andreas Brandt, MD, Harald Schwacke, MD, Caroline Bergmeier, MD, Ralf Zahn, MD, Jochen Senges, MD, FACC

Ludwigshafen, Germany

Effective AC 1076 pts

Conventional strategy 357 pts

CV performed 355 pts

CV not performed 2 pts

Spontaneous CV 1 pt

Physician's decision 1 pt

CV performed 586 pts

CV not performed 133 pts

Spontaneous CV 25 pts

Pathologic TEE 104 pts

Physician's decision 3 pts

Patient declined 1 pt

A
Pathologic TEE inhibiting CV

- LAT 55/719 pts 7.7%
- LA-SEC (severe??) 49/719 pts 6.8%
- These 55 + 49 pts excluded from CV
- Despite the fact that 104pts did not have CV because of “TEE THROMBUS or SEC” the rate of embolism was similar in the 2 groups

TEE before direct-current cardioversion is not needed in patients with effective anticoagulation at least three weeks before cardioversion;

Seidl JACC 2002
13559 pts with n-r AF. NEJM 2003;349:1019-26
TEE and TTE: immediately before cardioversion

ELECTRICAL CARDIOVERSION

TEE and TTE: immediately after cardioversion

TEE and TTE: 2 weeks after cardioversion if patient still in sinus rhythm

TTE: 4 weeks after cardioversion if patient still in sinus rhythm
In 17% of pts with any stroke coexists atrial fibrillation.

Approximately 35% of pts with nonrheumatic AF will sustain a stroke within 10 years.
Thromboembolism and Cardioversion in AF

- Thrombus
- Atrial size
- LA SEC
- Atrial Stunning
- Duration of AF
Υπερδιάγνωση κολπικών θρόμβων με ΤΕΕ

• Η διενέργεια ΤΕΕ σε ασθενείς που έχουν πάρει πλήρη αγωγή για 3 εβδομάδες δεν μειώνει τον κίνδυνο εμβολής.

• Αντίθετα αποτρέπει την ανάταξη σε ασθενείς που θα μπορούσαν να επωφεληθούν απ’ αυτή.

• Το ΤΕΕ ενδείκνυται όταν γίνεται ανάταξη χωρίς να έχει συμπληρωθεί η αγωγή.
Figure 2: Comparison of LAAEV (cm/s) between Irbesartan group and Control group over time (pre, post, 2 wks).

- **Irbesartan group**
  - p < 0.05
  - p = NS
  - p < 0.05

- **Control group**
  - p < 0.05
  - p < 0.05
Figure 4

- **Irbesartan group**
  - 15 (75%)
  - 5 (25%)
  - **p = 0.039**

- **Control group**
  - 8 (38%)
  - 13 (62%)

Legend:
- ■ New or increased LAEC
- □ No increase
Conclusion

- Echo is important in AF
- Evaluation of the underlying pathology
- Assessment of atrial stunning
- Strategy for DC. THROMBI in LAA?
- No clinical indication for TEE in pts with adequate anticoagulation.
The aim of this study was to investigate the influence of mitral regurgitation (MR) on left atrial (LA) thrombus formation and spontaneous echocardiographic contrast in patients with rheumatic mitral valve disease. LA thrombus and spontaneous contrast are considered risk factors for embolic complications. The presence of MR has been related to a low incidence of embolization; however, its effect on thrombus formation and spontaneous contrast has not been clarified. We studied by transesophageal echocardiography 55 patients with rheumatic mitral valve disease, who were receiving anticoagulant treatment. Atrial thrombus was detected in 13 patients who had a lower incidence of significant MR (p < 0.03), a smaller regurgitant jet (p < 0.02), and a higher incidence of atrial fibrillation (p < 0.05) than the rest of the group. Spontaneous contrast was detected in 34 patients with larger atria (p < 0.006), smaller regurgitant jets (p < 0.05), a smaller mitral valve area (p < 0.008), and a higher incidence of atrial fibrillation (p < 0.002) than the rest of the group. Patients without significant MR are at high risk for LA thrombus formation and subsequent embolization and represent a subgroup in whom careful anticoagulation is needed. Conversely, the presence of significant MR correlates with a lower incidence of spontaneous contrast, thrombi, and embolization.
Embolic Events in Patients With Atrial Fibrillation and Effective Anticoagulation: Value of Transesophageal Echocardiography to Guide Direct-Current Cardioversion

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Ludwigshafen, Germany

**Transesophageal echocardiography group.** Transesophageal echocardiography before cardioversion. A thrombus was found in the left atrium in 55 (7.7%) of 719 patients. In 374 (52%) of the 719 patients, TEE revealed spontaneous echo contrast. The grade of spontaneous echo contrast was mild in 63%, moderate in 27% and severe in 10% of patients (Table 1).
09.00 - 10.30 ΣΤΡΟΓΓΥΛΟ ΤΡΑΠΕΖΙ Α’
Ο ρόλος της απεικόνισης στην πλεκτροφυσιολογία I
Πρόεδροι: Α. Κατσίβας (Αθήνα), Αντ. Μανώλης (Αθήνα)

Υπόστρωμα Κόλποι – Ωτία

09.00 - 09.15 Προπαρασκευή – διάγνωση / Ανατομία ωοειδού τρήματος -
Κ. Αγγέλη (Αθήνα)
09.15 - 09.30 Αριστερός κόλπος / Πνευμονικές φλέβες - Γ. Πούλος (Αθήνα)
09.30 - 09.45 Αξιολόγηση ουλής - Κ. Παπαδόπουλος (Αθήνα)
09.45 - 10.00 Θρομβωτικό υλικό στους κόλπους. Από τη στάση στο θρόμβο -
Γ. Καρατασάκης (Αθήνα)
10.00 - 10.30 Συζήτηση: Κ. Λιάγκας (Αθήνα), Ε. Χαμόδρακα (Αθήνα),
Ε. Καλκανδή (Αθήνα)
FLOW AND THROMBI IN THE ATRIA

G. Karatasakis

No disclosures
Figure 3
Example from the Irbesartan group
before cardioversion

Example from the control group
before cardioversion

after cardioversion

after cardioversion
• **CONCLUSION:**
Irbesartan significantly attenuates LA stunning after electrical cardioversion of AF. Therefore, ARBs may represent an important pharmacological supplementation in patients being prepared for cardioversion.