## 本會成立宗旨：

以非營利為目的之社會團體，以結合國內外熱心人士從事血脂異常及其相關疾病之衛生教育、學術研究、疾病預防及臨床服務等工作為宗旨。

## 本會成立目標：

- 舉辦衛教活動
- 推動學術研究
- 舉辦學術演講及討論會
- 參與國際相關組織及活動
- 結合熱心人士及團體以推動血脂異常之防治工作
- 其他與章程所訂宗旨及任務相關事項

## 委員會成員名單：

理 事 長：陳文鍾

常務理事：陳茂元、許勝雄

理　　事：陳明豐、葉宏一、陳肇文、林幸榮、胡啟民
殷偉賢、王國陽、李源德、王宗道、李啟明

常務監事：廖朝崧

監　　事：王水深、林中生

秘 書 長：吳造中

秘書(聯絡人)：陳素文

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# 台灣血脂衛教協會 *Taiwan Association of Lipid Educators*

**日期：102 年 12 月 28 日 ( 六 ) 14:00-17:00**

地點：[ 台北 ] 國立台灣大學公共衛生學院公衛大樓 ( 台北市中正區徐州路 17 號 )

## Holistic Care for the Patients with Cardiovascular Diseases: Controversies and Issues in 2013 (Part II)

| Time | Topic | Speaker | Moderator |
|---|---|---|---|
| 14:00-14:05 | **Opening Remarks** | | 吳造中 教授 |
| 14:05-14:35 | From AF management to stroke prevention | 謝敏雄 主任 | |
| 14:35-15:05 | Choices among antiplatelets-A permanent trade off between event prevention and bleeding risk | 李貽恆 主任 | |
| 15:05-15:20 | **Discussion** | | |
| 15:20-15:35 | **Coffee Break** | | |
| 15:35-15:40 | **Opening Remarks** | | 殷偉賢 主任 |
| 15:40-16:05 | Debate: Which one is better - Factor II or Xa inhibitor? Factor II inhibitor is better! | 邱昱偉 醫師 | |
| 16:05-16:30 | Debate: Which one is better - Factor II or Xa inhibitor? Factor Xa inhibitor is better! | 蔡佳醍 醫師 | |
| 16:30-16:35 | Rebuttal – Factor II inhibitor | 邱昱偉 醫師 | |
| 16:35-16:40 | Rebuttal – Factor Xa inhibitor | 蔡佳醍 醫師 | |
| 16:40-16:55 | **Discussion** | | |
| 16:55-17:00 | **Closing Remarks** | | |

**日期：102 年 12 月 29 日 ( 日 ) 08:50-16:45**

地點：[ 台北 ] 國立台灣大學公共衛生學院公衛大樓 ( 台北市中正區徐州路 17 號 )

## Holistic Care for the Patients with Cardiovascular Diseases: Controversies and Issues in 2013 (Part II)

| Time | Topic | Speaker | Moderator |
|---|---|---|---|
| 08:50-09:00 | 會員大會 | | 陳文鍾 理事長 |
| 09:00-09:05 | **Opening Remarks** | | 陳文鍾 理事長 |
| 09:05-09:35 | Insight and implication from NHI guideline relaxation to optimal statin therapy | 蘇正煌 主任 | |
| 09:35-10:00 | Debate: Is new-onset DM an issue for statin therapy? - Pros | 黃金洲 醫師 | |
| 10:00-10:25 | Debate: Is new-onset DM an issue for statin therapy? - Cons | 廖本智 醫師 | |
| 10:25-10:30 | Rebuttal – Pros | 黃金洲 醫師 | |
| 10:30-10:35 | Rebuttal –-Cons | 廖本智 醫師 | |
| 10:35-10:50 | **Discussion** | | |
| 10:50-11:05 | **Coffee Break** | | |
| 11:05-11:10 | **Opening Remarks** | | 吳造中 教授 |
| 11:10-11:40 | LDL-C goal achievement: Role of aggressive approach with low dose dual inhibition | 王治元 醫師 | |
| 11:40-12:10 | Beyond LDL-C, how can we achieve comprehensive lipid goal in Ty2D with mixed-type dyslipidemia? | 賴超倫 醫師 | |
| 12:10-12:40 | Diet therapy and life style modification for weight reduction in obesity | 陳茂元 教授 | |
| 12:40-12:55 | **Discussion** | | |
| 12:55-13:45 | **Lunch** | | |
| 13:45-13:50 | **Opening Remarks** | | 王水深 教授 |
| 13:50-14:20 | The strategy for hypertension management – How to get more patients to goal? | 吳卓鍇 醫師 | |
| 14:20-14:50 | ESC/ESH guideline update: evidence-based recommendations for establishing BP control – the role of CCBs | 蔡適吉 醫師 | |
| 14:50-15:05 | **Discussion** | | |
| 15:05-15:20 | **Coffee Break** | | |
| 15:20-15:25 | **Opening Remarks** | | 陳明豐 教授 |
| 15:25-15:55 | Recent advance for the treatment of T2DM -- Comparison between DPP-4 inhibitor and GLP-1 receptor agonists | 胡啓民 教授 | |
| 15:55-16:25 | From Optimal Glycemic Control to CVD protection | 黃兆山 醫師 | |
| 16:25-16:40 | **Discussion** | | |
| 16:40-16:45 | **Closing Remark** | | |

《最終節目表依大會公告爲準》

# 台灣血脂衛教協會 *Taiwan Association of Lipid Educators*

**日期：103 年 1 月 19 日 ( 日 ) 08:55-17:15**

**地點：[ 台中 ] 中山醫學大學 - 正心樓一樓 0112 教室 (40201 台中市南區建國北路一段 110 號 )**

## Holistic Care for the Patients with Cardiovascular Diseases: Controversies and Issues in 2013 (Part II)

| Time | Topic | Speaker | Moderator |
|---|---|---|---|
| 08:55-09:00 | **Opening Remarks** | | 林中生 校長 |
| 09:00-09:30 | Insights and Implications from NHI Lipid Guideline Change | 林中生 校長 | 吳造中 教授 |
| 09:30-10:00 | LDL-C Goal Attainment: Role of Aggressive Approach with Low Dose Dual Inhibition | 張詩聖 醫師 | 林中生 校長 |
| 10:00-10:30 | Balancing Efficacy and Safety in dyslipidemia management in CVD and diabetic patients | 蘇矢立 主任 | |
| 10:30-10:40 | **Discussion** | | |
| 10:40-10:50 | **Coffee Break** | | |
| 10:50-10:55 | **Opening Remarks** | | 王宗道 教授 |
| 10:55-11:25 | Treatment for DM dyslipidemia --- Initiating drug from statin! | 蘇峻弘 主任 | |
| 11:25-11:55 | Beyond LDL-C, how can we achieve comprehensive lipid goal in Ty2D with mixed-type dyslipidemia? | 許智能 主任 | |
| 11:55-12:05 | **Discussion** | | |
| 12:05-12:55 | **Lunch** | | |
| 12:55-13:00 | **Opening Remarks** | | 吳造中 教授 |
| 13:00-13:30 | From AF management to stroke prevention | 王俊傑 主任 | |
| 13:30-14:00 | How to personalize antiplatelet therapy for your ACS patients | 李貽恆 主任 | |
| 14:00-14:25 | Debate: Which one is better - Factor II or Xa inhibitor? Factor II inhibitor is better! | 翁國昌 教授 | |
| 14:25-14:50 | Debate: Which one is better - Factor II or Xa inhibitor? Factor Xa inhibitor is better! | 白培英 醫師 | |
| 14:50-14:55 | Rebuttal – Factor II inhibitor | 翁國昌 教授 | |
| 14:55-15:00 | Rebuttal – Factor Xa inhibitor | 白培英 醫師 | |
| 15:00-15:10 | **Discussion** | | |
| 15:10-15:20 | **Coffee Break** | | |
| 15:20-15:25 | **Opening Remark** | | 王國陽 教授 |
| 15:25-15:55 | From Optimal Glycemic Control to CVD protection | 李奕德 醫師 | |
| 15:55-16:25 | Current Challenges in Management of morbidity and Mortality in T2DM "nephropathy" | 吳允升 醫師 | |
| 16:25-16:55 | The strategy for hypertension management –How to get more patients to goal? | 林維文 主任 | |
| 16:55-17:10 | **Discussion** | | |
| 17:10-17:15 | **Closing Remark** | | |

註：台中 - 免費停車 詳情請上網 http://www.lipid.com.tw。

《最終節目表依大會公告爲準》

# 台灣血脂衛教協會 *Taiwan Association of Lipid Educators*

**日期：103 年 1 月 26 日 ( 日 ) 08:55-17:45**

**地點：[ 高雄 ] 高雄醫學大學附設中和紀念醫院 - 高醫啓川大樓 6F 第一會議室**
**( 高雄市三民區自由一路 100 號 )**

| Holistic Care for the Patients with Cardiovascular Diseases: Controversies and Issues in 2013 (Part II) | | | |
|---|---|---|---|
| **Time** | **Topic** | **Speaker** | **Moderator** |
| 08:55-09:00 | **Opening Remarks** | | 許勝雄 院長 |
| 09:00-09:30 | Insights and Implications from NHI Lipid Guideline Change | 朱志生 主任 | |
| 09:30-10:00 | LDL-C Goal Attainment: Role of Aggressive Approach with Low Dose Dual Inhibition | 李貽恒 主任 | |
| 10:00-10:30 | Balancing Efficacy and Safety in dyslipidemia management in CVD and diabetic patients | 謝棟漢 醫師 | |
| 10:30-10:40 | **Discussion** | | |
| 10:40-10:50 | **Coffee Break** | | |
| 10:50-10:55 | **Opening Remarks** | | 吳造中 教授 |
| 10:55-11:25 | Treatment for DM dyslipidemia --- Initiating drug from statin! | 曾維功主任 | |
| 11:25-11:55 | Beyond LDL-C, how can we achieve comprehensive lipid goal in Ty2D with mixed-type dyslipidemia? | 朱志勳 主任 | |
| 11:55-12:05 | **Discussion** | | |
| 12:05-12:55 | **Lunch** | | |
| 12:55-13:00 | **Opening Remarks** | | 許寬立 部長 |
| 13:00-13:30 | From AF management to stroke prevention | 王俊傑 教授 | |
| 13:30-14:00 | Turn-key solutions in ACS management | 翁國昌 教授 | |
| 14:00-14:30 | Choices among antiplatelets – An Review of Antiplatelet Agents | 邱俊仁 醫師 | |
| 14:30-14:55 | Debate: Which one is better - Factor II or Xa inhibitor? Factor II inhibitor is better! | 趙嘉倫 副院長 | |
| 14:55-15:20 | Debate: Which one is better - Factor II or Xa inhibitor? Factor Xa inhibitor is better! | 郭風裕 醫師 | |
| 15:20-15:25 | Rebuttal – Factor II inhibitor | 趙嘉倫 副院長 | |
| 15:25-15:30 | Rebuttal – Factor Xa inhibitor | 郭風裕 醫師 | |
| 15:30-15:40 | **Discussion** | | |
| 15:40-15:50 | **Coffee Break** | | |
| 15:50-15:55 | **Opening Remark** | | 賴文德 院長 |
| 15:55-16:25 | Current Challenges in Management of morbidity and Mortality in T2DM "nephropathy" | 吳允升 醫師 | |
| 16:25-16:55 | From Optimal Glycemic Control to CVD protection | 李美月 醫師 | |
| 16:55-17:25 | The strategy for hypertension management –How to get more patients to goal? | 方志元 主任 | |
| 17:25-17:40 | **Discussion** | | |
| 17:40-17:45 | **Closing Remark** | | |

預計申請學分：中華民國醫師公會全國聯合會 / 中華民國心臟學會 / 內科醫學會 / 台灣家庭醫學醫學會 / 台灣神經學學會 / 中華民國糖尿病學會 / 台灣老人暨老年醫學會 / 台灣腎臟醫學會 / 中華民國重症醫學會…等。

註：台中 - 免費停車 詳情請上網 http://www.lipid.com.tw/，台北 / 高雄：停車請自理。

《最終節目表依大會公告爲準》

# 理事長的話

各位同好：

有鑑於心血管疾病為國人十大死因之第二位，而心血管疾病相關知識、診斷及治療之研究日新月異，推陳出新。本會之宗旨包括推動專業人員之繼續教育，實有必要針對近期發表之相關學術論述與爭議，舉辦心血管全人照護研討會，以釐清相關之觀念。

有鑑於本會歷次在台北舉辦之相關研討會均得到相當大的迴響與佳評，因此本會擬每半年舉辦一次並擴大至北中南三區，以孚知識進展之時效性與普及性。爰此，本會擬於2013年12月28、29日於國立台灣大學公共衛生學院公衛大樓，2014年1月19日於中山醫學大學及1月26日高雄醫學大學附設中和紀念醫院，再次舉辦心血管疾病照護相關議題之整合性學術研討會，歡迎各位同好踴躍參加，共襄盛舉。

理事長

陳文鍾 敬邀

# 簡　　歷

**姓名：**謝敏雄 主任 (Ming-Hsiung Hsieh)

**現職：**
心臟內科 主任
萬芳醫院心臟內科專任主任
臺北醫學大學副教授
中華民國心臟醫學會電生理及節律器委員

**主治項目：**
心律不整電氣生理燒灼數、冠狀動脈心臟病及心導管手術、高血壓、心臟衰竭、瓣膜性心臟病、 心律調節器置放術

**學歷：**
高雄醫學院醫學系
國立陽明大學臨床醫學研究所博士班

**經歷：**
臺北榮民總醫院內科住院醫師
臺北榮民總醫院心臟內科總醫師

# From AF Management to Stroke Prevention

謝敏雄 主任

Patients with Atrial Fibrillation (AF) are at much higher risk for hospitalization and mortality and the quality of life is compromised by suffering from debilitating symptoms, including palpitations, chest pain, dyspnea, fatigue or lightheadedness. In addition to recurrent hospitalization, AF patients have an around 5-fold increased risk of stroke and 1 in every 6 strokes occurs in a patient with AF. Compared to patients who maintain sinus rhythm, recurrent hospitalization occurs much higher frequently in patients at AF. Patients with AF can benefit from rhythm management in improving unpleasant symptoms or increase exercise capacity. However, the AFFIRM and other prospective clinical trials did not demonstrate traditional AADs could make a difference in either mortality or hospitalization, compared with rate control therapy. The benefits of AADs would be offset by the risks of adverse events. Particular safety concerns for individual AADs should, therefore, be paid more attentions.

Dronedarone, a newer generation of AAD, is effective in the maintenance of sinus rhythm proved by several international clinical studies and being used by a growing number of patients worldwide. ATHENA study proved Dronedarone's efficacy in the decrease of cardiovascular hospitalization and cardiovascular mortality rates in patients with paroxysmal or persistent AF. No AAD before dronedarone had been shown to reduce cardiovascular (CV) outcomes, including unplanned CV hospitalizations and CV mortality. Dronedarone has also been demonstrated to significantly reduce the risk of first AF recurrence and stroke by 25% and 34%, respectively, while it's not associated with serious safety concerns.

Anticoagulation with warfarin is highly effectively in reducing stroke rates. For AF patients with at least one risk factor for CV events and above 65 years old, novel oral anticoagulants could also be considered for the prevention of stroke events. According to the ACTIVE A study results, the addition of clopidogrel to aspirin is superior to the use of aspirin alone and could be reserved for patients with AF in whom oral anticoagulation is considered unsuitable due to patient preference or the physician's assessment of the patient's ability to safely sustain anticoagulation.

# 簡　　歷

**姓名：**李貽恆 教授

**現職：**

成大心臟血管科主治醫師兼主任

**學歷：**

09/1981-06/1988　高雄醫學大學醫學系醫學士

09/1996-06/2000　國立成功大學醫學院基礎醫學研究所博士

**經歷：**

國立台灣大學醫學院附設醫院內科部住院醫師 1990 年 9 月至 1995 年 8 月

國立台灣大學醫學院附設醫院內科部主治醫師 1995 年 8 月至 1996 年 8 月

國立成功大學醫學系內科學科講師 1996 年 8 月至 2000 年 8 月

國立成功大學醫學系內科學科副教授 2000 年 8 月至 2008 年 8 月

國立成功大學醫學系內科學科教授 2008 年 8 月 - 迄今

中華民國心臟學會 副秘書長

中華民國血脂及動脈硬化學會 理事

**研究主題：**

動脈硬化的病態生理學

高血壓、高脂血症、冠狀動脈心臟病的分子遺傳研究

血管生物學

**研究成果：**

已發表八十多篇研究論文於國 SCI 醫學期刊，包括 *Journal of the American College of Cardiology, European Heart Journal, Chest, American Journal of Cardiology, Thrombosis and Haemostasis* 等

# Choices Among Antiplatelets - A Permanent Trade Off Between Event Prevention and Bleeding Risk

李貽恆 教授

Acute coronary syndromes (ACS) require rapid intervention with pharmacologic therapies to treat and prevent coronary thromboembolism, as well as to support revascularization procedures. Patients with ACS have a heterogeneous clinical presentation that often requires a complex treatment continuum. The inhibition of platelet function by antiplatelet therapy determines the improvement of the survival of patients. Given abundant clinical evidence, international clinical practice guidelines have recommended for years dual therapy with aspirin and clopidogrel is a standard of care for reducing the occurrence of cardiovascular events in ACS patients with or without ST-segment elevation.

Based on the medical evidences, antiplatelet agents exhibit an initial delay in the onset of their action in ST-segment–elevation myocardial infarction patients, compared to the results in patients with stable coronary artery disease. The possible reasons include the highly thrombotic milieu and impaired absorption in STEMI setting. There are also evidences showing that major bleeding is a relatively frequent non-cardiac complication of contemporary therapy for ACS and it is associated with a poor hospital prognosis. Correctly interpreting and having a closer look at the available evidences are critical and will help to correctly evaluate the efficacy and safety of antiplatelet agents and understand the clinical implications of those trials now and for the future. Considering the available evidences, it will further identify the role of P2Y12 inhibition and the position of each marketed antiplatelet agent in the management of the broad spectrum of ACS from the acute to the long-term setting.

# CURRICULUM VITAE

**Name:** Chiu, Yu-Wei, M.D. （邱昱偉）
**Sex:** Male
**Date of Birth:** January 28, 1974
**Mailing Address:** Cardiovascular Center
Far Eastern Memorial Hospital
21, Section 2, Nan-Ya South Road
Pan-Chiao, New Taipei City, Taiwan, 220
**Tel:** (886)2-89667000 ext 4277

## CURRENT POSITION:

Attending Physician, Section of Cardiology, Cardiovascular Center, Far Eastern Memorial Hospital

## EDUCATION:

| | |
|---|---|
| September, 1992 - June, 1999 | Medical College, National Taiwan University, Taipei, Taiwan |
| September, 2012 - | PhD candidate of College of Medicine, Graduate Institute of Clinical Medicine, National Taiwan University, Taipei, Taiwan |

## MAJOR WORK:

| | |
|---|---|
| 1999-7-1 - 2002-6-30 | Resident Physician, Department of Internal Medicine, National Taiwan University Hospital, Taipei |
| 2002-7-1 - 2004-6-30 | Fellowship training in Division of Cardiology, Department of Internal Medicine, National Taiwan University Hospital, Taipei |
| Since 2004-7 | Attending Physician, Section of Cardiology, Cardiovascular Center, Far Eastern Memorial Hospital |
| Since 2013-8 | Clinical assistant professor, Far Eastern Memorial Hospital |

## BOARD OF CERTIFICATE:

| | |
|---|---|
| 1999 | General License of Physician |
| 2002 | Specialist of Internal Medicine |
| 2004 | Specialist of Cardiology |
| 2006 | Specialist of Critical Care Medicine |
| 2007 | Specialist of Interventional Cardiology |
| 2009 | 心臟專科指導醫師 |

# Debate: Which one is better - Factor II or Xa inhibitor? Factor II inhibitor is better!

邱昱偉 醫師

# 簡　　歷

**姓名：**蔡佳醍 醫師

**個人專長：**
心臟內科
冠狀動脈心導管手術
心律不整電燒手術
心律調節器及體內去顫器植入

**現任職務：**
台大醫院內科部主治醫師
國立台灣大學醫學院內科專任副教授

**主要學歷：**
國立台灣大學醫學院醫學系畢業

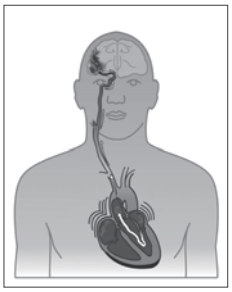
**過去成果：**
蔡醫師專精於一般內科、心臟內科、冠狀動脈心臟病、心律不整等各種心臟病，且精通冠狀動脈心導管手術及支架植入、心律不整電燒手術、心律調節器及體內去顫器植入等各種心臟病相關手術。在醫學研究方面，蔡醫師發表研究結果於眾多國內外知名期刊，且曾獲得中華國民心臟學會青年醫師研究獎、中華民國心臟學會最高榮譽國內學人丁農獎、台大醫院傑出研究獎、台大醫學院傑出著作獎、青杏醫學獎、國科會吳大猷獎及國科會最高榮譽傑出研究獎。

## Xarelto® (Rivaroxaban): a novel, oral, direct Factor Xa Inhibitor for prevention of stroke and embolism in atrial fibrillation patients



## AF and stroke

- Stroke is the most serious ongoing risk associated with AF[1]
- In patients with AF, blood clots tend to form in the atria, particularly within the left atrial appendage, as a result of abnormal blood flow and pooling[2,3]
- These clots may travel to the brain, causing an ischaemic stroke[2]
- Approximately 20% of ischaemic strokes are caused by blood clots that originate in the heart (cardioembolic); of these, AF is the most common cause[4]



1. Wolf PA *et al*, 1991; 2. National Heart Lung and Blood Institute. 2011; 3. Fuster V *et al*, 2006; 4. Paciaroni M *et al*, 2007.



2

## AF has serious consequences

- Independent risk factor for stroke
  - ~Fivefold increased risk[1]
  - One in six strokes occur in patients with AF[2]
  - AF-related strokes are typically more severe than strokes due to other aetiologies[3,4]
  - Stroke risk persists even in patients with asymptomatic or intermittent AF[5]
- Independent risk factor for mortality
  - ~Twofold increased risk[6]
- Independent risk factor for heart failure
  - Heart failure further aggravates AF, worsening overall prognosis[7]

1. Wolf PA *et al*, 1991; 2. Fuster V *et al*, 2006; 3. Lin HJ *et al*, 1996; 4. Jørgensen HS *et al*, 1996; 5. Page RL *et al*, 2003; 6. Benjamin EJ *et al*, 1998; 7. Wang T *et al*, 2003.



3

## $CHADS_2$ and $CHA_2DS_2$-VASc

| Risk factors | Points assigned | |
|---|---|---|
| | $CHADS_2$ | $CHA_2DS_2$-VASc |
| Age (years) | | |
| 65–74 | | +1 |
| ≥75 | | +2 |
| >75 | +1 | |
| Congestive heart failure | +1 | +1 |
| Hypertension | +1 | +1 |
| Diabetes mellitus | +1 | +1 |
| Stroke/TIA | +2 | +2 |
| Vascular disease* | | +1 |
| Female gender | | +1 |
| | Cumulative score: 0–6 | Cumulative score: 0–9 |

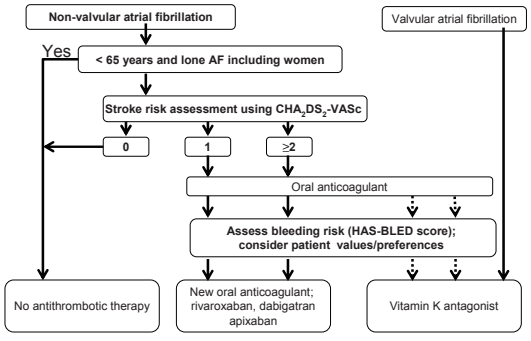
*MI, peripheral artery disease or aortic plaque

Lip GY *et al*, 2010.



4

## ESC 2012 guidelines: selection of patients for OACs




Camm AJ *et al. Eur Heart J* 2012 Slide line preferred; dotted line alternative



## The limitations of VKA therapy

- Significant inter- and intra-patient variability in dose–response,[1] due to:
  - Co-morbid conditions
  - Genetic polymorphisms
  - Numerous interactions with food and concomitant drugs
  - Unpredictable pharmacology
- Narrow therapeutic window (INR 2–3)[1]
  - Regular coagulation monitoring and dose adjustments required
  - Failure to stay within the therapeutic range increases the risk of stroke or adverse bleeding events[2]
- Underuse[2–4]
  - Fear of haemorrhage;[5] intracranial haemorrhage is the most devastating bleeding event[4]
  - Particularly in elderly patients because of high perceived risk of bleeding versus possible benefits[5]

1. Ansell J *et al*, 2008; 2. Nieuwlaat R *et al*, 2007; 3. Ogilvie IM *et al*, 2010; 4. Nieuwlaat R *et al*, 2005; 5. Waldo A *et al*, 2005.



6

## Novel oral anticoagulants

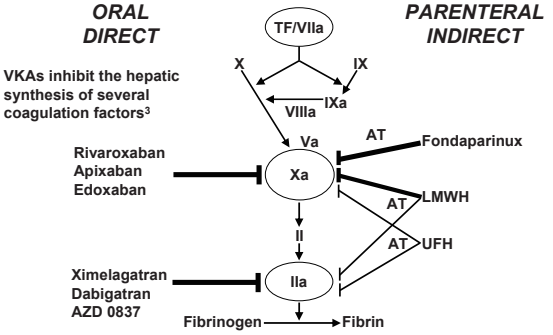
- Novel OACs in development fulfil the following unmet needs:
  - Predictable pharmacology
    - Target a single coagulation factor
    - Fewer interactions with food or concomitant drugs
    - Can be used at a fixed dose
    - No requirement for routine coagulation monitoring
  - Improved benefit–risk profile compared with VKAs

Bauer KA. 2010; Turpie AG. 2007; Weitz JI. 2010.





## Targets for anticoagulants




Adapted from: 1. Weitz *et al*, 2005 and 2. Weitz *et al*, 2008; 3. Ansell *et al*, 2008.





## Rivaroxaban: absorption, distribution, metabolism, elimination

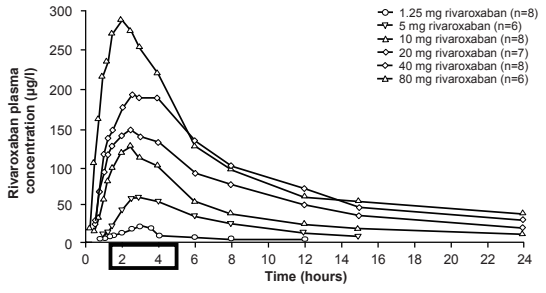
- Absorption
  - 10 mg tablet: oral bioavailability of 80-100% (independent of food intake)
  - 20 mg tablet: oral bioavailability of 66% under fasting conditions( due to reduced extent of absorption)
  - 20 mg tablets taken together with food increased mean AUC by 39%, indicating almost complete absorption and high oral bioavailability
    - → Xarelto 15 mg and 20 mg are to be taken with food
- Distribution
  - Plasma protein binding 92–95%, with moderate volume of distribution
- Metabolism
  - ~Two-thirds metabolized (one-third not metabolised, excreted unchanged in urine)
  - No major or active circulating metabolites
- Elimination
  - ~One-third excreted as unchanged active substance in urine
  - Of the two-thirds metabolized: half eliminated renally, half eliminated by the hepatobiliary route
  - Elimination of rivaroxaban from plasma occurs with terminal half-lives of 5 - 9 hours in [illegible] and with a terminal half-life of 11–13 hours in the elderly

Xarelto® SPC 2013; Stampfuss J *et al. Int J Clin Pharmacol Ther* 2013



## Rivaroxaban is absorbed rapidly, $C_{max}$ within 2–4 hours and a dose-dependent exposure

**A single-dose study in healthy volunteers**




Adapted from Kubitza D *et al. Clin Pharmacol Ther* 2005;78:412–421, with permission from the Nature Publishing Group



# ROCKET AF

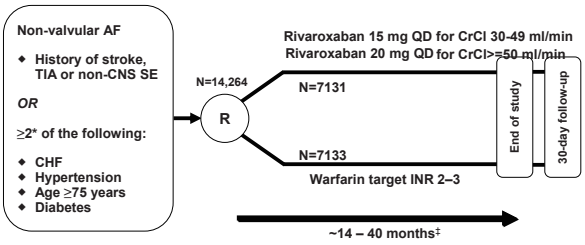
**Rivaroxaban Once Daily Oral Direct Factor Xa Inhibition Compared with Vitamin K Antagonism for Prevention of Stroke and Embolism Trial in Atrial Fibrillation**

**Rationale:**

To evaluate the efficacy and safety of rivaroxaban compared with warfarin for the prevention of stroke and systemic embolism in a moderate to high-risk set of patients with non-valvular AF



## ROCKET AF – study design




*Enrolment of patients with <3 risk factors or without prior stroke/TIA or non-CNS SE was limited to 10%.
#Patients with CrCl 30–49 ml/min: 15 mg rivaroxaban once daily.
‡Duration of therapy varied for each patient as study was event-driven.

Patel MR et al, 2011

ROCKET AF

## ROCKET AF – study endpoints

Primary efficacy endpoint

- Composite of stroke and sytemic embolism (SE)

Secondary efficacy endpoints

- Composite of stroke, SE and cardiovascular death
- Composite of stroke, SE, cardiovascular death and MI
- Individual components of the above endpoints

Principal safety endpoint

- Composite of major and non-major clinically relevant bleeding

Patel MR *et al*, 2011.





## ROCKET AF – study analyses

**Prespecified**

- **Non-inferiority (per-protocol population, on treatment)**
  - All patients having ≥1 dose of study drug and no major protocol violations; included events up to 2 days after last double-blind dose
- **Superiority (safety population, on treatment)**
  - All patients having ≥1 dose of study drug; included events up to 2 days after last double-blind dose
- **Additional analysis: intention-to-treat (ITT) analysis**
  - All patients randomized, including events up to end of study

**Post hoc**

- ITT populations: on and off treatment
- In those completing the study as planned, events occurring during transition to open-label therapy at study end

Patel MR *et al*, 2011.





## ROCKET AF – baseline characteristics (1)

| Characteristic | Rivaroxaban (N=7,131) | Warfarin (N=7,133) |
|---|---|---|
| Age, median (25th, 75th), years | 73 (65, 78) | 73 (65, 78) |
| Female, % | 39.7 | 39. 7 |
| Body mass index, median, kg/m² | 28.3 | 28.1 |
| Blood pressure, median, mmHg | | |
| Systolic | 130 | 130 |
| Diastolic | 80 | 80 |
| Clinical presentation, n (%) | | |
| Type of atrial fibrillation | | |
| Persistent | 5,786 (81.1) | 5,762 (80.8) |
| Paroxysmal | 1,245 (17.5) | 1,269 (17.8) |
| Newly diagnosed/new onset | 100 (1.4) | 102 (1.4) |
| Previous ASA use | 2,586 (36.3) | 2,619 (36.7) |
| Previous VKA use | 4,443 (62.3) | 4,461 (62.5) |

ITT population

Patel MR *et al*, 2011.





## ROCKET AF – baseline characteristics (2)

| Characteristic | Rivaroxaban (N=7,131) | Warfarin (N=7,133) |
|---|---|---|
| $CHADS_2$ score, mean ± SD | 3.48±0.94 | 3.46±0.95 |
| 2, n (%) | 925 (13.0) | 934 (13.1) |
| 3, n (%) | 3,058 (42.9) | 3,158 (44.3) |
| 4, n (%) | 2,092 (29.3) | 1,999 (28.0) |
| 5, n (%) | 932 (13.1) | 881 (12.4) |
| 6, n (%) | 123 (1.7) | 159 (2.2) |
| Co-existing conditions, n (%) | | |
| Previous stroke/TIA or SE | 3,916 (54.9) | 3,895 (54.6) |
| Congestive heart failure | 4,467 (62.6) | 4,441 (62.3) |
| Hypertension | 6,436 (90.3) | 6,474 (90.8) |
| Diabetes mellitus | 2,878 (40.4) | 2,817 (39.5) |
| Previous MI | 1,182 (16.6) | 1,286 (18.0) |
| Peripheral vascular disease | 401 (5.6) | 438 (6.1) |
| Chronic obstructive pulmonary disease | 754 (10.6) | 743 (10.4) |
| CrCl, median (25th, 75th), ml/min | 67 (52, 88) | 67 (52, 86) |

ITT population

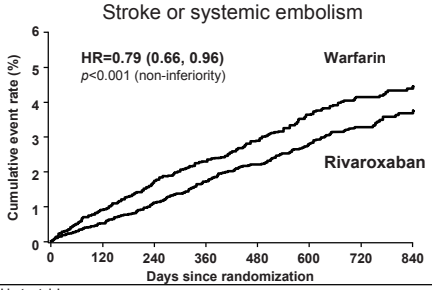
Patel MR *et al*, 2011.





## ROCKET AF – primary efficacy endpoint

Stroke or systemic embolism




| Number of subjects at risk | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Rivaroxaban | 6,958 | 6,211 | 5,786 | 5,468 | 4,406 | 3,407 | 2,472 | 1,496 |
| Warfarin | 7,004 | 6,327 | 5,911 | 5,542 | 4,461 | 3,478 | 2,539 | 1,538 |

Per-protocol population – as treated

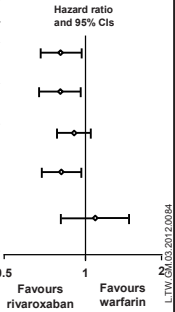
Patel MR *et al*, 2011.





## ROCKET AF – primary efficacy endpoint on and off treatment

| | Rivaroxaban n/N (% per year) | Warfarin n/N (% per year) | Hazard ratio (95% CI) | *p*-value Non-inf. | *p*-value Sup. |
|---|---|---|---|---|---|
| **Per protocol, on treatment** | **188/6,958 (1.7)** | **241/7,004 (2.2)** | **0.79 (0.66,0.96)** | **<0.001** | |
| **Safety, on treatment** | **189/7,061 (1.7)** | **243 /7,082 (2.2)** | **0.79 (0.65,0.95)** | | **0.02** |
| **ITT** | **269/7,081 (2.1)** | **306/7,090 (2.4)** | **0.88 (0.75,1.03)** | **<0.001** | **0.12** |
| ITT, on treatment | 188 (1.7) | 240 (2.2) | 0.79 (0.66,0.96) | | 0.02 |
| ITT, off treatment | 81 (4.7) | 66 (4.3) | 1.10 (0.79,1.52) | | 0.58 |

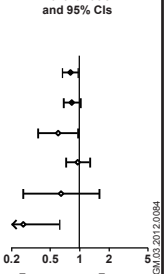



Primary efficacy endpoint: stroke or systemic embolism
ITT on- and off-treatment: *post hoc* analyses

Patel MR *et al*, 2011.

## ROCKET AF – primary efficacy endpoint components

| Endpoints | Rivaroxaban (N=7,061) n (% per year) | Warfarin (N=7,082) n (% per year) | Hazard ratio (95% CI) |
|---|---|---|---|
| **Primary efficacy endpoint** | 189 (1.7) | 243 (2.2) | 0.79 (0.65, 0.95)* |
| All-cause stroke | 184 (1.7) | 221 (2.0) | 0.85 (0.70,1.03) |
| Haemorrhagic stroke | 29 (0.3) | 50 (0.4) | 0.59 (0.37,0.93)* |
| Ischaemic stroke | 149 (1.3) | 161 (1.4) | 0.94 (0.75,1.17) |
| Unknown stroke type | 7 (0.1) | 11 (0.1) | 0.65 (0.25,1.67) |
| Non-CNS SE | 5 (0.04) | 22 (0.2) | 0.23 (0.09, 0.61)* |



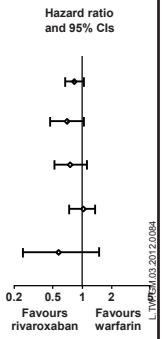

Safety population – on-treatment analysis
*Statistically significant

Patel MR *et al*, 2011.



## ROCKET AF – stroke outcomes

| Endpoints | Rivaroxaban (N=7,061) n (% per year) | Warfarin (N=7,082) n (% per year) | Hazard ratio (95% CI) |
|---|---|---|---|
| **Stroke** | 184 (1.7) | 221 (2.0) | 0.85 (0.70–1.03) |
| Stroke leading to death* | 47 (0.4) | 67 (0.6) | 0.71 (0.49–1.03) |
| Disabling* | 43 (0.4) | 57 (0.5) | 0.77 (0.52–1.14) |
| Non-disabling* | 88 (0.8) | 87 (0.8) | 1.03 (0.76–1.38) |
| Unknown | 7 (0.1) | 12 (0.1) | 0.59 (0.23–1.50) |




Safety population – on-treatment analysis
*Based on investigator's assessment of modified Rankin scale score: 0–2=non-disabling, 3–5=disabling, and 6=death

Patel MR *et al*, 2011.



## ROCKET AF – secondary endpoints

| Endpoints | Rivaroxaban (N=7,061) n (% per year) | Warfarin (N=7,082) n (% per year) | Hazard ratio (95% CI) |
|---|---|---|---|
| **Composite of stroke, non-CNS SE, vascular death** | 346 (3.1) | 410 (3.6) | 0.86 (0.74, 0.99)* |
| **Composite of stroke, non-CNS SE, vascular death and MI** | 433 (3.9) | 519 (4.6) | 0.85 (0.74, 0.96)* |
| **Components of major secondary endpoints** | | | |
| All-cause stroke | 184 (1.7) | 221 (2.0) | 0.85 (0.70, 1.03) |
| Non-CNS SE | 5 (0.04) | 22 (0.2) | 0.23 (0.09, 0.61)* |
| MI | 101 (0.9) | 126 (1.1) | 0.81 (0.63, 1.06) |
| Vascular death | 170 (1.5) | 193 (1.7) | 0.89 (0.73, 1.10) |
| **All-cause mortality** | 208 (1.9) | 250 (2.2) | 0.85 (0.70, 1.02) |

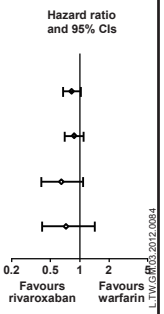
Safety population – on-treatment analysis. *Statistically significant

Patel MR *et al*, 2011.



## ROCKET AF – all-cause mortality

| Endpoints | Rivaroxaban (N=7,061) n (% per year) | Warfarin (N=7,082) n (% per year) | Hazard ratio (95% CI) |
|---|---|---|---|
| **All-cause mortality** | 208 (1.9) | 250 (2.2) | 0.85 (0.70,1.02) |
| Vascular death | 170 (1.5) | 193 (1.7) | 0.89 (0.73, 1.10) |
| Non-vascular death | 21 (0.2) | 34 (0.3) | 0.63 (0.36, 1.08) |
| Unknown cause | 17 (0.2) | 23 (0.2) | 0.75 (0.40, 1.41) |



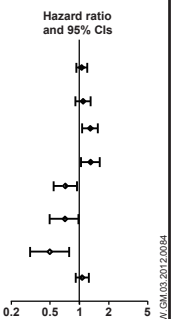

Safety population – on-treatment analysis

Patel MR *et al*, 2011.



## ROCKET AF – bleeding analysis

| Parameter | Rivaroxaban (N=7,111) n (% per year) | Warfarin (N=7,125) n (% per year) | Hazard ratio (95% CI) |
|---|---|---|---|
| **Principal safety endpoint** | 1,475 (14.9) | 1,449 (14.5) | 1.03 (0.96,1.11) |
| **Major bleeding** | 395 (3.6) | 386 (3.4) | 1.04 (0.90,1.20) |
| Haemoglobin drop (≥2 g/dl) | 305 (2.8) | 254 (2.3) | 1.22 (1.03,1.44)* |
| Transfusion | 183 (1.6) | 149 (1.3) | 1.25 (1.01,1.55)* |
| Critical organ bleeding | 91 (0.8) | 133 (1.2) | 0.69 (0.53,0.91)* |
| Intracranial haemorrhage | 55 (0.5) | 84 (0.7) | 0.67 (0.47,0.93)* |
| Fatal bleeding | 27 (0.2) | 55 (0.5) | 0.50 (0.31,0.79)* |
| **Non-major clinically relevant bleeding** | 1,185 (11.8) | 1,151 (11.4) | 1.04 (0.96,1.13) |




Major bleeding from gastrointestinal site (upper, lower and rectal): rivaroxaban=224 events (3.2%); warfarin=154 events (2.2%); $p<0.001$*
Safety population – on-treatment analysis; *Statistically significant

Patel MR *et al*, 2011.



## ROCKET AF – major bleeding by site

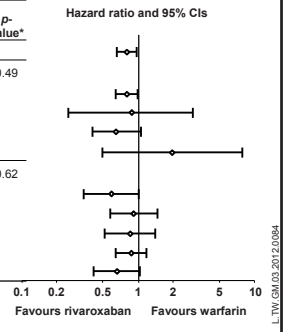
| Site* | Rivaroxaban (N=7,111) | Warfarin (N=7,125) |
|---|---|---|
| Major bleeding, n (%) | 395 (5.6) | 386 (5.4) |
| Gastrointestinal (upper, lower, rectal)# | 224 (3.2) | 154 (2.2) |
| Intracranial‡ | 55 (0.8) | 84 (1.2) |
| Intraparenchymal‡ | 37 (0.5) | 56 (0.8) |
| Non-traumatic‡ | 33 (0.5) | 54 (1.8) |
| Traumatic | 4 (0.1) | 2 (0.03) |
| Intraventricular | 2 (0.03) | 4 (0.1) |
| Subdural haematoma | 12 (0.2) | 22 (0.3) |
| Subarachnoid | 4 (0.1) | 1 (0.01) |
| Epidural haematoma | 0 | 1 (0.01) |
| Macroscopic haematuria | 26 (0.4) | 21 (0.3) |
| Bleeding associated with non-cardiac surgery | 19 (0.3) | 26 (0.4) |
| Intraocular/retinal | 17 (0.2) | 24 (0.3) |
| Intraarticular | 16 (0.2) | 21 (0.3) |
| Epistaxis | 13 (0.2) | 14 (0.2) |

*Site based on blinded adjudication.
#Combined gastrointestinal bleed rate $p<0.001$; ‡$p<0.05$

Patel MR *et al*, 2011.

## ROCKET AF – primary efficacy endpoint subgroup analysis

| | Rivaroxaban n/N | Rivaroxaban (%) | Warfarin n/N | Warfarin (%) | *p*-value* |
|---|---|---|---|---|---|
| Overall | 189/7,061 | 2.7 | 243/7,082 | 3.4 | |
| Race | | | | | 0.49 |
| White | 151/5,856 | 2.6 | 194/5,909 | 3.3 | |
| Black | 5/94 | 5.3 | 5/85 | 5.9 | |
| Asian | 27/894 | 3.0 | 41/887 | 4.6 | |
| Other | 6/217 | 2.8 | 3/201 | 1.5 | |
| Region | | | | | 0.62 |
| North America | 20/1,334 | 1.5 | 36/1,339 | 2.7 | |
| Latin America | 33/939 | 3.5 | 37/938 | 3.9 | |
| West Europe | 28/1,040 | 2.7 | 34/1,049 | 3.2 | |
| East Europe | 78/2,696 | 2.9 | 91/2,704 | 3.4 | |
| Asia-Pacific | 30/1,052 | 2.9 | 45/1,052 | 4.3 | |




*p-value for interaction.
Safety population – on-treatment analysis.

Patel MR *et al*, 2011.

ROCKET AF

L-TW-GM.03.2012.0084

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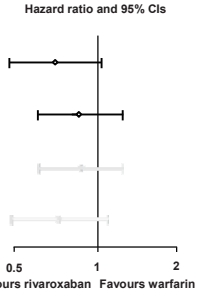
## ROCKET AF – primary efficacy endpoint centre-based INR control*

| cTTR | Rivaroxaban (% per year) | Warfarin (% per year) | Hazard ratio (95% CI) |
|---|---|---|---|
| 0.0–50.6% | 1.8 | 2.5 | 0.70 (0.48, 1.03) |
| 50.7–58.5% | 1.9 | 2.2 | 0.89 (0.62, 1.29) |
| 58.6–65.7% | 1.9 | 2.1 | 0.89 (0.62, 1.28) |
| 65.7–100.0% | 1.3 | 1.8 | 0.74 (0.49, 1.12) |

Better INR control




cTTR, centre-based time in therapeutic range
Based on Rosendaal method with all INR values included
*p-value for interaction=0.74
Safety population (N=7,061 [rivaroxaban], N=7,082 [warfarin])

Patel MR *et al*, 2011.

ROCKET AF

L-TW-GM.03.2012.0084

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# CURRICULUM VITAE

**Name:** 蘇正煌, M.D., Ph.D.
Assistant Professor of Medicine
Cardiovascular Division
Lab of Heart and Circulatory Physiology
Mackay Memorial Hospital Taipei TAIWAN

## EDUCATION:

| | |
|---|---|
| 1982 - 1989 | Undergraduate School: M.D. China Medical University, TAIWAN |
| Sep. 2002 - May. 2006 | Postgraduate School: Ph.D. School of Medicine and Biomedical Sciences, University of Sheffield, UK |

## ADMINISTRATIVE APPOINTMENTS:

| | |
|---|---|
| 2007 - 2009 | June Administrative Leader, 38 Medical Ward (Tamshui branch), MMH |
| 2009 July - 2013 July | Director, Intensive Care Unit (Tamshui branch), MMH |
| 2013 Sep - | Program Director in Charge of Education Services of Clinical Rotation at the Department of Internal Medicine, MMH |

## MAJOR RESEARCH GRANTS:

| | |
|---|---|
| Aug 2007 - July 2008 | NSC 96-2314-B-195-017 (Investigator) |
| Aug 2007 - July 2010 | NSC 96-2314-B-195-007-MY3 (Co-Investigator) |
| Aug 2009 - July 2010 | NSC 98-2221-E-195-027-017 (Co-Investigator) |
| Aug 2010 - July 2012 | NSC 99-2221-E-027-003- MY2 (Co-Investigator) |
| Aug 2010 - July 2012 | NSC 99-2314-B-195-010-MY2 (Investigator) |
| Aug 2012 - July 2013 | NSC 101-2314-B-195-016- (Investigator) |
| Aug 2013 - July 2014 | NSC 102-2314-B-195-010- (Investigator) |

## SELECTED PEER-REVIEWED JOURNAL ARTICLES:

1. Non-viral Technologies for Gene Therapy in Cardiovascular Research. International Journal of Gerontology June; 2(2): 35-47, 2008.
2. Ultrasonic Microbubble-mediated Gene Delivery Causes Phenotypic Changes of Human Aortic Endothelial Cells. Ultrasound in M. & B., Vol. 36, p. 449–458, 2010.
3. Therapeutic Angiogenesis of Human Early Endothelial Progenitor Cells Is Enhanced by Thrombomodulin. ATVB 2011 Nov; 31(11):2518-25.
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5. The Increase of VEGF Secretion From Endothelial Progenitor Cells Post Ultrasonic VEGF Gene Delivery Enhances The Proliferation and Migration of Endothelial Cells. Ultrasound in M. & B., 2013 Jan;39(1):134-45.
6. Reduction of connexin43 in human endothelial progenitor cells impairs the angiogenic potential. Angiogenesis 2013 Jul;16(3):553-60.

# Implication from an Updated NHIA Lipid Guideline to Optimal Statin Therapy

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Atherosclerotic cardiovascular disease (CVD) is the leading cause of death in the world. Lowering of low density lipoprotein cholesterol (LDL-C), is the established risk factor and cornerstone of prevention and treatment. Large-scale, randomized, prospective trials involving patients with CVD have consistently demonstrated that statin therapy significantly reduces LDL-C levels. Consequently, the incidence of all-cause mortality and major coronary events as compared to control in both secondary and primary prevention decreases.

It is known that more aggressive LDL-C lowering strategies by high dose potent statin therapies can reduce rates of non fatal events and need for intervention. However, an incremental coronary events and mortality still occur in the intensive statin groups, such as in the PROVE IT-TIMI 22, IDEAL and TNT trials. In other words, more intensive lipid-lowering therapies with potent statins are highly effective for lowering LDL-C levels and cardiovascular event rates. In reality, the strategy never eliminate cardiovascular risk. In addition, myopathy and renal events have been a significant concern via the use of high potency statins.

Residual cardiovascular risk may stem from mixed dyslipidaemia which typically presented in patients with type 2 diabetes and metabolic syndrome. The combined atherogenic dyslipidaemia include hypertriglyceridaemia, low high-density lipoprotein (HDL)-cholesterol levels, an accumulation of cholesterol-rich remnant particles and small, dense LDL particles.

In this talk, attention is being paid to residual CVD risk, the role of combined atherogenic dyslipidaemia, and an “optimal” statin therapy related to the updated NHIA lipid guideline 2013.

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中華民國心臟學會優秀論文獎第三名 (2010)
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# Is New-onset DM an Issue for Statin Therapy?

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Guidelines for lipid control are based on the results of clinical studies. Statin is a major contributor supported that the reduction of LDL-C which is beneficial for CV protection. Such findings also apply in patients with type 2 diabetes mellitus (T2DM), the lipid profile of whom is characterized by low HDL-C and high TG. Trials have been conducted to lower TG and/or raise HDL-C by traditional drugs or novel agents other than statins without showing CV outcome improvements. On the other hand, the beneficial effects of statins in patients with T2DM is comparable to those without. Therefore, statins remained the first-line drug for diabetic dyslipidemia not reaching the LDL-C goal.

However, data from real world practice showed that a substantial portion of patients can not tolerate statins. In recent years statins were reported to be diabetogenic. In addition, drug-drug interaction is a potential threat in multi-drug users, such as patients with T2DM. Although these concerns of statins weigh much less compared to the beneficial CV protective effects, statins without such properties, for example, pitavastatin, may be a better choice in T2DM. Japan PREvention Trial of Diabetes by Pitavastatin in Patients With Impaired GluCose Tolerance ( J-PREDICT) has confirmed that the hazard ratio for progression from IGT to diabetes in the pitavastatin group was 0.82 (95% CI: 0.68-0.99; P = 0.041). Even in any subgroups, pitavastatin did not accelerate the incidence, unlike the effects of statins in previous reports. Pitavastatin in combination with lifestyle modification was associated with a lower incidence of diabetes than was lifestyle modification alone in Japanese patients with IGT. Statins are now used with the understanding that a slightly increased risk of diabetes is outweighed by cardiovascular benefits of the drugs. However, based on our results, it may be necessary to reconsider whether all statins really increase the risk of developing diabetes.

# 簡　　歷

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# Debate: Is new-onset DM an issue for statin therapy? - *Cons*

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# LDL-C goal achievement: Role of aggressive approach with low dose dual inhibition

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http://accessibility.hch.gov.tw/public/doctor/5f0197b66c0c24be1c6cf570a51f1661.pdf

# Beyond LDL-C, how can we achieve comprehensive lipid goal in Ty2D with mixed-type dyslipidemia?

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1969-1970　中華民國陸軍少尉醫官

1970-1973　芝加哥大學病理研究 (博士學位)

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1977-1978　芝大醫院病理研究助理教授

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## *Diet therapy and life style modification for weight reduction in obesity*

**Robert M Chen MD, PhD.**
**Pathologist, TaiXing Clinic.**
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## *Obesity is the hallmark of civilization*

1. Over nourishment and inadequate exercise are common.
2. Overeating of fried junk food is prevalent.
3. These will cause overweight and high blood lipid, causally related to heart attack and CVA.
4. These will cause 5 cancers. (Breast, endometrium and prostate by hormone, Colon and gall bladder Cancer because of excessive bile salt)

## *Traditional Dieting not satisfactory*

1. Traditional Dieting restrict fat and meat, be replaced by more carbohydrate, but $CH_2O$ will convert to fat.
2. It will take less food and less calorie, but it will make subject hungry and sleepy (can't work).
3. Hunger will force subject to look for snacks, but the taste of snack will stimulate appetite, more hunger, thus tend to overeat, and reverse dieting.
4. It was so uncomfortable and no effect. So discontinue.

## *Atkin's diet happy and effective*

1. Atkin's diet : no carbohydrate, unlimited meat and fat.
2. You will take more vegetables and fruit to balance, but restrict very sweaty fruit. (grapes, pineapple, watermelon).
3. You may go to all banquet with no restrictions.
4. You don't feel hungry, but very energetic and allert.
5. Measure body weight and TG, you will see drastic reduction in 2 weeks.

## *Debate about Atkin's Diet (I)*

1. This diet is not healthful. It will provoke acidic, heart problem and cancer. True?
2. Yes, excessive protein will provide acidic fluid balance, but eating a lot vegetable and fruit will balance it.
3. Atkin's diet does not include excessive fat, and lean meat contains very low cholesterol.
4. Actually, this diet will reduce body weight rapidly, and reduce TG, with no hunger and collapse.

## *Debate about Atkin's Diet (II)*

1. We need carbohydrate and glucose as a fuel.
   Yes, but protein will convert to carbohydrate,
   Excessive $CH_2O$ will convert to lipid, not vise versa.
2. Too much meat will cause problems. (acidic, heart, cancer)
   Eat more vegetable and fruit will balance acid.
   Lean meat has very low cholesterol.
   Plant protein (beans) can balance meat.
3. If not sure, try it, you will see it is comfortable, healthy, easy to go leading to reduced weight.

## *A sacred fight to reduce weight*

1. Keep in mind, this is a sacred fight. It will make you beautiful, healthy, energetic, and successful. This will change your life, so fight with strong desire.
2. Measure you body weight every morning. Set the target of reducing one pound per week, or one kg per two weeks.
3. If not reaching the target, observe rigid dieting rule.
4. IF feel hungry, you may take a small candy, but not peanut or sweet chocolate.

## *Lift style change (I)*

1. Stop eating Junk food. Good taste will arouse appetite, Can't stop.
2. Stop drinking high sugar beverage.500c.c. or 1000c.c. milk tea is poisonous.
3. Strip away fat and skin in your meal.
4. Keep away chocolate & peanuts. Other nuts ok. Dark chocolate ok.
5. Skip breakfast is o.k. – The epinephrine will maintain bold glucose, and hunger will reduce cholesterol.

## *Lift style change (II)*

1. Have enough sleep. Otherwise you tend to overeat with less activity and become fat.
2. Eat less in dinner. You can take breakfast stuff for dinner.
3. Eat only 1/4 rice, or no rice at all.
4. Keep away bread, cake, donnut and ice cream. They look innocent, but hiding calorie and are poisonous.
5. Fast walk and Jogging daily, 1/2 hour is best.

# 簡　　歷

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2012　臺大醫院傑出研究獎
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2013　歐洲心臟科學會青年研究獎
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# The strategy for hypertension management – How to get more patients to goal?

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# ESC/ESH Guideline Update: Evidence-based Recommendations for Establishing BP Control – the Role of CCBs

蔡適吉 醫師
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Upon reviewing evidence from large randomised trials, the ESC/ESH concluded that the benefits of antihypertensive treatment were due to lowering of blood pressure (BP) per se (independent of the drugs employed). Indeed, the current guidelines reconfirm from 2003 and 2007 guidelines that diuretics, beta-blockers, calcium channel blockers (CCBs), angiotension-converting-enzyme inhibitors (ACE-I), and angiotensin receptor blockers (ARBs) are all suitable for initiation and maintenance of antihypertensive treatment either as monotherapy or, in some, in combination with each other. BP lowering regimens are selected on a subjective basis.[1] CCBs have demonstrated to be effective at office BP/ambulatory BP reduction, cardiovascular (CV) protection, and are considered protective against asymptomatic OD. Indeed, in the INSIGHT trial, despite having similar BP-lowering capabilities, nifedipine OROS (CCB) reduced coronary calcification across three years in addition to carotid artery intima-media thickness progression, compared with co-amilozide.[2] As such, while guidelines stipulate that greater attention is still required for organ damage (OD)-guided therapy and the prognostic significance of asymptomatic OD, CCBs are indicated as a preferred drug class in left ventricular hypertension (LVH) and asymptomatic atherosclerosis.[1] It is recognised that monotherapy has limited capacity to effectively reduce BP and keep a hypertensive patient within goal. Across a number of trials, when CCB or any other drug class has been added on to existing antihypertensive drug therapies, clinically significant reductions in systolic BP ensue compared with respective monotherapies. In particular, there is a marked BP lowering effect when CCB is combined with a renin-angiotensin system inhibitor (i.e., ACE-Is and ARBs) across a number of trials; significant superiority in improving CV outcomes with an ACE inhibitor in combination with CCB versus in combination with a diuretic was reported in the only trial to directly compare two combination regimens in all patients (ACCOMPLISH).[3]

There is room for improvement in BP control, and it is accepted that in some people BP management poses a greater challenge e.g. in old age, and in subjects with isolated systolic hypertension, diabetes, LVH, and renal damage/failure. Nevertheless, it is emphasised in the guidelines that achieving early BP control is important to improve on CV outcomes. Indeed, findings from the VALUE study suggest that recommended BP goals need to be reached within a relatively short time, at least in patients with hypertension who are at high CVD risk, and emphasises the clinical relevance of minor BP differences.[4,5] Notably, early BP control (at 6 weeks) with nifedipineOROS monotherapy has been found associated with improved CV outcomes (ACTION), and as early as 2 weeks in combination with telmisartan (TALENT).[6]

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2. Motro M &Shemesh J. Hypertension 2001; 37: 1410–3.
3. Jamerson K et al. N Engl J Med 2008; 359: 2417–2428.
4. Julius S et al. Lancet 2004; 363: 2022−31.
5. Weber MA et al. Lancet 2004; 363: 2049-51.
6. Mancia G et al. J Hypert 2011; 29 :600

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# Recent advance for the treatment of T2DM -- Comparison between DPP-4 inhibitor and GLP-1 receptor agonists

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# From Optimal Glycemic Control to CVD Protection

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Blood glucose levels have previously been the interest of internists/diabetologists, but accumulating evidence indicates that dysglycemia, especially elevated postprandial glucose (PPG), is widespread among patients with coronary artery disease. In fact, it is more common than normoglycemia in these patients and coexistence of dysglycemia and cardiovascular disease presents significant health risks. Recent studies have shown that that both conditions should be treated early to reduce the development of complications.

The cardiovascular toxicity of postprandial hyperglycemia is mediated by oxidant stress, which is directly proportional to the increase in glucose after a meal. This transient increase in free radicals acutely triggers inflammation, endothelial dysfunction, hypercoagulability, sympathetic hyperactivity, and a cascade of other atherogenic changes. Early randomized controlled trials indicate that reducing postprandial hyperglycemia appears to significantly slow atherosclerotic progression and may improve cardiovascular prognosis. Diet, exercise, and various pharmacologic agents can improve postprandial hyperglycemia. Agents specifically target PPG have been shown in several randomized controlled trials to be effective in controlling blood glucose, whether they are used as monotherapy or in combination with other antidiabetic medications. Studies have also suggested that acarbose, one of most commonly used AGIs, could decrease the risk of cardiovascular disease, both in IGT and in diabetes. Effect of GLP-1 analogues or DPP IV inhibitor on cardiovascular outcome await further study.