

# Archives of Cardiovascular Diseases Supplements



**S** **J** OURNÉES  
**E** UROPÉENNES de la  
**S** OCIÉTÉ  
**F** RANÇAISE de  
**C** ARDIOLOGIE

**14-17  
janvier  
2015**

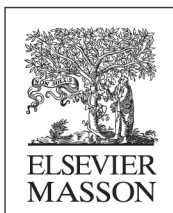
**Paris**  
**Palais des Congrès**  
2, place de la Porte-Maillot  
75017 Paris



**JOURNÉES MIREILLE BROCHIER**



Société  
Française  
de Cardiologie



# Archives of Cardiovascular Diseases Supplements



Official journal of the French Society of Cardiology

## Editor-in-Chief

Ariel A. Cohen

## Deputy editors

Yves Cottin, Yves Juillière

## Editorial board

Philippe Acar, Hélène Eltchaninoff, Jean Ferrières, Jérôme Garot, Stéphane Hatem, Bernard Jung, Pierre Lantelme, Christophe Leclercq, Philippe Ménasché

## Statistical consultant

Emanuele Di Angelantonio

## How to contact the journal

Ariel A. Cohen, Service de cardiologie, Hôpital Saint-Antoine (pavillon Lemierre), 184, rue du Faubourg-Saint-Antoine, 75571 Paris cedex 12  
Tel.: 33 (0)1 49282886 - Fax.: 33 (0)1 49282884 - E-mail: clarisse.barille@sat.aphp.fr or ariel.cohen@sat.aphp.fr

## Scientific committee

E. Aliot (France), P. Amouyel (France), M. Böehm (Germany), P. Bonhoeffer (United Kingdom), D. Bonnet (France), E. Bruckert (France), T. Carrel (Switzerland), M. Cohen (United States), A. Cribier (France), N. Danchin (France), J.-C. Daubert (France), J. Davignon (Canada), G. Derumeaux (France), E. Eeckhout (Switzerland), F. Follath (Switzerland), B. Gerber (Belgium), P. Guéret (France), G. Habib (France), A. Hagege (France), M. Komadja (France), B. Kreitmann (France), R. Lang (United States), S. Laurent (France), H. le Marec (France), J. Lima (United States), N. Ludwig (United Kingdom), Z. Mallat (France), G. Marx (United States), J.-L. Monin (France), E. Mousseaux (France), P. Nataf (France), P. Nihoyannopoulos (United Kingdom), G. Parati (Italy), L. Perrault (Canada), L. Pierard (Belgium), B. Prendergast (United Kingdom), S. Priori (Italy), D. Revel (France), V. Roger (United States), R. Rosenhek (Austria), M. Safar (France), M. Sarano (United States), E.J. Schaefer (United States), M. Scherrer Crosbie (United States), J. Schwitler (Switzerland), P. Serruys (Netherlands), M. Simoons (Netherlands), P.G. Steg (France), G. Tomaselli (United States), P. Tornos (Spain), C. Tribouilloy (France), A. Vahanian

*Archives of Cardiovascular Diseases Supplements* (ISSN 1878-6480)

Address order and payment to Elsevier Masson SAS, Service Abonnements, 62, rue Camille-Desmoulins, 92442 Issy-les-Moulineaux cedex: payment by check or credit card (CB, EuroCard, MasterCard or Visa: indicate no, and expiration date); CCP Paris no 30041 00001 1904540 H 020/70.

Subscriptions begin 4 weeks after receipt of payment and start with the first issue of the calendar year. Back issues and volumes are available from the publisher. Claims for missing issues should be made within 6 months of publication. Includes air delivery.

**Journal manager** – Brad Stucky. Tel.: (33) 01 71 16 54 42. Fax: (33) 01 71 16 51 84. E-mail: b.stucky@elsevier.com.

**Commercial manager – Advertising** – Frédérique Baudoin. Tel.: (33) 01 71 16 51 03. Fax: (33) 01 71 16 51 84. E-mail: f.baudoin@elsevier.com. Website: www.compharma.fr

**Subscriptions** – Tel.: (33) 01 71 16 55 55. Fax: (33) 01 71 16 55 88. E-mail: infos@elsevier-masson.fr

**Publisher** – Perle Bodossian. E-mail: p.bodossian@elsevier.com

**General manager and publishing director / Directeur de la Publication** – Daniel Rodriguez

## Author enquiries

The contents of each issue as well as the abstracts of the articles published in *Archives of Cardiovascular Diseases Supplements* are available on the website of Elsevier: [em-consulte.com](http://em-consulte.com)

© 2015 Elsevier Masson SAS. All rights reserved

Édité par Elsevier Masson SAS, Société par actions simplifiée à associé unique, au capital social de 47 275 384 € – RCS Nanterre B 542 037 031

Siège social : 62 rue Camille-Desmoulins, 92130 Issy-les-Moulineaux

Actionnaire : Elsevier Holding France

This journal and the individual contributions contained in it are protected under copyright by Elsevier Masson SAS, and the following terms and conditions apply to their use:

## Photocopying

Single photocopies of single articles may be made for personal use as allowed by national copyright laws. Permission of the Publisher and payment of a fee is required for all other photocopying, including multiple or systematic copying, copying for advertising or promotional purposes, resale, and all forms of document delivery. Special rates are available for educational institutions that wish to make photocopies for non-profit educational classroom use. Individuals may obtain the necessary permission and pay the corresponding royalties at the Centre français d'exploitation du droit de la copie (20, rue des Grands-Augustins, 75006 Paris, France).

## Derivative works

Subscribers may reproduce tables of contents or prepare lists of articles including abstracts for internal circulation within their institutions. Permission of the Publisher is required for resale or distribution outside the institution. Permission of the Publisher is required for all other derivative works, including compilations and translations.

## Electronic storage or usage

Permission of the Publisher is required to store or use electronically any material contained in this journal, including any article or part of an article. Except as outlined above, no part of this publication may be reproduced, stored in a retrieval system or transmitted in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, without prior written permission of the Publisher. Address permissions requests to the publisher.

## Notice

No responsibility is assumed by the Publisher for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions or ideas contained in the material herein. Because of rapid advances in the medical sciences, in particular, independent verification of diagnoses and drug dosages should be made. Although all advertising material is expected to conform to ethical (medical) standards, inclusion in this publication does not constitute a guarantee or endorsement of the quality or value of such product or of the claims made of it by its manufacturer.

Imprimé en France par Imprimerie de Champagne, 52 Langres. CPPAP: en cours  
Dépôt légal à parution.

**Methods and Results:** From 1998 to 2013, 10 cases of pulmonary embolism were found amongst BD patients in the department of internal medicine of Hedi Chaker Hospital. All these patients fulfilled International Study Group criteria for BD.

10 patients (all men, mean aged at  $29.9 \pm 9.08$  years) were diagnosed as having pulmonary embolism. This was inaugural in 7 cases. Peripheral venous thrombosis was present in 50% of patients; cardiac thrombosis was presented in 2 cases and pulmonary aneurysm in 2 patients. Pulmonary infarction has been noted in 3 cases. Pulmonary artery pressure may be elevated, and may indicate a poor prognosis. Mediastinal lymphadenopathy and mild pleural and pericardial effusions was also observed in 1 case. Protein C, protein S, antithrombin III and homocysteinemia levels were normal in all cases. One patient was positive for IgG anticardiolipin antibody. All our patients were treated successfully by anticoagulation therapy combined with high dose prednisone, colchicine and intravenous cyclophosphamide in 2 patients. After a mean follow-up of  $106 \pm 49$  months, 4 patients had a recurrence of pulmonary embolism and only 1 patient was dead.

**Conclusion:** Pulmonary embolism is one of the severe and worst prognostic manifestations of the BD. Our knowledge about pulmonary complications of Behçet's disease continues to evolve, but we need controlled trials for the management of the disease. The main goal should be to elucidate the pathogenesis and standardize the management according to the underlying pathologic process.

## 0299

### Interleukins 33 and 1 $\beta$ , left ventricular geometry and diastolic dysfunction in hypertensive patients with obesity

Oleksii Honchar

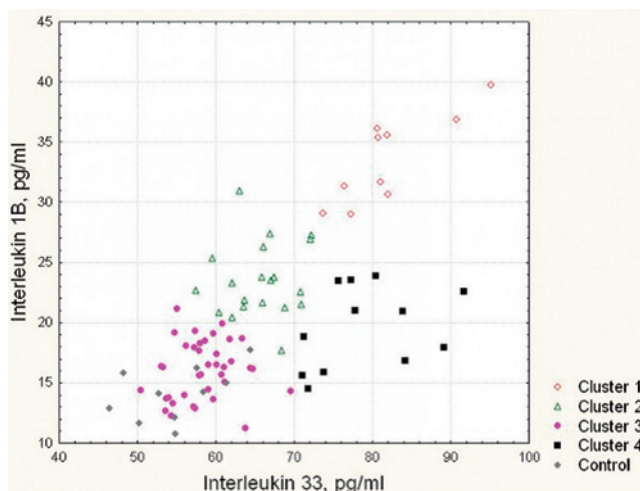
Kharkiv National Medical University, Propedeutics To Internal Medicine No.1, Kharkiv, Ukraine

**Purpose:** To investigate interrelations between interleukin 33 (IL-33) and 1 $\beta$  (IL-1 $\beta$ ) serum levels, left ventricular (LV) remodeling and diastolic dysfunction (DD) in hypertensive patients with obesity.

**Materials and methods:** 80 hypertensive patients (51 obese) underwent transthoracic echocardiography. LV geometric pattern by A.Ganau, E/A and E/E' ratios, pulmonary wedge pressure (PWP) by S.Nagueh were calculated. IL-33 and IL-1 $\beta$  serum levels were estimated using ELISA.

**Results:** IL-33 and IL-1 $\beta$  were higher in hypertensive patients ( $p < 0.001$ ), independently of BMI, and formed 4 clusters (see pic.) Cluster 1 was associated with the highest LV myocardial mass index (MMI) ( $160.5$  ( $142.8$ ;  $185.8$ )  $g/m^2$ ,  $p < 0.05$ ), highest prevalence of LV hypertrophy (LVH) (100.0%, 90.0% of concentric LVH), moderate decrease in E' velocity ( $9.95$  ( $8.32$ ;  $10.60$ )  $cm/sec$ ), relatively low PWP ( $9.23$  ( $8.83$ ;  $13.03$ )  $mmHg$ ) and 70.0% prevalence of LVDD (60.0% of type I). Cluster 2 had LVMMI of ( $116.9$  ( $104.4$ ;  $163.1$ )  $g/m^2$ ), 55.0% prevalence of LVH plus 30.0% of concentric remodeling, lowest E' ( $7.68$  ( $6.50$ ;  $9.67$ )  $cm/sec$ ,  $p < 0.01$ ), highest PWP ( $12.26$  ( $10.72$ ;  $13.12$ )  $mmHg$ ,  $p < 0.05$ ) and highest rate of DD (85.0%, 70.0% of type I). Cluster 4 was associated with MMI of  $121.4$  ( $111.7$ ;  $140.5$ )  $g/m^2$ , 66.7% rate of LVH (equal for concentric and eccentric variants), highest values of E' ( $11.04$  ( $9.49$ ;  $12.00$ )  $cm/sec$ ), lowest PWP ( $9.07$  ( $7.04$ ;  $11.51$ )  $mmHg$ ) and lowest prevalence of LVDD (66.7%, 50.0% of type I). Cluster 3 ( $p > 0.05$  vs control group) had intermediate characteristics of mentioned parameters.

**Conclusion:** Significant increase in IL-33 and IL-1 $\beta$  levels in hypertensive patients independently of BMI was revealed. Increase in both cytokines' levels was associated with highest rates of LVH and DD. Prevalent increase in IL-1 $\beta$  was connected to the worst state of diastolic function despite low rates of hypertrophy. Prevalent increase in IL-33 had the most favorable influence on the severity of LVH as well as diastolic filling (figure above).



Abstract 0299 – Figure

## 0383

### The peculiarities of structural and functional state of myocardium and daily blood pressure profile in hypertensive patients with diabetes mellitus

Lesya Bobronnikova, Iryna Ilchenko, Maryna Filonenko

Kharkiv National Medical University, Kharkiv, Ukraine

**Purpose:** To establish the relationship between structural and functional parameters of the left ventricle (LV) and daily blood pressure (BP) profile in patients with arterial hypertension (AH) of II stage comorbid with type 2 diabetes mellitus (DM2).

**Methods:** 68 patients (32 men and 36 women, mean age  $49.6 \pm 5.2$  years); 32 – with isolated hypertension (group 1), 36 – with hypertension and DM2 (group 2) were examined. We evaluated the data of transthoracic echocardiography and parameters of ambulatory blood pressure monitoring.

**Results:** Patients of the 1<sup>st</sup> group had a significant increase of the LV posterior wall dimension (LVPWd) – 14% ( $p < 0.05$ ) and left atrial size – 17% ( $p < 0.05$ ). In patients of the 2<sup>nd</sup> group a direct correlation between BP variability and LVPWd ( $r = 0.67$ ;  $p < 0.05$ ) was revealed; as well as inverse correlation between daily average BP variability and a maximum velocity of early wave of mitral inflow ( $r = -0.51$ ;  $p < 0.05$ ).

**Conclusions:** In patients with hypertension comorbid with type 2 diabetes mellitus the following changes can be observed: the increase of BP variability, predominance of concentric left ventricular hypertrophy (65% of patients) over the eccentric one (29% patients) with the formation of diastolic dysfunction and impaired LV relaxation (51% of patients).

## 0430

### Features of carbohydrate metabolism, cytokines activity and I/D gene ACE polymorphism at patients with arterial hypertension and overweight

Mariia Kulikova, Tatiana Ashcheulova

Kharkiv National Medical University, Propedeutics To Internal Medicine, Kharkiv, Ukraine

The purpose of our research was to study features of carbohydrate metabolism, activation of the pro-inflammatory cytokine-interleukin-18 (IL-18) and anti-inflammatory cytokine-interleukin-10 (IL-10), I/D gene ACE polymorphism at patients with arterial hypertension and overweight. Design and Methods: 103 hypertensive patients were examined, which have been divided

into 2 groups depending on body mass index (BMI): 1st group-24 patients with BMI less than 25, the 2nd group-79 patients with BMI more than 25. The control group – 10 almost healthy subjects.

**Results:** At patients of the 2nd group reliable increase of SBP and DBP is noted-162.5 mmHg and 100.0 mmHg,  $p < 0.05$ . The analysis of indicators of a carbohydrate exchange showed HbA1c increase at patients of the 1st and 2nd groups in comparison with control-6.97 (3.9-11.6)%, 6.5 (3.7-12.4)% and 5.1 (3.6- 8.1)% respectively,  $p = 0.0321$ . In the 1st group ID genotype-9 patients (35.62%), DD genotype – 13 patients (53.42%); in the 2nd group – ID genotype-37 patients (46.61%), DD genotype-27 patients (30.77%),  $p = 0.03821$ . Besides, at patients of the 2nd group statistically significant increase of level anti-inflammatory IL-10-89.3 (61.3-97.5) pg/ml in comparison with the 1st

group and group of control-79.5 (58.8-93.0) pg/ml and 82.0 (58.8-97.5) pg/ml respectively,  $p = 0.0001$  against increase of level pro-inflammatory IL-18 in both groups (173.0 (110.0-210.0) pg/ml 1st group and 176.0 (125.0-205.0) pg/ml – the 2nd group,  $p = 0.0001$ ) was noted.

**Conclusion:** The obtained data allow to assume that at patients with arterial hypertension and overweight against prevalence of adverse genotypes of ID and the DD polymorphism of I/D gene ACE polymorphism heavier current of arterial hypertension is observed that is characterized by higher of blood pressure levels and of a carbohydrate metabolism parameters such as HbA1c is noted. Increase of level IL-10 against increase IL-18 at patients with arterial hypertension and overweight, probably, has compensatory character.

