5月17日〈星期六〉08:30-10:00 sATURDAY may 18, 2025

台北國際會議中心(TICC)1F Theater 101

TSOC-\*JCS JOINT SESSION

precision Medicine

08: 30 Opening Remarks 洪大川

(Ta-Chuan Hung)

**Chair: 郭炫孚(Hsuan-Fu Kuo)**

08:35 Precision Medicine in Hyperlipidemia and Coronary Intervention: Tailoring Therapies for Optimal Outcomes………………………………………………………….Mixuki Miura

**Chair:謝育整(Yu-Cheng Hsieh)**

08:55 Precision Medicine in Patients with Inherited Cardiac Arrhythmia Syndromes………………………………………………………………Hiroshige MURATA

**Chair:許百豐(Pai-Feng Hsu)**

09:15 Precision Medicine in hypertension management………………………….…李俊偉

(Chun-Wei Lee)

**Chair:**

09:35 Panel Discussion all

09:55 Closing Remarks 謝敏雄

(Ming-Hsiung Hsieh)

\*Japanese Circulation Society

5月17日〈星期六〉10:30-12:00 sATURDAY may 17, 2025

台北國際會議中心(TICC)1F Theater 101

TSOC-Asian Region Joint Session

Innovative Technologies in Cardiovascular Health

**目前待排建議講者 如下:**

1. **越南  TBD**
2. **百靈佳:**

**Prof Michel Jadoul (KDIGO Co-Chair)**

**“From KDIGO guuideline udpate to optimization management in CKD”**

1. **Servier: 提議講者: 張鴻猷 醫師**

**「Defining the Optimal Heart Rate in HFrEF Care」**

**4.李俊偉委員推薦**

**Teruhiko Imamura, Toyama University**

**He will talk about heart failure medicine**

5月17日〈星期六〉14:00-15:30 sATURDAY may 17, 2025

台北國際會議中心(TICC)1F Theater 101

TSOC-\*ESC JOINT SESSION

Heart Failure

14: 00 Opening Remarks 吳彥雯

(Yen-Wen Wu) **Chair: 李任光(Jen-Kuang Lee)**

14:05 Implementation of Guidelines in Heart Failure: Why Is It So Important?...............………………………………………………………Michel Komajda

**Chair:林佳濱(Chia-Pin Lin)**

14:30 Heart Failure in Genetic Cardiomyopathies: Exploring the Role of ATTR, Fabry Disease, and HCM in Management Strategies…………………Roberto Barriales

**Chair:陳昱瑋(Yu-Wei Chen)**

14:55 Heart Failure with Preserved Ejection Fraction in Taiwan………………….…徐千彝

(Chien-Yi Hsu)

**Chair: 林佳濱(Chia-Pin Lin)**

15:15 Panel Discussion all

15:25 Closing Remarks 黃偉春

(Wei-Chun Huang)

\* European Society of Cardiology

5月17日〈星期六〉16:00-17:30 sATURDAY may 17, 2025

台北國際會議中心(TICC)1F Theater 101

TSOC--\*KSC JOINT SESSION

Digital Medicine (AI)

16: 00 Opening Remarks 黃玄禮

(Hsuan-Li Huang) **Chair: 曹玄明(Hsuan-Ming Tsao)**

16:05 ECG as a Digital Medicine for the Management of Atrial

Fibrillation………………………………………………………….………Ki Hong Lee

**Chair:張坤正(Kuan-Cheng Chang)**

16:25 Bridging Cardiology and AI: Innovative Solutions for Coronary Artery Disease Treatment………………………………………………..…………………. …..……張詩聖

(Shih-Sheng Chang)

**Chair:李文領(Wen-Lieng Lee)**

16:45 AI in the ablation therapy of atrial fibrillation………………………...……….…林彥璋

(Yenn-Jiang Lin)

**Chair: 李文領(Wen-Lieng Lee)**

17:05 Panel Discussion all

17:25 Closing Remarks 蘇峻弘

(Chun-Hung Su)

**\***Korean Society of Cardiology

5月17日〈星期六〉08:30-10:00 sATURDAY may 17, 2025

台北國際會議中心(TICC)1F Theater 102

Pediatrics (I)

建議邀請印度Smita Mishra演講者(ASD+Mitral Valve)，內容可規劃主題

及病例分享…等，由傅主委安排及請廠商安排贊助。

5月17日〈星期六〉10:30-12:00 sATURDAY may 17, 2025

台北國際會議中心(TICC)1F Theater 102

Pediatrics (II)

以Mitral valve in valve(兒科)為主，上半場安排4個專題演講(包括術前評

估、imaging評估、治療步驟及最後結果)，下半場規劃外賓演講及各醫院

Case的分享。

5月17日〈星期六〉14:00-15:30 sATURDAY, may 17, 2025

台北國際會議中心(TICC)1F, Room 102

Comprehensive Update on the Diagnosis and Treatment of Tachyarrhythmias

**14:00 Opening Remarks** 溫明賢

(Ming-Shien Wen)

**Chair: 林彥璋(Yenn-Jiang Lin)**

14:05 Recent Advances in Pulsed Field Ablation for Atrial Fibrillation 李政鴻

(Cheng-Hung Li)

**Chair: 羅力瑋(Li-Wei Lo)**

14:20 Emerging Developments in Image-Guided Ablation Techniques  
for the Management of Atrial and Ventricular Arrhythmias 郭 泠

(Ling Kuo)

**Chair: 林永國(Yung-Kuo Lin)**

14:35 Arrhythmogenic Mitral Valve Prolapse as a Cause of Unexplained  
Cardiac Arrest: Strategies for Identifying High-Risk Patients 廖英傑

(Ying-Chieh Liao)

**Chair: 陳永隆(Yung-Lung Chen)**

14:50 Timing of VT Ablation in Ischemic Heart Disease: Insights from  
the VANISH and VANISH-2 Trials 李柏增

(Po-Tseng Lee)

15:05 Panel Discussion .....all

**Panelists: 劉言彬(Yen-Bin Liu)、卓士傑(Shih-Jie Jhuo)、**

**陳儒逸(Ju-Yi Chen)、蔡青峰(Chin-Feng Tsai)、**

**謝育整(Yu-Cheng Hsieh)、張伯丞(Po-Cheng Chang)**

**15:25 Closing Remarks** 陳文鍾

(Wen-Jone Chen)

5月17日〈星期六〉16”00-17:30 sATURDAY, may 17, 2025

台北國際會議中心(TICC)1F, Room 102

Comprehensive Update on Device-Based Treatments for Cardiac Arrhythmias: Update of Device Treatment

**16:00 Opening Remarks** 張坤正

(Kuan-Cheng Chang)

**Chair: 林亮宇(Lian-Yu Lin)**

16:05 Update on Non-Vascular Implantable Cardioverter-Defibrillators  
(EV-ICD vs. s-ICD) 賀立婷

(Li-Ting Ho)

**Chair: 葉勇信(Yung-Hsin Yeh)**

16:20 Recent Breakthroughs and Clinical Implications of Leadless  
Pacemakers in Cardiac Electrophysiology 蔡適吉

(Su-Kiat Chua)

**Chair: 王俊傑(Chun-Chieh Wan)**

16:35 Latest Clinical Trials and Innovations in Physiological Pacing for   
Improved Patient Outcomes 林晏年

(Yen-Nien Lin)

**Chair: 郭任遠(Jen-Yuan Kuo)**

16:50 Comparative Effectiveness of Different Treatment Strategies in  
Patients with Heart Failure and Atrial Fibrillation:   
A Comprehensive Review 江承鴻

(Cheng-Hung Chiang)

17:05 Panel Discussion .....all

**Panelists: 柯文欽(Wen-Chin Ko)、吳宏彬(Hung-Pin Wu)、**

**張盛雄(Sheng-Hsiung Chang)、馮安寧(An-Ning Feng)、**

**游治節(Chih-Chieh Yu)、葉冠宏(Kuan-Hung Yeh)**

**17:25 Closing Remarks** 曹玄明

(Hsuan-Ming Tsao)

5月17日〈星期六〉08:30-10:00 SATURDAY, mAY 20, 2023

台北國際會議中心(TICC) 1F Room 103

**INTERNATIONAL YOUNG INVESTIGATORS PRESENTATION**

08:30 Opening Remarks

08:05 (Thailand)

08:25 (Philippines):

08:45 (Indonesia or Vietnam ):

09:05 (Taiwan)劉威廷

09:25 Closing Remarks

5月17日〈星期六〉10:30-12:00 SATURDAY, MAY 17, 2025

台北國際會議中心1樓Room 103

TSOC YOUNG INVESTIGATORS AWARD

FINALISTS COMPETITION

**Referees:**

建議5篇發表

6月1日〈星期六〉14:00-15:30 sATURDAY, june 1, 2024

台北國際會議中心(TICC) 1F Room 103

**Cardiovascular Surgery(I)**

6月1日〈星期六〉16:00-17:30 sATURDAY, MAY 17, 2025

台北國際會議中心(TICC) 1F Room 103

**Cardiovascular Surgery(II)**

5月17日〈星期六〉08:30-10:00 saturday, mAY 17, 2025

台北國際會議中心(TICC) 2F Room 201A

ORAL PRESENTATION

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| --- | --- |
| **Chairs: 3位評審+6位演講者** | |
| 08:30 |
| 08:45 |
| 09:00 |
| 09:15 |
| 09:30 |
| 09:45 |

5月17日〈星期六〉10:30-12:00 saturday, may 17, 2025

台北國際會議中心(TICC) 2F, Room 201A

**Innovative Technologies in**

**pulmonary hypertension**

**10:30 Opening Remarks** 林彥宏

(Yen-Hung Lin)

**Chair: 林彥宏 (Yen-Hung Lin)**

10:35 Overview of Evolving PAH Treatment Landscape:  
What’s New? Edmund Lau

10:55 Q & A

**Chair: 吳懿哲 (Yih-Jer Wu)**

11:00 Genetic Backgrounds of PAH in Taiwan: Focusing on the  
Pathogenic Variants in BMPR2 吳書豪

(Shu-Hao Wu)

11:15 Q & A

**Chairs: 許志新 (Chih-Hsin Hsu)**

11:20 Emerging Imaging Techniques for Pulmonary  
Hypertension 吳銘庭(or吳俊賢)

(Ming-Ting Wu) (or Chun-Hsien Wu)

11:35 Q & A

**Chairs: 宋思賢 (Shih-Hsien Sung)**

11:40 AI and Early Diagnosis of Pulmonary Hypertension 胡瑜峰

(Yu-Feng Hu)

11:55 Q & A

**12:00 Closing Remarks** 劉維新

(Wei-Shin Liu)

5月17日〈星期六〉13:20-15:00 SATURDAY, May 17, 2025

TICC國際會議中心 Room 201A

攝護腺癌心血管評估共識發表

13:20 Opening Remarks 張瑋婷

(奇美醫院)

**13:21 Chair: 馮思中教授 (泌尿醫學會)**

13:22 Taiwan Consensus for the Management of Cardiovascular Risks and

Complications in Patients with Prostate Cancer 洪健華

(台大醫院)

**13:37 Chair:陳文鍾教授 (心臟醫學會)**

13:38 Taiwan Consensus for the Management of Cardiovascular Risks and Complications in Patients with Prostate Cancer 陳東藝

(林口長庚)

13:53 Panelist: **馮思中**、**陳文鍾**、洪健華、陳東藝

13:54 Q&A All

**14:00**

癌心新境界--免疫治療的心血管議題

**14:01 Chair:** **張文震理事長**

14:02 TBD……………………………………………………………………………….彭孟婷

(林口長庚)

**14:15 Chair:謝宜璋秘書長**

14:16 Immune Checkpoint Inhibitor-Associated Myocarditis:Mechanisms, Diagnosis, and Evolving Management Strategies 陳彥舟(北醫)

14:31 Panelist: 張文震、謝宜璋、彭孟婷、陳彥舟

14:32 Q&A All

14:58 Closing Remarks 余文鍾

(台北榮總)

5月17日〈星期六〉15:20-16:40 SATURDAY, May 17, 2025

TICC國際會議中心2F Room 201A

Why Vaccination Coverage Needs to be Increased in Patients with CVD?

— Highlights of 2025 TSOC and IDST Consensus

15:20 Opening Remarks 李貽恒

(Yi-Heng Li)

15:25 **Chair: 吳彥雯 (Yen-Wen Wu)**

15:28 Influenza 林維文

(Wei-Wen Lin)

15:38 Pneumococcal……………………………………………………………………林鴻儒

(Hung-Ju Lin)

**15:48** Zoster………………………………………………………………………………….林柏霖

**(Po-Lin Lin)**

**15:58 Chair: 陳宜君(Yee-Chun Chen)**

16:01 COVID-19………………………………………………………………………….沈靜芬

(Ching-Fen Shen)

16:13 Tetanus–diphtheria–acellular Pertussis and RSV…………………………….紀鑫

(Chi Hsin)

**Chair: 黃立民(Li-Ming Huang)**

16:25 Panel Discussion

16:35 Closing Remarks………………………………………………………………….張峰義

(Feng-Yi Chang)

5月17日〈星期六〉08:30-10:00 sATURDAY, mAY 17, 2025

台北國際會議中心(TICC) 2F Room 201BC

TSOC-TSH JOINT SESSION(I)

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**Chair:**

08:30 Cardiac Mirror 鉅怡智慧推薦

(TBD)

**Chair:**

08:50 Cuffless BP-smart ring Hae-Young Lee(視訊)

(Korea)

**Chair:**

09:10 Energy PI Monitoring 鄭耿璽

(Geng-Shi Jeng)

**Chair:**

09:30 Optic sensor for Blood Pressure Measurement……………………………..王威智

(Wei-Chih Wang)

**Chair:**

09:50 Panel Discussion

5月17日〈星期六〉10:30-12:00 sATURDAY, mAY 17, 2025

台北國際會議中心(TICC) 2F Room 201BC

TSOC-TSH JOINT SESSION(II)

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**Chair:**

10:30 New Advancement of Renal Denervation 蔡維中

(Wei-Chung Tsai)

**Chair:**

10:50 Digital Therapeutics for Hypertension 潘人豪

(Ren-Hao Pan)

**Chair:**

11:10 Novel Strategyfor Saltreduction Fromfood Industry 劉昌樹

(Chang-Shu Liu)

**Chair:**

11:30 New Drug in Hypertension Management…………….……………………….陳建豪

(Chien-Hao Chen)

**Chair:**

11:50 Panel Discussion

5月17日〈星期六〉14:00-15:30 saturday, may 17, 2025

台北國際會議中心(TICC) 2F, Room 201BC

**Experience Sharing of HF Accreditation & Post-acute Care**

**14:00 Opening Remarks** 李貽恒

(Yi-Heng Li)

**Chair: 林維文 (Wei-Wen Lin)**

14:05 Experience Sharing of HF Accreditation & Post-acute Care 趙庭興

(Ting-Hsing Chao)

**Chair: 洪崇烈 (Chung-Lieh Hung)**

14:25 HF Quality Improvement Program from AHA 吳彥雯

(Yen-Wen Wu)

**Chair: 莊志明 (Jimmy Jyh-Ming Juang)**

14:45 Post-acute Care Program in Taiwan 王兆弘

(Chao-Hung Wang)

**Chairs: 林宗憲(Tsung-Hsien Lin)**

15:05 Panel Dission all

**15:25 Closing Remarks** 林宗憲

(Tsung-Hsien Lin)

5月17日〈星期六〉16:00-17:20 saturday, may 17, 2025

台北國際會議中心(TICC) 2F, Room 201BC

**HOT TOPICS ON HEART FAILURE MANAGEMENT:**

**DATA FROM TSOC HEART FAILURE REGISTRY 2020 IN COMPARISON WITH RECENT INTERNATIONAL HEART FAILURE REGISTRIES**

**16:00 Opening Remarks** 李貽恒

(Yi-Heng Li)

**Chair: 李貽恒 (Yi-Heng Li)**

16:03 Baseline Information and Clinical Characteristics of TSOC  
Heart Failure Registry 2020 王俊傑

(Chun-Chieh Wang)

**Chair: 李啟明 (Chii-Ming Le)**

16:18 Pharmacological Treatments and One-Year Outcomes of  
TSOC Heart Failure Registry 2020 張鴻猷

(Hung-Yu Chang)

**Chair: 吳彥雯 (Yen-Wen Wu)**

16:33 Two-year Prognostic Significance of Left Ventricular Ejection  
Fraction Trajectory from TSOC-HF Registry 2020 陳儀聲

(Yi-Sheng Chen)

**Chairs: 宋思賢 (Shih-Hsien Sung)**

16:48 The Effect of Heart Failure Post-Acute Care Program on Mortality  
and Rehospitalization in Patients with Different Phenotypes  
of Heart Failure: Insights from the TSOC HF Registry 2020 鄭淇文

(Yi-Sheng Chen)

**Chairs: 林宗憲 (Tsung-Hsien Lin)**

17:03 TBA

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**17:18 Closing Remarks** 林宗憲

(Tsung-Hsien Lin)

\* Each speaker has 12 minutes for presentation and 3 minutes for discussion.\*

5月17日〈星期六〉08:30-12:00 SATURDAY, May 17, 2025

Demo：**Chung Shan Medical University Hospital**、**Kaohsiung Veterans General Hospital**

Lecture：TICC 2F, Room 201DE (台北國際會議中心)

**LIVE DEMONSTRATION**

08:30 Opening Remarks……………………………………………………………………TBA

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08:35 Session I

|  |  |
| --- | --- |
| **CSMUH** | **Operators : (Rm. II) 李文領 (Wen-Lieng Lee)、莊曜聰 (Yao-Tsung Chuang ) CHIP**  **(Rm. Hy) 林茂欣 (Mao-Shin Lin)、蘇峻弘 (Chun-Hung Su)、 TAVI**  **吳怡良 (Yi-Liang Wu)**  **TEE Experts: 林維文 (Wei-Wen Lin)**  **IVUS, OCT, FFR Experts: 林佳濱 (Chia-Pin Lin)** |
| **KSVGH** | **Operators : (Rm. I) Kenya Nasu**  **(Rm. II) 顧博明(Po-Ming Ku)**  **TEE Experts:**  **IVUS, OCT, FFR Experts: 鄧欣一(Hsin-I Teng)** |

Chairs :

Commentators :

09:15-09:25 Lecture 1: TBA Matoa Habara

10:30 Session II

|  |  |
| --- | --- |
| **CSMUH** | **Operators : (Rm. I) 謝慕揚 (Mu-Yang Hsieh)、羅健賢 (Chien-Hsien Lo) Carotid Stent**  **(Rm. II) 劉世奇 (Shih-Chi Liu)、張凱為 (Kai-Wei Chang) CTO**  **IVUS, OCT, FFR Experts: 林佳濱 (Chia-Pin Lin)** |
| **KSVGH** | **Operators : (Rm. I) Matoa Habara**  **(Rm. II) 郭風裕(Feng-Yu Kuo)**  **TEE Experts:**  **IVUS, OCT, FFR Experts: 鄧欣一(Hsin-I Teng)** |

Chairs :

Commentators :

11:00-11:10 Lecture 2:

12:00 Lunch Time

5月17日〈星期六〉13:30-17:30 SATURDAY, May 17, 2025

Demo：**Chung Shan Medical University Hospital**、**Kaohsiung Veterans General Hospital**

Lecture：TICC 2F, Room 201DE (台北國際會議中心)

**LIVE DEMONSTRATION**

13:30 Session III

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| --- | --- |
| **CSMUH** | **Operators : (Rm. II) 李信賦 (Hsin-Fu Lee)、蕭文智 (Wun-Zhih Siao) CHIP**  **(Rm. Hy) 蘇峻弘 (Chun-Hung Su)、吳怡良 (Yi-Liang Wu) TAVI**  **TEE Experts: 林維文 (Wei-Wen Lin)**  **IVUS, OCT, FFR Experts: 林佳濱 (Chia-Pin Lin)** |
| **KSVGH** | **Operators : (Rm. I) 方修御(Hsiu-Yu Fang )**  **(Rm. II) 宋思賢(Shih-Hsien Sung) Mitral Clip**  **TEE Experts:**  **IVUS, OCT, FFR Experts: 鄧欣一(Hsin-I Teng)** |

Chairs :

Commentators :

14:20-14:30 Lecture 3: TBA…………………………………………………………Kenya Nasu

15:30 Session IV

|  |  |
| --- | --- |
| **CSMUH** | **Operators : (Rm. I) 簡思齊 (Szu-Chi Chien)、黃聖瑋 (Sheng-Wei Huang) CTO**  **(Rm. II) 徐中和 (Chung-Ho Hsu)、楊宗元 (Tsung-Yuan Young) PAD**  **IVUS, OCT, FFR Experts: 林佳濱 (Chia-Pin Lin)** |
| **KSVGH** | **Operators : (Rm. I) 李政翰(Cheng-Han Lee)、張其任(Chi-Jen Chang)**  **(Rm. II) 許榮城(Jung-Cheng Hsu) TAVI**  **TEE Experts:**  **IVUS, OCT, FFR Experts: 鄧欣一(Hsin-I Teng)** |

Chairs :

Commentators :

16:20-16:30 Lecture 4:

17:25 Closing Remarks………………………………………………………………….TBA

5月17日〈星期六〉08:30-10:00 saturday, mAY 17, 2025

台北國際會議中心(TICC) 2F Room 201F

ORAL PRESENTATION

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| --- | --- |
| **Chairs: 3位評審+6位演講者** | |
| 08:30 |
| 08:45 |
| 09:00 |
| 09:15 |
| 09:30 |
| 09:45 |

5月17日〈星期六〉10:30-12:00 sATURDAY, mAY 17, 2025

台北國際會議中心(TICC) 2F Room 201F

Innovative & Update Strategy in Cardiovascular Critical Care

**10:30 Opening Remarks** 李貽恒

(Yi-Heng Li)

**Chair: 林宗憲 (Tsung-Hsien Lin)**

10:35 Innovative Diagnostic Strategy From ECG to Heart Failure 黃昱彰

(Yu-Chang Huang)

**Chair: 藍偉仁 (Weiren Lan)**

10:55 Innovative Intervention Strategy in Acute Coronary Syndrome 李志國

(Chih-Kuo Lee)

**Chair: 黃靜惠 (Ching-Hui Huang)**

11:15 Update Strategy in Arrhythmia Intervention Under Cardiovascular or Cerebral Thromboembolic Event…… 余朝宏

(Chao-Hung Yu)

**Chair: 吳承學 (Cheng-Hsueh Wu)**

11:35 Update Strategy in Cardiogenic Shock, Focused on Diagnosis & Intervention…… 吳勃銳

(Po-Jui Wu)

**11:55 Closing Remarks** 黃群耀

(Chun-Yao Huang)

5月17日〈星期六〉14:00-15:30 saturday, may 17, 2025

台北國際會議中心(TICC) 2F, Room 201F

**Harnessing the power of omics in cardiovascular diseases**

**14:00 Opening Remarks** TBA

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**Chair: 楊鎧鍵 (Kai-Chien Yang)**

14:05 Genomics in Cardiovascular Diseases 陳沛隆

(Pei-Lung Chen)

**Chair: 胡瑜峰 (Yu-Feng Hu)**

14:25 Proteomics in Cardiovascular Diseases 潘思樺

(Szu-Hua Pan)

**Chair: 劉秉彥 (Ping-Yen Liu)**

14:45 Transcriptomics in Cardiovascular Diseases 柯泰名

(Tai-Ming Ko)

**Chair: 黃柏勳 (Po-Hsun Huang)**

15:05 Metabolomics in Cardiovascular Diseases 吳偉愷

(Wei-Kai Wu)

**15:25 Closing Remarks** TBA

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5月17日〈星期六〉16:00-17:30 saturday, may 17, 2025

台北國際會議中心(TICC) 2F, Room 201F

**siran & EV ?**

**16:00 Opening Remarks** TBA

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**Chair: 黃柏勳 (Po-Hsun Huang)**

16:03 Overview of siRNAs Design and Clinical applications 曾冠喬

( )

**Chair: 王黃舟 (Huang-Joe Wang)**

16:18 Efficacy and Safety of Inclisiran in Asian Patients 江晨恩

(Chern-En Chiang)

**Chair: 柯毓麟 (Yu-Lin Ko)**

16:33 Efficacy and Safety of RNA Interference Therapeutics in  
Transthyretin Amyloidosis in Asian Patient 林彥宏

(Yen-Hung Lin)

**Chair:**

16:48 Extracellular Vesicle Interplay in Cardiovascular Pathophysiology TBA

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**Chair:**

17:03 TOPIC? TBA

( )

**Chair:**

17:18 Discussion All

**17:28 Closing Remarks** TBA

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5月17日〈星期六〉08:30-10:00 saturday, mAY 17, 2025

台北國際會議中心(TICC) 4F VIP Room A

ORAL PRESENTATION

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| **Chairs: 3位評審+6位演講者** | |
| 08:30 |
| 08:45 |
| 09:00 |
| 09:15 |
| 09:30 |
| 09:45 |

5月17日〈星期六〉10:30-12:00 saturday, may 17, 2025

台北國際會議中心(TICC) 4F, VIP Room A

**Challenging Imaging**

**10:28 Opening Remarks** 洪明銳

(Ming-Jui Hung)

**Session I: Imaging the Myocardial Hypertrophy**

**Chair: 余文鍾(Wen-Chung Yu)**

**Panelists:宋國慈(Kuo-Tzu Sung)、楊荔丹(Li-Tan Yang)、劉文浩(Wen-Hao Liu)**

10:30 Image Case 廖若男

(Jo-Nan Liao)

10:50 Cardiac Magnetic Resonance Imaging for Left Ventricular  
Hypertrophy 郭 泠

(Ling Kuo)

**Session II: Imaging the Tricuspid Regurgitation**

**Chair: 王俊力(Chun-Li Wang)**

**Panelists:蔡惟全(Wei-Chuan Tsai)、林隆君(Lung-Chun Lin)、李慶威(Ching-Wei Lee)**

11:00 Image Case 盧政諱

(Cheng-Hui Lu)

11:20 Echocardiography for Tricuspid Valve Intervention 王朝永

(Chao-Yung Wang)

**Session III: Imaging the Heart Failure with**

**Preserved Ejection Fraction**

**Chair: 梁馨月(Hsin-Yueh Liang)**

**Panelists:陳美綾(Mei-Ling Che)、蔡蕙如(Huey-Ru Tsai)、李香君(Hsiang-Chun Lee)**

11:30 Image Case 劉家豪

(Chia-Hao Liu)

11:50 Echocardiography in Differentiating Heart Failure with  
Preserved Ejection Fraction 董承昌

(Cheng-Chang Tung)

**12:00 Closing Remarks** 秦志輝

(Chih-Hui Chin)

5月17日〈星期六〉14:00-15:30 saturday, may 17, 2025

台北國際會議中心(TICC) 4F, VIP Room A

**Application of Stress Echo in Non-Ischemic Heart Disease**

**14:00 Opening Remarks** 洪明銳

(Ming-Jui Hung)

**Chair: 江正文(Cheng-Wen Chiang)**

14:02 Severe Aortic Stenosis, D2 呂岱穎(視訊)

(Dai-Yin Lu)

14:16 Q & A

**Chair: 張坤正(Kuan-Cheng Chang)**

14:19 Pulmonary Hypertension 董承昌

(Cheng-Chang Tung)

14:33 Q & A

**Chairs: 梁馨月(Hsin-Yueh Liang)**

14:36 Diastolic Dsfunction 李文煌

(Wen-Huang Lee)

14:50 Labile Hypertrophic Cardiomyopathy 張瑋婷

(Wei-Ting Chang)

15:04 Coronary Flow Velocity Reserve 洪明銳

(Ming-Jui Hung)

15:18 Panel Dission

**15:28 Closing Remarks** 秦志輝

(Chih-Hui Chin)

5月17日〈星期六〉16:00-17:30 sATURDAY, MAY 17, 2025

台北國際會議中心 TICC 4F VIP ROOM

\*TASL-TSOC Joint Session:

\* Taiwan Association for the Study of the Liver

5月17日〈星期六〉08:30-10:00 saturday, mAY 17, 2025

台北國際會議中心(TICC) 4F VIP Room B

ORAL PRESENTATION

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| --- | --- |
| **Chairs: 3位評審+6位演講者** | |
| 08:30 |
| 08:45 |
| 09:00 |
| 09:15 |
| 09:30 |
| 09:45 |

5月17日〈星期六〉10:30-12:00 saturday, MAY 17, 2025

台北國際會議中心(TICC) 4F VIP Room B

**STRUCTURAL HEART DISEASE**

**Innovative Technologies in Cardiovascular Health**

**10:30 Opening Remarks** 曹殿萍

(Tien-Ping Tsao)

**Chair: 鄭正一(Cheng-I Cheng)**

10:35 Integrating AI and Fluid Dynamics: Innovative Applications in Precision and Personalized Interventions for Structure Heart Disease Mohammad Alkhouli

(U.S.A.)

**Chairs: 陳科維(Ke-Wei Chen)、施志遠(Jhih-Yuan Shih)**

11:05 Q & A

**Chair:陳俊吉(Chun-Chi Chen)**

11:10 3D Printing Models before Complex Structure Heart Interventions 許榮城

(Jung-Cheng Hsu)

**Chairs: 黃睦翔(Mu-Shiang Huang)、劉尊睿(Tsun-Jui Liu)**

11:25 Q & A

**Chair: 林茂欣(Mao-Shin Lin)**

11:30 Applications of Transcatheter Electrocautery in Structure Heart Interventions………………………………………………………………………殷偉賢

(Wei-Hsian Yin)

**Chairs: 李應湘(Ying-Hsiang Lee)、詹仕戎(Shih-Jung Jang)**

11:50 Q & A

11:55 Closing Remarks……………………………………………………謝宜璋

(I-Chang Hsieh)

5月17日〈星期六〉13:20-14:40 sATURDAY, june 1, 2024

台北國際會議中心(TICC) 4F VIP Room B

**Residual Risk factors控制**

13:20 Opening Remarks TBD

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**Chair:**

13:22 Residual CVD Risk in Atherosclerotic Diseases: What To Do and How To Do It……….. Kausik K. Ray

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13:47 Q & A

**Chair: 簡國龍(Kuo-Liong Chien)**

13:52 Inflammation.. 林鴻儒

(Hung-Ju Lin)

**Chair:**

14:15 Lp(a). 王宇澄

(Yu-Chen Wang)

14:38 Closing Remarks TBD

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5月17日〈星期六〉15:20-16:40 sATURDAY, june 1, 2024

台北國際會議中心(TICC) 4F VIP Room B

**Obesity**

15:20 Opening Remarks TBD

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**Chair:**

15:22 Overview……….. 莊海華

(Hai-Hwa Chuang)

**Chair:**

15:42 外賓…………………………………………………………………….(Novo Nordisk)

**Chair:**

16:07 Management.. 江晨恩

(Chern-En Chiang)

**Chair:**

16:27 Panel Discussion

16:37 Closing Remarks TBD

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5月18日〈星期日〉08:20-10:20 sUNDAY, JUNE 2,2024

台北國際會議中心(TICC) 2F Room 201BC

08:20 TSOC President Address 李貽恒

(Yi-Heng Li)

**YOUNG INVESTIGATOR AWARD LECTURE**

**Chair:**

08:25 第一名得獎者

**Nong Ting Award Lecture**

**Chair:**

08:40 ………… TBD

(TBD)

**PLENARY SPEECH**

**Chair:**

09:00 Melanie B. Turner

(AHA)

**Chair:**

09:20 Thomas Lüscher

(ESC)

**Chair:**

09:40 Filippo Crea

(EHJ)

**TSOC GENERAL ASSEMBLY**

10:30 TSOC General Assembly

**Chairs: 李貽恒 (Yi-Heng Li) 理事長、**

**葉宏一 (Hung-I Yeh) 常務監事**

**Reporter: 謝宜璋 (I-Chang Hsieh) 秘書長**

5月18日〈星期日〉13:15-14:45 sunday, may 18, 2025

台北國際會議中心(TICC) 1F, Room 101

**PCI in reduced LVEF function**

**13:15 Opening Remarks** (名譽理事)

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**Session I: PCI in Patient with Reduced LVEF without CHF**

**Chair:**

13:20 PCI Better TBA

(TSCI推薦)

13:35 Medical Treatment Better TBA

(TSCI推薦)

13:50 Discussion

**Session II: PCI has no Role in Patient with CHF and Reduced LVEF?**

**Chair: 王植賢(Chih-Hsien Wang)** (or殷偉賢/陳益祥/許榮彬)

14:00 CABG is Better 劉國聖(or王植賢)

(Kuo-Sheng Liu)

14:15 HR-PCI with MCS is Effective 盧澤民

(Tse-Min Lu)

14:30 Discussion

**14:40 Closing Remarks** 黃瑞仁(or王志鴻)

(Juey-Jen Hwang)

5月18日〈星期日〉15:00-16:30 sUNDAY, may 18, 2025

台北國際會議中心(TICC) 1F, Room 101

**severe AS treated with TAVR**

**associated with CAD**

**15:00 Opening Remarks** 黃啟宏

(Chi-Hung Huang)

**Chair:** (TSCI ?)

15:05 How to Diagnose CAD in Severe AS Before TAVR: The Role  
of Invasive Functional Studies, Coronary Angiography,   
and CTA TSCI ?

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15:20 Q & A

**Chair:** (TSCI ?)

15:25 When to Treat CAD in TAVR Patients:  
Pre-TAVR vs. Post-TAVR TSCI ?

( )

15:40 Q & A

**Chair: 王怡智(Yi-Chih Wang)**

15:45 How to Treat CAD after TAVR: Key Points of Performing  
PCI Procedure 謝明哲

(Ming-Jer Hsieh)

16:00 Q & A

**Chair:** **曹殿萍(Tien-Ping Tsao)**

16:05 How to Prevent Coronary Occlusion during TAVR 許榮城

(Jung-Cheng Hsu)

16:20 Q & A

**16:25 Closing Remarks** TSCI ?

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5月18日〈星期日〉13:15-14:45 sUNDAY, may 18, 2025

台北國際會議中心(TICC) 1F, Room 103

年會建議主題:申請部訂次專科的同時，邀請能力導向學會專家來分享說明。舉辦形式看是與談還是研討會或工作坊再議

5月18日〈星期日〉15:00-16:30 sunday, may 18, 2025

台北國際會議中心(TICC) 1F, Room 103

**結合新科技（如人工智慧）**

**在醫療品質改善與病人安全方面的應用**

**15:00 Opening Remarks** 褚柏顯

(Pao-Hsien Chu)

**Chair: 林昭維 (Jou-Wei Lin)**

15:05 人工智慧在心血管疾病預測與診斷中的應用：  
在醫療品質改善與病人安全 林錦生

(Chin-Sheng Lin)

15:20 Q & A

**Chair: 洪大川 (Ta-Chuan Hung)**

15:25 穿戴式裝置與遠距醫療技術在心血管健康監測的角色：  
在醫療品質改善與病人安全 洪啟盛

(Chi-Sheng Hung)

15:40 Q & A

**Chair: 張恒嘉 (Heng-Chia Chang)**

15:45 心血管疾病治療中的數據驅動決策：  
在醫療品質改善與病人安全 吳健嘉

(Chien-Chia Wu)

16:00 Q & A

**Chair: 趙嘉倫 (Chia-Lun Chao)**

16:05 AI技術的倫理挑戰與心血管醫療安全的平衡 林庭光

(Tin-Kwang Lin)

16:20 Q & A

**16:25 Closing Remarks** 蔡佳醍

(Chia-Ti Tsai)

5月18日〈星期日〉13:15-14:45 sunday, may 18, 2025

台北國際會議中心(TICC) 2F, Room 201A

**基層醫師常見醫療問題**

**13:15 Opening Remarks** 詹貴川

(Kuei-Chuan Chan)

**Chair: 詹貴川(Kuei-Chuan Chan)**

13:20 Prediction and Detection of Atrial Fibrillation 趙子凡

(Tze-Fan Chao)

**Chair: 曹玄明 (Hsuan-Ming Tsao)**

13:40 Integrated Management of AF. Focusing on Risk Factor  
Modification and Stroke Prevention 黃嵩豪

(Sung-Hao Huang)

**Chair: 曹承榮 (Chen-Rong Tsao)**

14:00 Blood Pressure Effect after Renal Denervation: 1-year Result  
from Taiwan RND Registry and HTN Guideline 李應湘

(Ying-Hsiang Lee)

**Chair: 丁革新 (Ke-Hsin Ting)**

14:00 Individualizing ASCVD Prevention: Statin and Non-statin  
Therapies for Primary Prevention 林柏霖

(Po-Lin Lin)

**14:40 Closing Remarks** 曹玄明

(Hsuan-Ming Tsao)

5月18日〈星期日〉15:00-16:30 sunday, may 18, 2025

台北國際會議中心(TICC) 2F, Room 201A

**健保新制下的機會與挑戰：醫中與基層的對話**

**15:00 Opening Remarks** 張坤正

(Kuan-Cheng Chang)

**Chairs: 王志鴻(Ji-Hung Wang)、詹貴川(Kuei-Chuan Chan)、侯嘉殷(Charles Jia-Yin Hou)**

15:05 基層心臟科診所未來的展望 曾維功

(Wei-Kung Tseng)

15:20 Q & A

**Chair: 柯文欽(Wen-Chin Ko)、陳志鴻(Jyh-Hong Chen)、謝士明(Shyh-Ming Shieh)**

15:25 基層心臟科診所在垂直整合醫療扮演的角色(或自訂) 徐迺維

(Nai-Wei Hsu)

15:40 Q & A

**Chair: 張詩聖(Shih-Sheng Chang)、曹殿萍(Tien-Ping Tsao)、黃瑞仁(Juey-Jen Hwang)**

15:45 總額制度下介入性導管新技術和材料的發展 簡思齊

(Szu-Chi Chien)

16:00 Q & A

**Chair: 陳文鍾(Wen-Jone Chen)、林彥璋(Yenn-Jiang Lin)、蔡佳醍(Chia-Ti Tsai)**

16:05 總額制度下電氣生理新技術和材料的發展 林亮宇

(Lian-Yu Lin)

16:20 Q & A

**16:25 Closing Remarks** 陳清埤

(Ching-Pei Chen)

5月18日〈星期日〉13:15-14:45 sunday may 18, 2025

台北國際會議中心(TICC) 2F Room 201BC

ACC in Taiwan: microRNA

13: 15 Opening Remarks 劉秉彥

(Ping-Yen Liu)

**Chair: 黃柏勳(Po-Hsun Huang)**

13:20 Topic: MicroRNAs and Novel Therapeutics for Peripheral and Coronary Artery Disease…………………………………………………………………….. (Brian H. Annex)

**Chair:葉宏一(Hung-I Yeh)**

13:50 TBD （詢問中）（蘇正煌醫師） 蘇正煌

(Cheng-Huang Su)

**Chair:葉宏一(Hung-I Yeh)**

14:20 Panel Dission

14:40 Closing Remarks 王黃舟

(Huang-Joe Wang)

5月18日〈星期日〉15:00-16:30 sATURDAY, june 1, 2024

台北國際會議中心(TICC) 1F Room 101C

TSOC Guidelines

Preview of the Highlights of the Forthcoming TSOC-Endorsed Clinical

Guidelines/Consensus and Comparisons with Major International Guidelines

15:00 Opening Remarks 李貽恒

(Yi-Heng Li)

**Chair: 趙庭興 (Ting-Hsing Chao)**

15:05 Pulmonary Artery Hypertension 吳懿哲

(Yih-Jer Wu)

**Chair: 王宗道 (Tzung-Dau Wang)**

15:25 Elderly Hypertension 鄭浩民

(Hao-Min Cheng)

**Chair: 翁國昌 (Kwo-Chang Ueng)**

15:45 Atrial fibrillation 趙子凡

(Tze-Fan Chao)

**Chair: 林宗憲 Tsung-Hsien Lin)**

16:05 Panel Discussion

16:25 Closing Remarks 徐國基

(Kou-Gi Shyu)

5月18日〈星期日〉13:15-14:45 sunday, may 18, 2025

台北國際會議中心(TICC) 2F, Room 201DE

**青年醫師論壇**

**科技與智慧驅動之心血管醫學新世代**

**13:15 Opening Remarks** 林姝含

(Donna Shu-Hen Lin)

**Chair: 陳文鍾(Wen-Jone Chen)**

13:20 心血管疾病治療的研發：從科學突破到臨床轉化 吳造中

(Chau-Chung Wu)

**Chair: 王宗道(Tzung-Dau Wang)**

13:40 人工智慧在心血管風險分析中的應用 潘恆宇

(Heng-Yu Pan)

**Chair: 林亮宇(Lian-Yu Lin)**

14:00 光子刀技術治療嚴重性心律不整的創新與展望 賀立婷

(Li-Ting Ho)

**Chair: 梁懷文(Huai-Wen Liang)、劉宜學(Yi-Hsueh Liu)**

**劉家豪(Chia-Hao Liu)、宋亨佑(SUNG HENG YOU)**

14:20 Q & A

**14:40 Closing Remarks** 李貽恒

(Yi-Heng Li)

5月18日〈星期日〉15:00-16:30 sunday, may 18, 2025

台北國際會議中心(TICC) 2F, Room 201DE

**青年醫師論壇**

**未來科技在心血管介入治療中的創新應用**

**15:00 Opening Remarks** 陳政瑋

(Zheng-Wei Chen)

**Chair: 高憲立(Hsien-Li Kao)**

15:05 HoloLens在複雜冠狀動脈介入治療中的革新應用 謝慕揚

(Mu-Yang Hsieh)

**Chair: 羅秉漢(Ping-Han Lo)**

15:25 3D列印技術於左心耳封堵術的臨床應用 許榮城

(Jung-Cheng Hsu)

**Chair: 廖智冠(Chih-Kuan Liao)**

15:45 未來血管內影像技術的再進化 何明昀

(Ming-Yun Ho)

**Chair: 張捷宇(Chieh-Yu Chang)、柯呈諭(Cheng-Yu Ko)**

**許如瑩(Ju-Yin Hsu)、曾致學(Chih-Hsueh Tseng)**

16:05 Q & A

**16:25 Closing Remarks** 謝宜璋

(I-Chang Hsieh)

5月18日〈星期日〉13:15-16:30 sUNDAY, may 18, 2025

台北國際會議中心(TICC) 2F Room 201F

JOINT COMMITTEE OF CRITICAL CARE MEDICINE CERTIFIED COURSE

〔REGISTRATION ONLY〕

SESSION I: Update Information in End-of-Life of Critical Care Medicine

13:15 Opening Remarks 許志新

(Chih-Hsin Hsu)

**Chair: 蔡宗能 (Tsung-Neng Tsai)**

13:20 End-Of-Life Act in Taiwan: From Hospice Care, Patient Autonomy to Organ Transplant & Donation 蔡宏斌

(Hung-Pin Tsai)

**Chair: 辛和宗 (Ho-Tsung Hsin)**

14:00 End-of-Life Care, Shared Decision Making and Time-limited Trials in ICU…………………………………………………………………………………陳志金

(Che-KimTan)

14:40 Closing Remarks..………………………………………………………………徐國基

(Kou-Gi Shyu)

14:45 Healthy Break

**SESSION II: Essential End-of-Life Issues of Critical Care Medicine in Cardiology**

15:00 Opening Remarks 張維典

(Wei-Tien Chang)

**Chair: 王晨旭 (Chen-Hsu Wang)**

15:05 Update Strategy in Cardiac Arrest Patients post ROSC 蔡旼珊

(Wen-Shan Tsai)

**Chair: 陳益祥 (Yih-Sharng Chen)**

15:45 From DBD to DCD: A Glance of Organ Recruitment in Taiwan 鍾孟軒

(Meng-Hsuan Chung)

16:25 Closing Remarks 黃群耀

(Chun-Yao Huang)

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| **CV of Invited Faculty** | |
|  | **Sung-hao Huang** |
| **Job Title:** | Assistant Professor |
| **Position:** | Director of Department of Internal Medicine |
| **Institution:** | National Yang Ming Chiao Tung University Hospital, Yi-Lan, Taiwan |
| **Major Field:** | Conduction system pacing |
| **Education:** | Taipei Medical University |
| **Professional Experience:** | 2017-2025 Faulty and the Secretary-General of Taiwan Heart Rhythm Society |
| **Honor & Award:** | 2014, EHRA Certified Cardiac Device Specialist level 1  2021, 2nd prize of poster presentation, annual meeting of THRS  2022, 1st prize of poster presentation, annual meeting of THRS |
| **Short Bio(150 words):** | Dr. Huang is an electrophysiologist with experiences of physiological pacing therapy and leadless pacemaker implantation. He received complete internal medicine and cardiology training at Taipei Veterans General Hospital and Chang Chung Memorial Hospital. His studies focused on subclinical atrial fibrillation in CIEDs and physiological pacing.  Publications:   1. **The presence of ectopic atrial rhythm predicts adverse cardiovascular outcomes in a large hospital-based population. Huang SH**, Hu YF, Chen PF, Lin YJ, Chang SL, Lo LW, Chung FP, Tuan TC, Chao TF, Liao JN, Chang TY, Lin CY, Liu CM, Huang TC, Vicera JJB, Lee PT, Lugtu IC, Jain A, Wu IC, Chen SA. Heart Rhythm 2020;17:967–974 2. **“Longitudinal dissociation” – the fundamentals of electrical resynchronization in bundle branch block by His bundle pacing. Sung-Hao Huang**, Hsuan-Ming Tsao. Strait circulation journal. 2020; 2(3):9-12; DOI: 10.6907/SCJ.202009\_2(3).0003 3. **The differences of left ventricular deformation between cardiac pacing resynchronization therapy and His corrective left bundle branch block in a patient with heart failure. Sung-Hao Huang,** Chao-Feng Liao, Zu-Yin Chen, Hsuan-Ming Tsao. Acta Cardiol Sin 2022;38:526-29 4. **Distinct Atrial Remodeling in Patients with Subclinical Atrial Fibrillation: Lessons from Computed Tomographic Images. Sung-Hao Huang**, Chao-Feng Liao, Zu-Yin Chen, Tze-Fan Chao, Shih-Ann Chen, MD, Hsuan-Ming Tsao. Pharmacology Research & Perspectives 2022; DOI: 10.1002/prp2.927 5. **Multimodality imaging assessment of the Biatrial remodeling of the burden of atrial high-rate episodes in patients with cardiac implanted electronic devices.** [**Sung-Hao Huang**, Hsuan-Ming Tsao, Chao-FengLiao, Zu-Yin Chen, Tze-Fan Chao, Shih-Ann Chen](https://www.sciencedirect.com/science/article/pii/S0167527322014759#!). Int J Cardio. 2023;371:175-183. DOI: https://doi.org/10.1016/j.ijcard.2022.10.007. 6. **Deep Learning-based Automatic Left Atrial Appendage Filling Defects Assessment on Cardiac Computed Tomography for Clinical and Subclinical Atrial Fibrillation Patients.** Ling Chen, **Sung-Hao Huang,\*** Tzu-Hsiang Wang, Tzuo-Yun Lan, Vincent S. Tseng, Hsuan-Ming Tsao, Hsueh-Han Wang, Gau-Jun Tang. Heliyon 2023; e12945. DOI: [10.1016/j.heliyon.2023.e12945](https://doi.org/10.1016/j.heliyon.2023.e12945). **\*Corresponding author.** |

**蔡 旼 珊 履 歷 (*Curriculum Vitae*)**

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| 學 | 學 校 名 稱 | | | 系 所 | 學 位 | | 畢(肆)業起迄年月 | |
|  | 國立台灣大學 | | | 醫學系 | 學士 | | 82年9月~89年6月 | |
|  | 國立台灣大學 | | | 臨床醫學研究所 | 碩士 | | 93年09月~ (直升博士班) | |
| 歷 | 國立台灣大學 | | | 臨床醫學研究所 | 博士 | | 95年09月~100年12月 | |
| 專 長 領 域 | | 急診醫學、內科醫學、復甦醫學、重症加護醫學 | | | | | | |
| 經 | 機 關 名 稱 | | 職 稱 | | | 起 迄 年 月 | | |
|  | 臺灣大學附設醫院新竹分院 | | 急診醫學部主任 | | | 106年1 月1日~ 迄今 | | |
|  | 臺灣大學附設醫院 | | 急診醫學部主治醫師 | | | 94年7月1日~95年2月28日  95年12月20日~98年7月31日  99年8月1日~105年12月31日 | | |
|  | 臺灣大學附設醫院雲林分院 | | 急診醫學部主治醫師 | | | 95年3月1日~95年12月20日  98年8月1日~99年7月31日 | | |
|  | 臺灣大學附設醫院 | | 急診醫學部住院醫師 | | | 89年7月~94年6月 | | |
| 資 | 証 書 字 號 | | 級 別 | | | 核 發 機 關 | | 起算(核發)年月 |
|  | 醫字第032571號 | | 醫師 | | | 衛生署 | | 89年10月 |
|  | 內科專字第006580號 | | 內科專科醫師 | | | 衛生署 | | 92年10月 |
|  | 急專醫字第000823號 | | 急診專科醫師 | | | 衛生署 | | 94年10月 |
|  | 講字第091763號 | | 講師 | | | 教育部 | | 96年11月~ |
|  | 助理字第036015號 | | 助理教授 | | | 教育部 | | 101年8月~ |
|  | 副字第143999號 | | 副教授 | | | 教育部 | | 106年8月~ |
|  | 教字第146780號 | | 臨床教授 | | | 教育部 | | 111年8月~ |
|  | 中華民國高級心臟救命術聯合委員會第0544 號指導員 | | 高級心臟救命術指導員 (ACLS Instructor) | | | 急救加護醫學會 | | 93年6月 |
|  | 國家災難醫療隊(北區) | | 隊員 | | | 國家災難醫療隊 | | 94年7月 |
| 格 | 醫用超音波學會會員M1319 | | 會員 | | | 中華民國醫用超音波學會 | | 97年9月 |
|  | 醫用超音波學會 | | 急診超音波專業醫師 | | | 中華民國醫用超音波學會 | | 98年1月 |

**吳勃銳**

**教育經歷**

|  |  |
| --- | --- |
| 國立成功大學醫學系 | 2003年9月至2010年6月 |
| 國立成功大學附設醫院 | 2009年6月至2009年7月 |
| 實習醫師 | 2009年9月至2010年5月 |
| 辜公亮基金會和信治癌中心醫院 | 2009年8月 |
| 實習醫師 |  |

**專業經歷**

|  |  |
| --- | --- |
| 高雄市立鳳山醫院心臟內科（委託長庚醫療  財團法人經營） |  |
| 一般級主治醫師 | 2017年10月至2018年6月 |
| 講師級主治醫師 | 2018年7月至2018年12月 |
| 高雄長庚紀念醫院心臟內科 |  |
| 一般級主治醫師 | 2016年10月至2017年9月 |
| 講師級主治醫師 | 2019年1月迄今 |
| 高雄長庚紀念醫院心臟內科 |  |
| 總醫師 | 2014年7月至2016年9月 |
| 高雄長庚紀念醫院內科 |  |
| 住院醫師 | 2011年10月至2014年6月 |
|  |  |

**專科證書：**

* 內科專科 (內專醫字第010230號)
* 心臟內科專科 ((105)中心專醫字第019號)
* 介入性心臟專科 (No.0809)
* 重症照護專科 (重聯專字第03306號)

**專長：**

* 一般心臟內科
* 心臟超音波
* 心臟衰竭
* 心臟重症照護

**其他經歷：**

* 高雄長庚心臟內科加護病房主任 (2024年10月迄今)
* 台灣心臟學會急重症加護委員 (2024年7月迄今)
* 中華民國醫用超音波醫學會理事 (2024年11月迄今)

|  |  |  |
| --- | --- | --- |
| 姓名: | 黃金洲Chin-Chou Huang, MD, PhD |  |
| 地址: | 台北市北投區石牌路二段201號臺北榮民總醫院 |
| **目前職位:** |  | |
|  | 國立陽明交通大學內科學科教授  國立陽明交通大學藥理學科教授  臺北榮民總醫院內科部心臟內科主治醫師  中華民國血脂及動脈硬化學會副秘書長  台灣血脂衛教協會理事  台灣醫學教育學會副秘書長  財團法人心臟醫學研究發展基金會副秘書長  高級心臟救命術指導員(ACLS instructor)  中華民國心臟學會專科指導醫師  中華民國重症醫學會專科指導醫師 | |
| **學歷:** |  | |
|  | 國立陽明大學醫學系醫學士 | |
|  | 國立陽明大學藥理研究所博士 | |
| **經歷:** |  | |
|  | 臺北榮民總醫院內科部住院醫師  臺北榮民總醫院內科部心臟內科總醫師  德國柏林心臟醫學中心(German Heart Institute Berlin)研究員 | |
|  |  | |

**Professor Jinho Shin**

**MD, MS, PhD**

Professor Jinho Shin is Professor and Chief of Cardiology at the Division of Cardiology, Department of Internal Medicine, at Hanyang University Seoul Hospital, Seoul, Korea, a role he has held since 2013. Prof. Shin undertook his medical degree at Hanyang University College of Medicine, completing his MS and PhD at the same institution.

In 2002, Prof. Shin was a Clinical Fellow at Weil Cornell Medical College, New York, USA before returning to Korea to take up an Assistant Professor role the following year.

In addition to his role at Hanyang University Seoul Hospital, Prof. Shin is coordinator of the Guideline Committee for the Korean Society of Hypertension and Director of the PaikNam Cardiovascular Center. He is a member of a number of societies and has published extensively on his research interests.

National Defense Medical Center



**Speaker: Dr. Po-Shun Hsu**

**Nationality: Taiwan**

**E-mail:** [**hsuposhun@gmail.com**](mailto:hsuposhun@gmail.com)

**Title**

1. Attending Surgeon of Cardiovascular Surgery, Tri-Service General Hospital, 2010~present
2. Ward Director of Division of Cardiovascular Surgery, Tri-Service General Hospital, 2016~present

**Medical Education**

1. Degree: M.D., School of Medicine, National Defense Medical Center, Taiwan
2. Assistant Professor, Division of Surgery, National Defense Medical Center, 2018

**Post-Graduate Training**

1. Resident of Surgery, Tri-Service General Hospital, Taipei, Taiwan, 2005-2007
2. Resident in division of Cardiovascular Surgery, Tri-Service General Hospital, Taipei, Taiwan, 2007-2009
3. Chief Resident in division of Cardiovascular Surgery, Tri-Service General Hospital, Taipei, Taiwan, 2009-2010

**Board Certification**

1. Diplomate, Taiwan Society of Medical Examiners (No.036044)
2. Diplomate, Taiwan Society of Surgery (No.5931)
3. Diplomate, Taiwan Society of Thoracic and Cardiovascular Surgery (No. 414)
4. Instructor, Taiwan Society of Thoracic and Cardiovascular Surgery (No. 232)
5. Diplomate, Taiwan Society of Cardiology Surgery (No.1437)
6. Diplomate, Taiwan Society of Vascular Surgery (No. S461)
7. Diplomate, Taiwan Society of Critical Care Medicine (No. 3580)
8. Instructor, Taiwan Society of Critical Care Medicine (No. 1015)

**Specialties**

1. Ventricular assist device surgery
2. ECMO intervention and surgery
3. Heart failure surgery
4. Coronary artery bypass graft surgery
5. Valve replacement surgery
6. Aortic open and endovascular surgery
7. Peripheral intervention and surgery
8. AV-Access intervention and surgery
9. Post-cardiac surgery intensive critical care

**CURRICULUM VITAE**

Hsuan-Fu Kuo, MD., MSc. (郭炫孚 醫師)

medsnail@hotmail.com / +886-933-590-957

**Current Position:**

Attending physician, Division of Cardiology, Department of Internal Medicine, Kaohsiung Medical University Hospital

Attending physician, Department of Internal Medicine, Kaohsiung Municipal Ta-Tung Hospital

Adjunct assistant professor, School of Medicine, College of Medicine, Kaohsiung Medical University

**Date & Place of Birth:**

October 28, 1976, Kaohsiung, Taiwan, R.O.C.

**Education:**

1995 - 2002, M.D, College of Medicine, Kaohsiung Medical University.

2008 - 2013, MSc, Graduate Institute of Medicine, College of Medicine, Kaohsiung Medical University.

**Clinical Training:**

2001.6 - 2002.5, Internship, Kaohsiung Medical University Hospital

2004.8 - 2007.7, Resident, Department of Internal Medicine, Kaohsiung Medical University Hospital

2007.8 - 2009.7, Chief Resident, Division of Cardiology, Department of Internal Medicine, Kaohsiung Medical University Hospital

2014.5 - 2014.7, Clinical Research Fellow, Heart Disease Center, Kurashiki Central Hospital, Japan (supervised by Kazuaki Mitsudo)

**Professional Appointment:**

2009.08 - present, Attending physician, Division of Cardiology, Department of Internal Medicine, Kaohsiung Medical University Hospital

2010.03 - present, Attending physician, Department of Internal Medicine, Kaohsiung Municipal Ta-Tung Hospital.

2019.02 - present, Adjunct assistant professor, School of Medicine, College of Medicine, Kaohsiung Medical University

**Board Certification:**

2002, Registered Physician – Taiwan

2007, Board of Internal Medicine – Taiwan

2009, Board of Cardiology – Taiwan

2010, Board of Critical Care Medicine – Taiwan

2010, Board of Interventional Cardiology – Taiwan,

**Graduate Thesis:**

Master of Science: “Effects of Cholesterol on Inward Calcium Channel in H9c2 Cells”, supervised by Prof. Wen-Ter Lai

**Curriculum Vitae**

**Ruey-Feng Chang (**張瑞峰**), Ph.D.**

學歷

1984.06 國立成功大學電機工程學系學士

1988.06 國立清華大學計算機管理決策研究所碩士

1992.01 國立清華大學資訊科學研究所博士

現職及經歷

1992-2000 國立中正大學資訊工程學系暨研究所副教授

2000-2006 國立中正大學資訊工程學系暨研究所教授

2003-2006 國立中正大學資訊工程學系暨研究所系主任

2006- present 國立台灣大學資訊工程學系暨研究所教授

生醫電子與資訊學研究所教授

資訊網路與多媒體研究所教授

2018- present 國立台灣大學生醫電子與資訊學研究所所長

榮譽

2004 國科會傑出研究獎

2009 國科會傑出學者研究計畫

顧問

2018.01- present 資策會數位轉型研究所人工智慧乳房病變計畫顧問

2019.07- present 彰化基督教醫院顧問

2019.07- present 中國醫藥大學附設醫院醫療智慧中心顧問

2020.02- present 彰化基督教醫院人工智慧發展中心首席AI技術長

**Curriculum Vitae**

**Name: 李美靜 Lee Mei-Ching**

**現職**

國泰醫院神經內科主治醫師

輔仁大學醫學系臨床助理教授

**Education and Training**

臺灣大學醫學系

臺大醫學院分子醫學研究所 碩士在職專班遺傳諮詢組

**Board Certification**

神經專科醫師

老年醫學專科醫師

**經歷**

台大醫院神經部住院醫師

國泰醫院神經內科總醫師

台大醫院神經部兼任主治醫師

聖母醫護管理專科學校兼任講師

教育部部定講師

**專科學會**

台灣神經學會會員

台灣腦中風學會會員

老年醫學會會員

中華民國醫用超音波學會認證醫師

**Curriculum Vitae**

**Kuang-Tso Lee M.D.**

**Education:**

Doctor of Medicine, Chang Gung University, Taiwan

**Present Position:**

Attending Cardiologist, Assistant Prof, Chang Gung Memorial Hospital, Linkou

Assistant Supervisor of Cardiac Intensive Care Unit

Director of Taiwan Society of Echocardiography: 2018 ~

Certification of Critical Care Medicine

Certification of Cardiovascular Interventions

**Past Experiences:**

Aug 2012 ~ July 2014: Cardiology Fellowship, Chang Gung Memorial Hospital

Aug 2009 ~ July 2012: Internal Medicine Residency, Chang Gung Memorial Hospital

**Clinical** **Interests:**

Cardiac Critical Care

Echocardiography for Structural Heart Disease and Mechanical Cardiac Support

Interventional Echocardiography

**Publications:**

1. Chan CC1, **Lee KT1**, Ho WJ, Chan YH, Chu PH. Levosimendan use in patients with acute heart failure and reduced ejection fraction with or without severe renal dysfunction in critical cardiac care units: a multi-institution database study. Ann Intensive Care. 2021 Feb 8;11(1):27. doi: 10.1186/s13613-021-00810-y. PMID: 33555483; PMCID: PMC7869075.
2. **Lee KT1**, Chang SH1, Kuo CF, Chiou MJ, Wen MS. Incidence and Time Course of Symptomatic Paroxysmal Supraventricular Tachycardia During Pregnancy: A Nation-Wide Database Study. *Acta Cardiol Sin*. 2020;36(1):44‐49. doi:10.6515/ACS.202001\_36(1).20190707A
3. Chan YH1, **Lee KT1**, Kao YW, Huang CY, Chen YL, Hang SC, Chu PH\*. The comparison of non-vitamin K antagonist oral anticoagulants versus well-managed warfarin with a lower INR target of 1.5 to 2.5 in Asians patients with non-valvular atrial fibrillation. PLoS One. 2019 Mar 18;14(3):e0213517. 1081234388.

個人簡歷

姓名: 雷青熒 (Chin-Ying Ray) 藥師

工作單位與職稱:

林口長庚醫院臨床藥學科 心臟科臨床藥師

學歷:

1976/09-1981/06 中國醫藥大學 藥學系畢業

1999/09-2002/06 長庚大學 基礎醫學研究所畢業

工作經歷 :

1982-1986 藥劑科審核藥師

1986-1988 助理臨床藥師

1988- 迄今 心臟內科臨床藥師

2003-迄今 長庚科技大學藥理學 講師

專長:

心臟科藥物治療學

**CURRICULUM VITAE**

**NAME：** Yu-Ning Hu

**ELECTRONIC ADDRESS：**

**E-mail：** [windermere0209@gmail.com](mailto:windermere0209@gmail.com)

**Address：** 138 Sheng-Li Road, Tainan 704, Taiwan

**CLINICAL APPOINTMENT**

*08/2016~present* Attending Surgeon in Cardiovascular Surgery, National Cheng Kung University Hospital, Taiwan

*11/2014~07/2016* Attending Surgeon in Cardiovascular Surgery, National Cheng Kung University Hospital, Dou-Liou Branch, Taiwan

**ACADEMIC APPOINTMENT**

*08/2020~present* Assistant Professor,National Cheng Kung University

*08/2012~present* Clinical instructor, Department of Surgery, National Cheng Kung University Hospital, Tainan, Taiwan

**EDUCATION AND TRAINING**

1. *12/2016~05/2017* Clinical Visiting Scholar, Division of Cardiothoracic Surgery, Department of Surgery, The Chinese University of Hong Kong, Hong Kong
   * Mentors: Prof. Song Wan
   * Specialization in valve reconstruction surgery
2. *08/2013~10/2014* Chief Resident, Cardiovascular Surgery, National Cheng Kung University Hospital, Tainan, Taiwan

個人簡歷

**姓名：吳宏彬**

**現職：**

中國醫藥大學附設醫院心臟血管系主治醫師

**學經歷：**

中國醫藥大學中醫學系雙主修畢

中國醫藥大學 生物醫學研究所 碩士

中國醫藥大學附設醫院心臟內科研究醫師

中國醫藥大學附設醫院心臟內科主治醫師

中華民國內科專科醫師

中華民國心臟學會心臟內科專科醫師

中華民國心律醫學會專科會員

中華民國心律醫學會植入式心臟儀器委員會委員

**專長：**

心臟超音波、心律不整燒灼手術與心臟節律器植入手術

**吳彥雯個人資料表**

1. 基本資料

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| 中文姓名 | 吳彥雯 | 英文姓名 | Wu, Yen-Wen | | | | |
| (Last Name) (First Name) (Middle Name) | | | | |
| 國籍 | 中華民國，臺灣 | 性別 | □男 ■女 | | 出生日期 | 1972 年05月 02日 | |
| 聯絡地址 | (公) 220 新北市板橋區南雅南路二段21號 亞東紀念醫院 | | | | | | |
| 聯絡電話 | 02-8966-7000 ext: 1090, 4450 | | | | | |  |
| 傳真號碼 | (02) 7728-2378 | | | E-MAIL | [wuyw0502@gmail.com](mailto:wuyw0502@gmail.com); wuyw@ntuh.gov.tw | | |

1. 主要學歷

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| 畢／肄業學校 | 國別 | 主修學門系所 | 學位 | 起訖年月 |
| 國立臺灣大學 | 我國 | 醫學系 | 學士 | 1990/09至1997/06 |
| 國立臺灣大學 | 我國 | 臨床醫學研究所碩士班 | 碩士 | 2000/09 至2002/06 |
| 國立臺灣大學 | 我國 | 臨床醫學研究所博士班 | 博士 | 2002/09至 2009/01 |

1. 現職及與專長相關之經歷

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| 服務機關 | 服務部門／系所 | | 職稱 | 起訖年月 |
| **現職：**亞東紀念醫院 | 心臟血管醫學中心 | | 主任 | 2020/07迄今 |
| 心臟血管內科 | | 主任 | 2012/08迄今 |
| 心臟內科/核子醫學科 | | 主治醫師 | 2012/03迄今 |
| 國立陽明交通大學 | 醫學院醫學系 | | 兼任教授 | 2018/08迄今 |
| 臺大醫院 | 核子醫學部/心臟內科 | | 兼任主治醫師 | 2012/03 迄今 |
| **經歷：**亞東紀念醫院 | 心臟血管醫學中心 | | 副主任 | 2015/07 至2020/06 |
| 核子醫學科 | | 主任 | 2012/03 至2015/06 |
| 國立陽明大學 | 醫學院醫學系 | | 教授 | 2017/08 至2018/07 |
| 陽明大學 | 醫學院醫學系 | | 副教授 | 2013/02 至2017/07 |
| 臺大醫院 | 核子醫學部/心臟內科 | | 主治醫師 | 2004/07 至 2007/10  2008/11 至 2010/02 |
| 國立臺灣大學 醫學院 放射線科 | | | 臨床助理教授 | 2009/02至 2012/02 |
| 兼任助理教授 | 2008/02 至2009/02  2012/03 至2013/02 |
| (借調) 台大醫院新竹分院影像醫學部/心臟內科 | | | 主任/主治醫師 | 2010/03 至 2012/02 |
| (全時支援) 台大醫院雲林分院 核子醫學科 | | | 主任 | 2007/11 至2008/10 |
| 國立臺灣大學 | | 醫學院 放射線科 | 兼任講師 | 2005/08 至 2008/01 |
| 日本京都大學/日本北海道大學/附設醫院 | | 核醫學分野 | Foreign Collaborate Investigator | 2005/11 至 2006/11 |
| 臺大醫院 | | 核子醫學部 | 住院醫師 | 2002/07 至 2004/06 |
| 臺大醫院 | | 心臟內科 | 臨床研修員 | 2000/07 至 2002/06 |
| 臺大醫院 | | 內科部 | 住院醫師 | 1997/07 至 2000/06 |

四、 專長

|  |  |  |  |
| --- | --- | --- | --- |
| 1. Cardiology | 1. Internal Medicine | 1. Nuclear Medicine | 1. Molecular Imaging |

五、 學術團體/證書

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| 資 格 | 證 書 字 號 | 級 別 | 核 發 機 關 | 起算(核發)年月 |
| **International Medical Society Memberships** | European Society of Cardiology | Fellow (FESC) | 2016/01 迄今 |
| American College of Cardiology (ACC) | Fellow (FACC) | 2019/11迄今 |
| American Heart Association (AHA) | Membership |  |
| Society of Nuclear Medicine and Molecular Imaging (SNMMI) | Membership |  |
| American Society of Nuclear Cardiology (ASNC) | Membership |  |
| 學術團體/職務 | 名 稱 | | 擔 任 職 務 | 起 迄 年 月 |
| 現任 | 醫學會 | | | |
| 中華民國核醫學會 | | 常務理事 | 2018/11至2022/10 |
| 中華民國核醫學會 (核醫心臟委員會) | | 主任委員 | 2018/11至2022/10 |
| 台灣動脈硬化暨血管病醫學會 | | 理事 | 2018/07至2021/06 |
| 中華民國血脂及動脈硬化學會 | | 理事 | 2018/10至2021/09 |
| 台灣高血壓學會 | | 理事 | 2019/01至2023/12 |
| 台灣血脂衛教協會 | | 秘書長 | 2019/03至2022/02 |
| 台灣健康醫學協會 | | 理事 | 2020/06至2023/06 |
| 中華民國心臟學會 (跨領域合作小組) | | 召集人 | 2020/08至2022/05 |
| 財團法人中華民國心臟基金會 | | 董事 | 2020/11至2022/10 |
| 學會雜誌 | | | |
| 中華民國心臟學會雜誌:  (Acta Cardiologica Sinica, ACS) SCI journal | | 副總編輯 | 2015/01 迄今 |
| 中華民國核子醫學暨分子影像雜誌: (Annals of Nuclear Medicine and Molecular Imaging, ANMMI) | | 副總編輯 | 2015/01 迄今 |
| 海峽循環學雜誌: (Strait Circulation Journal) | | 編輯委員 | 2018/07迄今 |
|  | Korean Circulation Journal (KCJ) SCI journal | | 編輯委員 (Editorial Board) | 2020/12-2022/10 |

六、獲獎

1. 全美核子醫學第五十四屆年會青年學者研究獎 (2007/06/06)。
2. 台灣動脈硬化暨血管病醫學會(第二屆)醫學論文獎首獎 (2007/12/01)。
3. 中華民國心臟學會第40屆青年學者獎 (2010/05/20)。
4. 台灣動脈硬化暨血管病醫學會(第五屆)醫學論文獎第二名 (2010/11/28)。
5. 中華民國核醫學學會2013年年會海報論文獎首獎及第二名 (2013/10/26)。
6. 第十三屆有庠傑出教授獎 (醫療技術) (2015/8/14)。

七、論文著作：2003~2021/04 SCI publication: 200, Selected publications (1st and correspondence\*)

1. Wu YW, Yen RF, Chieng PU, Huang PJ. [Tl-201 myocardial SPECT in differentiation of ischemic from nonischemic dilated cardiomyopathy in patients with left ventricular dysfunction.](http://www.ncbi.nlm.nih.gov/pubmed/12900741?itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum&ordinalpos=48) J Nucl Cardiol. 2003 Jul-Aug;10(4):369-74.
2. Wu YW, Yen RF, Lee CM, Ho YL, et al. Diagnostic and prognostic value of dobutamine thallium-201 single-photon emission computed tomography after heart transplantation. J Heart Lung Transplant. 2005 May;24(5):544-50.
3. Wu YW, Huang PJ, Lee CM, Ho YL, et al. Assessment of myocardial viability using F-18 fluorodeoxyglucose/Tc-99m sestamibi dual-isotope simultaneous acquisition SPECT: comparison with Tl-201 stress-reinjection SPECT. J Nucl Cardiol. 2005 Jul-Aug;12(4):451-9.
4. Wu YW, Kao HL, Chen MF, et al. Characterization of plaques using 18F-FDG PET/CT in patients with carotid atherosclerosis and correlation with matrix metalloproteinase-1. J Nucl Med. 2007 Feb;48(2):227-33.
5. Wu YW, Tadamura E, Yamamuro M, et al. Comparison of contrast-enhanced MRI with (18)F-FDG PET/201Tl SPECT in dysfunctional myocardium: relation to early functional outcome after surgical revascularization in chronic ischemic heart disease. J Nucl Med. 2007 Jul;48(7):1096-103.

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**Education:**

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2011-2015 Department of Public Health, National Yang-Ming University, Taipei, Taiwan

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1994-2001 National Yang-Ming University, Taipei, Taiwan

**Clinical Interests:**

Heart failure, Structural heart intervention, Coronary intervention, Echocardiography

**Experience:**

1999-2001 Intern, Veterans General Hospital, Taipei, Taiwan

2002-2005 Resident Physician, Department of Medicine, Veterans General Hospital, Taipei, Taiwan

2005-2009 Fellow in Cardiology, Veterans General Hospital, Taipei, Taiwan

2007-2008 Chief Resident in Internal Medicine, Veterans General Hospital, Taipei, Taiwan

2008-2008 Researcher, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan

2009-2010 Researcher, Graduate School of Medical Sciences, Chinese University of Hong Kong

2012-2018 Member of Heart Failure Committee, Taiwan society of cardiology

2012-2018 Member of Pulmonary artery hypertension working group, Taiwan society of cardiology

2014-2016 Deputy Secretary-General, Taiwan society of cardiovascular intervention

2015-2015 Trainee of structural heart intervention, Mainz University Medical Center, Germany

2018-2020 Deputy Secretary-General, Taiwan society of cardiology

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**Professional societies and organizations:**

Member, Taiwan Society of Internal Medicine

Member, Taiwan Society of Cardiology

Member, Taiwan Society of Lipids and Atherosclerosis

Member, Taiwan Society of Critical Care Medicine

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**Licensure and Certification:**

2001.9 Board of General Medicine

2003.1 Board of Internal Medicine

2007.11 Board of Cardiovascular Subspecialty

2007.12 Board of Critical Care Medicine

2007.11 Board of Interventional Cardiologist, Taiwan Society of Cardiology

2017.4 Instructor of Fellowship Training in Clinical Cardiology, Taiwan Society of Cardiology

**Academic Appointments:**

2008-2013 Instructor in Faculty of Medicine, National Yang-Ming University, Taipei, Taiwan

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**Professional Memberships**

Member of the Taiwan Internal Medicine Society

Member of Medical Oncology of the Chinese Oncology Society

Member of the Hematology Society of Taiwan

Member of the Taiwan Society of Blood and Marrow Transplantation

**Publications:**

1. Tan TD. Successful Eradication of Rituximab-Refractory EBV-Related Post-Transplant Lymphoproliferative Disorder after Second Haplo-Identical Allogeneic Hematopoietic Stem Cell Transplantation for Very Severe Aplastic Anemia. Ann Case Rep: 7:926. [WWW.doi.org/10.29011/2574-7754.100926](http://WWW.doi.org/10.29011/2574-7754.100926). [www.gavinpublishers.com](http://www.gavinpublishers.com)
2. Yang KL, Tan TD, et al. Discovery of the novel HLA-DRB1\*07:136 allele in a Taiwanese patient. HLA. 2022; 100(3): 285-286. Doi: 10.1111/tan.14685
3. Prognostic Factors of Poor Survival in Newly Diagnosed Advanced-Stage Hodgkin Lymphoma in Taiwan: A Nationwide Retrospective Study. HemaSphere 2021. European Hematology Association annual meeting, 2021.
4. Venetocalx and Posaconazole plus standard dose Cytarabine for newly diagnosed AML. Blood. American Society of Hematology annual meeting, 2020.
5. Tan TD, Chiou LW, Wu JS, Lee MY, Huang YY, Chen SS, and LuSM. Long-term rather than short-term survival benefit in mantle cell lymphoma patients treated with intensive chemo-immunotherapy and hematopoietic stem cell transplantation in real world experience. Haematol Int J 2020; 4(2): 00072.
6. Tan TD, Hong YC, Li SS, Yu JT, Sung YC, Wang PN, Teng CL, The multiple myeloma working group, Hematology’s Society of Taiwan. Lenalidomide with dexamethasone to multiple myeloma patients relapsing from Bortezomib-based induction therapies: a prospective, observational study. Chin J Physiol 2020; 63: XX-XX.
7. Tan TD, Chiou LW. Frontline treatment with Venetocalx, Posaconazole, and standard dose Cytarabine or Azacitidine followed by allogeneic hematopoietic stem cell transplantation for newly diagnosed acute myeloid leukemia. HemaSphere 2020, in press.
8. Tan TD, Chiou LW. Intensive chemo-immunotherapy plus hematopoietic stem cell transplantation or non-intensive treatment for mantle cell lymphoma –Real world clinical outcome in single institute experience. HemaSphere 2020, in press.
9. Huang TC, Huang SY, Yao M, Tan TD, et al. Autologous stem cell transplantation in multiple myeloma: post-transplant outcomes of Taiwan Blood and Marrow Transplantation Registry. J of the Formosan Medical Association 2019; 118: 471-480.
10. Tan TD, Chiou LW, Wu MC, Wu JS, Lee MY, Huang YY, Chen SS. The Impact of First Complete Remission by PET-CT and Time to Next Treatment on Survival of Follicular lymphoma patients. Clinical Hematology International. In Press, Corrected Proof, Available Online: 7 June 2019.
11. Co-authors. Prognostic value of end-of-induction PET response after first-line immune-chemotherapy for follicular lymphoma (GALLIUM): secondary analysis of a randomized, phase 3 trial. Lancet Oncol 2018; 19(11): 1530-1542.
12. Huang TC, Huang SY, Yao M, Lin CY, Hwang WL, Tan TD et al. Autologous stem cell transplantation in multiple myeloma: post-transplant outcomes of Taiwan Blood and Marrow Transplantation Registry. J Formos Med Assoc. 16 August 2018.
13. Kuo CY, Wang PN, Hwang WL, Tan TD et al. Safety and efficacy of Nilotinib in routine clinical practice in patients with chronic myeloid leukemia in chronic or accelerated phase with resistance or utterance to Imatinib: result from the NOVEL study. Therapeutic Advances in Hematology 2018; 9(3): 65-78.
14. Hsu YT, Tsai HJ, Chang JS, Li SS, Tan TD et al. Stem cell transplantation for T-cell lymphomas in Taiwan. Bone Marrow Transplantation 2018; 53: 993-1000.
15. Tran-Der Tan, Mau-Ching Wu , Lun-Wei Chiou , Peng-Yu Chen , Ja-Shing Wu. FRONTLINE AUTOLOGOUS STEM CELL TRANSPLANTATION FOR AGGRESSIVE B AND T CELL LYMPHOMA. Haematologica 2016 Jun sup. T.D. Tan, M.-C. Wu, J.L.W. Chiou, A. C. Feng. HBV Clearance in HBsAg+ Patients after Allogeneic Stem Cell Transplantation is Heralded by GVHD of Liver. Haematologica 2015 Jun sup.
16. Ching-Yuan Kuo Po-Nan Wang, Wen Li Hwang, Tran-Der Tan, et al. Safety and efficacy of Nilotinib in patients with chronic phase or accelerated phases Philadelphia chromosome positive chronic myelogenous leukemia with resistance or intolerance to Imatinib Mesylate: results from the multi-center, observational NOVEL study in Taiwan. Blood (ASH Annual Meeting Abstracts) 2015: Poster Abstract.
17. Ko BS, Tan TD, et al. Guidelines for treating iron overload in myelodysplastic syndromes: a Taiwan consensus statement. Int J Hematology 2014; 100(1): 7-15.
18. Win KT, Lee MY, Tan TD et al. Nasopharyngeal alveolar rhabdomyosarcoma expressing CD56: a mimicker of extra nodal natural killer/T-cell lymphoma. Int J Clin Exp Pathol 2013; 7(1): 451-455.
19. T.D. Tan, M.-C. Wu, J.L.W. Chiou. Outcome of second allo-transplantation for relapsed hematologic malignancies after stem cell transplantation. Blood (ASH Annual Meeting Abstracts) 2012:120 Abstract 4461.
20. T.D. Tan, M.-C. Wu, J.L.W. Combination of gut GVHD and CMV enterocolitis in allogeneic hematopoietic stem cell transplant patients--10 years’ experience of a single institution. Bone Marrow Transplantation Vol 47, Supplement 1, S164, Apr 2012
21. T.D. Tan. Pulmonary Complications in Hematopoietic Stem Cell Transplant Patients--A Single Institution Experience. Bone Marrow Transplantation Vol 47, Supplement 1, S200, Apr 2012
22. T.D. Tan, M.-C. Wu, J.L.W. Chiou. CD56-Positive Acute Myeloid Leukemia Has Inferior Outcome After Allogeneic Hematopoietic Stem Cell Transplantation (Abstract). Blood (ASH Annual Meeting Abstracts) 2011 118: Abstract 4493.
23. T.D. Tan, M.-C. Wu, J.L.W. Chiou. The impact of velcade treatment on survival of relapsed myeloma patients – A single institution experience. Haematologica 96(s2), p535, 2011.

**CURRICULUM VITAE**

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**University Education**

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| 1981-1988 | Bachelor of Medicine, Department of Medicine, College of Medicine, National Taiwan University |
| 1995-2000 | Ph.D., Section of Preventive Medicine, Institute of Epidemiology, College of Public Health, National Taiwan University |

**Professional Education and Certifications**

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| 1987-1988 | Internship in National Taiwan University Hospital |
| 1990-1993 | Resident in Department of Internal Medicine, National Taiwan University Hospital |
| 1993-1995 | Cardiology Fellowship in Section of Cardiology & Chief Resident in Department of Internal Medicine, National Taiwan University Hospital |
| 1995-Date | Attending Physician in Department of Internal Medicine, National Taiwan University Hospital |
| 2001/10- 2002/4 | Researcher, National Institute of Health and Nutrition, Tokyo, Japan, Directed by Prof. Heizo Tanaka |
| 2006-2007 | Visiting Scholar, Dept. of Nutrition, Harvard School of Public Health and Channing Laboratory, Brigham and Women's Hospital, USA, Directed by Prof. Walter Willett & Prof. Frank B. Hu |
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**Education and Experience**

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**2013/9 –2014/8 Brigham and Women's Hospital, Harvard University, MA, USA**

*Research Fellow*, Cardiac Muscle Research Laboratory

**2019/8 – Graduate Institute of Clinical Medicine, National Cheng Kung University, Taiwan** *PhD candidate*

**Professional Experience**

**2014/8- Chi-Mei Medical Center, Tainan, Taiwan**

*Attending physician*, Department of Cardiology

**2020/08-**   **Southern Taiwan University of Science and Technology**

*Associate Professor,* Department of Biotechnology

**Awards and Honors**

**2017-2019**  The best annual research award in Chi-Mei Medical Center

**2018** TA-YOU WU MEMORIAL AWARD **(吳大猷先生紀念獎)**

**2017** Young investigator award in Taiwan Society of Cardiology

**Publications (120 SCI papers, presented from 2022 till now)**

**Publications**

1. Wu NC…. **Chang WT** (Corresponding author). Clinical Features and Outcomes of Immune Checkpoint Inhibitor-Associated Cardiovascular Toxicities. *Acta Cardiol Sin*. 2022 Jan;38(1):39-46|
2. Lu LS, Wu YW, Chang TC, **Chang WT**….et al. Risk Management for Radiation-Induced Cardiovascular Disease (RICVD): The 2021 Consensus Statement of the Taiwan Society for Therapeutic Radiology and Oncology (TASTRO) and Taiwan Society of Cardiology (TSOC). *Acta Cardiol Sin*. 2022 Jan;38(1):1-12.
3. Huang H, **Chang WT**, Huang CC. High-Spatiotemporal-Resolution Visualization of Myocardial Strains Through Vector Doppler Estimation: A Small-Animal Study. IEEE Trans Ultrason Ferroelectr Freq Control. 2022 Feb 2;PP.
4. **Chang WT**….Hsu CH. Dynamic Changes in miR-21 Regulate Right Ventricular Dysfunction in Congenital Heart Disease-Related Pulmonary Arterial Hypertension. Cells. 2022;11(3):564
5. Hong CS….**Chang WT (Corresponding author)**. Association of gonadotropin-releasing hormone therapies with venous thromboembolic events in patients with prostate cancer: A national cohort study. *Frontiers in Cardiovascular Medicine.* 2022 (accepted)
6. **Chang WT**, Chen PW, Lin HW, Kuo YH, Lin SH, Li YH. [Risks of Aromatase Inhibitor-Related Cardiotoxicity in Patients with Breast Cancer in Asia.](https://pubmed.ncbi.nlm.nih.gov/35158776/) Cancers (Basel). 2022 Jan 20;14(3):508.
7. **Chang WT** et al. The impact of Sildenafil on ischemic outcomes in patients with pulmonary hypertension – a nationwide cohort study. *Acta Cardiologica Sinica*. 2022 Sep;38(5):623-630
8. **Chang WT** et al. Future perspectives of pulmonary hypertension treatment" *Acta Cardiologica Sinica*. 2022
9. **Chang WT** et al. Deletion of miR-21 impairs neovascularization following limb ischemia: from bedside to bench. *Frontiers in Cardiovascular Medicine.* 2022
10. **Chang WT** et al. Dapagliflozin Protects Against Doxorubicin-induced Cardiotoxicity by Restoring STAT3. *Archives of Toxicology.* 2022
11. **Chang WT** et al. [An artificial intelligence approach for predicting cardiotoxicity in breast cancer patients receiving anthracycline.](https://pubmed.ncbi.nlm.nih.gov/35876889/) Arch Toxicol. 2022 Oct;96(10):2731-2737
12. **Chang WT** et al. [Effects of STAT3 on aging-dependent neovascularization impairment following limb ischemia: from bedside to bench.](https://pubmed.ncbi.nlm.nih.gov/35696641/) Aging (Albany NY). 2022 Jun 13;14(11):4897-4913. doi: 10.18632/aging.204122. Epub 2022 Jun 13.
13. Pilz PM, Ward JE, **Chang WT** et al. [Large and Small Animal Models of Heart Failure With Reduced Ejection Fraction.](https://pubmed.ncbi.nlm.nih.gov/35679365/) Circ Res. 2022 Jun 10;130(12):1888-1905. doi: 10.1161/CIRCRESAHA.
14. **Chang WT**, et al. MiR-21 upregulation exacerbates pressure overload-induced cardiac hypertrophy in aged hearts. *Aging (Albany NY)*. 2022
15. Chang HY, Chen PW, **Chang WT**, Yeh JK, Liu PY, Hsu CH, Lin CC. Evolutionary changes in thrombus features on computed tomography-an effective approach for identifying subacute pulmonary embolism. J Vasc Surg Venous Lymphat Disord. 2022 Aug 9:S2213-333X(22)00343-2
16. Gupta S, Strohbehn IA, Wang Q, Hanna PE, Seethapathy R, Prosek JM, Herrmann SM, Abudayyeh A, Malik AB, Loew S, Carlos CA, **Chang WT**, et al. [Acute Kidney Injury in Patients Receiving Pembrolizumab Combination Therapy versus Pembrolizumab Monotherapy for Advanced Lung Cancer.](https://pubmed.ncbi.nlm.nih.gov/35964800/) *Kidney Int.* 2022 Aug 11:S0085-2538(22)00611-1
17. **Chang WT**, Lin YW, Chen CY, Chen ZC, Shih JY, Wu CC, Luo CY, Liu PY. [Mineralocorticoid Receptor Antagonists Mitigate Mitral Regurgitation-Induced Myocardial Dysfunction.](https://pubmed.ncbi.nlm.nih.gov/36078158/) *Cells.* 2022 Sep 3;11(17):2750
18. Lee MC, Liao CT, Feng IJ, Yu T, **Chang WT, et al.** [Recurrent thromboembolism, bleeding, and mortality in Asian patients with venous thromboembolism receiving different oral anticoagulants: A nationwide analysis.](https://pubmed.ncbi.nlm.nih.gov/36123930/) *Medicine (Baltimore)*. 2022 Sep 16;101(37):e30412
19. **Chang WT**, Hong CS, Hsieh KL, Chen YC, Ho CH, Shih JY, Kan WC, Chen ZC, Lin YC. [Regular use of aspirin is associated with a lower cardiovascular risk in prostate cancer patients receiving gonadotropin-releasing hormone therapy.](https://pubmed.ncbi.nlm.nih.gov/36172150/) *Front Oncol.* 2022 Sep 12;12:952370.
20. Liao CT, Toh HS, Yang CT, Hsu CY, Lee MC, **Chang WT**, et al. [Economic evaluation of new blood pressure target for hypertensive patients in Taiwan according to the 2022 hypertension clinical practice guidelines of the Taiwan society of cardiology: a simulation modeling study.](https://pubmed.ncbi.nlm.nih.gov/36229527/) *Hypertens Res.* 2022 Oct 14
21. Association of sepsis-induced cardiomyopathy and mortality: a systematic review and meta-analysis. Lin YM, Lee MC, Toh HS, **Chang WT**..et al. Ann Intensive Care. 2022 Dec 13;12(1):112

Abstract Taiwanese Society of Cardiology, may 2025

Professor Michel Jadoul

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Chronic Kidney Disease (CKD) is now recognized as an independent risk factor for various adverse outcomes , including kidney failure, cardiovascular outcomes such as MI, stroke and AFib, as well as death (1). CKD is expected to become the fifth leading global cause of death by 2040 and the awareness of this major burden is limited (2). Thus, the early recognition of CKD, especially in high risk patients, such as those with diabetes, cardiovascular disease or hypertension, is crucial to improve outcomes.

This appears particularly important because the field of nephroprotection is moving rapidly. The revolution of SGLT2-inhibitors has been one of the major triggers of the 2024 update of the Kidney Disease : Improving Global Outcomes (KDIGO) CKD Guideline (1). EMPA-Kidney was the largest study included in the meta-analysis of trials with SGLT2-i and a primary kidney outcome. SGLT2-i are now recommended by KDIGO in subjects with albuminuric CKD (1A) and suggested in those with CKD , an eGFR of 20-45 ml/min and albuminuria < 200 mg /g créatinine (2B). The long-term results of EMPA-Kidney confirm and extend the demonstrated benefits of empagliflozin treatement in CKD (3).

However, unless already treated by SGLT2-i for heart failure, patients with CKD are unlikely to be treated with these drugs if CKD is not diagnosed, based on eGFR and urinalysis (UACR). This is the time to implement the revolution of SGLT2-i as nephroprotective agents. The good news is that additional trials are underway, making the combination on nephroprotective agents (4) more and more a reality . The positive results of the Phase 2 trial with vicadrostat , an aldosterone synthase inhibitor are promising (5). The phase 3 EASY-Kidney trial has started.

Overall, for CKD patients, many of which are known to cardiologists, the future looks much brighter, but if and only if the ongoing revolution of SGLT2-i, such as empagliflozin, is widely implemented.

References

1. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int. 105(4):S117-S314 (2024).

2. Jadoul M, Aoun M, Masimango Imani M. The major global burden of chronic kidney disease. Lancet Glob Health. 2023

3. EMPA-KIDNEY Collaborative Group; William G Herrington 1, et al. N Engl J Med. 2025 Long-Term Effects of Empagliflozin in Patients with CKD

4. Michel Jadoul , Peter Rossing Nephrol Dial Transplant. 2025. The era of combination of nephroprotective agents in CKD is there

5. Katherine R Tuttle 1, et al. Lancet. 2024. Efficacy and safety of aldosterone synthase inhibition with and without empagliflozin for chronic kidney disease: a randomised, controlled, phase 2 trial

**Abstract Taiwanese Society of Cardiology, may 2025**

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Chronic Kidney Disease (CKD) is now recognized as an independent risk factor for various adverse outcomes , including kidney failure, cardiovascular outcomes such as MI, stroke and AFib, as well as death (1). CKD is expected to become the fifth leading global cause of death by 2040 and the awareness of this major burden is limited (2). Thus, the early recognition of CKD, especially in high risk patients, such as those with diabetes, cardiovascular disease or hypertension, is crucial to improve outcomes.

This appears particularly important because the field of nephroprotection is moving rapidly. The revolution of SGLT2-inhibitors has been one of the major triggers of the 2024 update of the Kidney Disease : Improving Global Outcomes (KDIGO) CKD Guideline (1). EMPA-Kidney was the largest study included in the meta-analysis of trials with SGLT2-i and a primary kidney outcome. SGLT2-i are now recommended by KDIGO in subjects with albuminuric CKD (1A) and suggested in those with CKD , an eGFR of 20-45 ml/min and albuminuria < 200 mg /g créatinine (2B). The long-term results of EMPA-Kidney confirm and extend the demonstrated benefits of empagliflozin treatement in CKD (3).

However, unless already treated by SGLT2-i for heart failure, patients with CKD are unlikely to be treated with these drugs if CKD is not diagnosed, based on eGFR and urinalysis (UACR). This is the time to implement the revolution of SGLT2-i as nephroprotective agents. The good news is that additional trials are underway, making the combination on nephroprotective agents (4) more and more a reality . The positive results of the Phase 2 trial with vicadrostat , an aldosterone synthase inhibitor are promising (5). The phase 3 EASY-Kidney trial has started.

Overall, for CKD patients, many of which are known to cardiologists, the future looks much brighter, but if and only if the ongoing revolution of SGLT2-i, such as empagliflozin, is widely implemented.

References

1. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease*. Kidney Int.* 105(4):S117-S314 (2024).
2. Jadoul M, Aoun M, Masimango Imani M. The major global burden of chronic kidney disease. *Lancet Glob Health*. 2023
3. [EMPA-KIDNEY Collaborative Group](https://pubmed.ncbi.nlm.nih.gov/?sort=date&term=EMPA-KIDNEY+Collaborative+Group%5BCorporate+Author%5D); [William G Herrington](https://pubmed.ncbi.nlm.nih.gov/?sort=date&term=Herrington+WG&cauthor_id=39453837)[1](https://pubmed.ncbi.nlm.nih.gov/39453837/#full-view-affiliation-1),  et al. *N Engl J Med*. 2025 Long-Term Effects of Empagliflozin in Patients with CKD
4. [Michel Jadoul](https://pubmed.ncbi.nlm.nih.gov/?sort=date&term=Jadoul+M&cauthor_id=39907537), [Peter Rossing](https://pubmed.ncbi.nlm.nih.gov/?sort=date&term=Rossing+P&cauthor_id=39907537)*Nephrol Dial Transplant*. 2025. The era of combination of nephroprotective agents in CKD is there

**Abstract:**

For the past 2 decades, agents targeting the prostacyclin, endothelin, and nitric oxide pathways have formed the "3 pillars" of PAH therapy. These agents are used in combination and often from the time of diagnosis. Despite maximal combination therapy, many patients may not achieve treatment targets. Recently, novel agents such as activin signalling inhibitors have gained regulatory approval. This talk will focus on novel therapeutics in PAH including how they should be incorporated into PAH treatment algorithm, and how treatment paradigms may change as therapeutic options expands.



PROGNOSTIC IMPLICATIONS OF LEFT VENTRICULAR HYPERTROPHY AND MECHANICAL FUNCTION IN FABRY DISEASE: A LONGITUDINAL COHORT STUDY  
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**Background**

The effect of different grades of left ventricular hypertrophy (LVH) and left ventricular (LV) mechanical function on the clinical outcomes in Fabry disease is unclear. We aimed to evaluate the association between the severity of LVH, LV mechanical function, and clinical outcomes in Fabry disease.

**Methods**

We conducted a retrospective cohort study from a single-center registry of adult patients with Fabry disease. LV mass index (LVMI) was measured using the area-length method by echocardiography. The severity of LVH was categorized according to LVMI using the sex-specific cutoff values recommended by the American Society of Echocardiography. LV mechanical function was measured as LV global longitudinal strain (GLS) by speckle tracking analysis and was reported as the absolute value. The primary outcome was the composite of major adverse cardiovascular events (MACE) at five years, including heart failure hospitalization, sustained ventricular tachycardia, acute ischemic stroke, and all-cause mortality.

**Results**

The study included a total of 268 patients (age 50.4 ± 15.4 years, men 46.6%) with Fabry disease (83.2% IVS4+919G>A mutation) and 106 patients (39.6%) had LVH. The 5-year MACE rates were 7.4%, 10%, and 30.5% in patients with mild, moderate, and severe LVH, respectively (P< 0.001). Moreover, there was a higher 5-year MACE rate in patients with impaired LV GLS (<16%) than those with preserved LV GLS (≥16%) (23.5% vs. 1.6%, P< 0.001). In multivariable Cox regression analysis, severe LVH was an independent predictor of poor clinical outcomes as compared with no LVH, after adjusting for age, sex, hypertension, hyperlipidemia, atrial fibrillation, renal function, average E/e’, enzyme replacement therapy, and LV GLS (adjusted hazard ratio, 11.59; 95% confidence interval, 1.19-113.41; P= 0.04). Patients with severe LVH and impaired LV GLS (<16%) represented the highest risk group when compared with the other groups (log-rank P< 0.001).

**Conclusions**

Severe LVH with impaired LV GLS is independently associated with an increased risk of MACE in Fabry disease. Sex-specific grading of LVH by LVMI and LV GLS can be applied to Fabry disease for a refined risk stratification upon the initial diagnosis.

**PROGNOSTIC VALUE OF FULLY AUTOMATED LEFT ATRIAL STRAIN IN PATIENTS WITH ASYMPTOMATIC CHRONIC SEVERE AORTIC REGURGITATION**Kang Liu, MD1 , Chieh-Mei Tsai, MD1 , Chi-Ching Huang, MD1 , Masaaki Takeuchi, MD, PhD2, Yi-Lwun Ho, MD PhD1, Li-Tan Yang, MD1  
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Objective: To examine whether fully automated left atrial (LA) strain predicted all-cause death (ACD) in patients with asymptomatic hemodynamically-significant chronic aortic regurgitation (AR).  
  
Methods: Consecutive asymptomatic patients with isolated ≥moderate-severe AR undergoing echocardiograms were retrospectively identified from 2008 through 2022 from a tertiary referral center. LA strain, including reservoir strain (LASr), contractile strain (LAScd), and conduit strain (LASct) were measured using vendor-independent analytical software (AutoStrain LA Analysis, LOT 31.0; TomTec Imaging Systems, Unterschleissheim, Germany) from apical 4 chamber view. Endpoint was ACD at medical follow-up.  
Results: Of the enrolled 359 patients (mean age 59±17 years; 19% female), mean left ventricular ejection fraction (LVEF) was 60±8%, Charlson score 0.93±1.49, LV end-systolic dimension index (LVESDi) 21.9±3.9mm/m 2 , LV end-systolic volume index (LVESVi) 40±18 ml/m 2 and LASr 36.2%. In 7% cases, tracking border was partially corrected. Median follow-up under medical treatment was 3.6 (IQR:0.86-7.37) years; during which 57 patients died. Multivariate analysis showed that older age, higher Charlson score, larger LVESDi (hazard ratio[HR] per 1mm/m 2 : 1.07), larger LVESVi (HR per 1ml/m 2 : 1.02) and lower LASr (HR per 1% increase: 0.97) were independently associated with ACD (all P≤0.036); LV longitudinal strain (LVLS), LAScd, and LASct were not. Lower LASr was also independently associated with aortic valve surgery (HR per 1% increase: 0.97) after adjusting for age, sex, Chalrson score, LVESDi and LVLS. When we classified patients according to median LASr (36.2%), Kaplan-Meier curves showed that patients having LASr< 36.2% had worse survival (P<0.0001) as compared to those having LASr ≥36.2%.  
Conclusions: Fully-automated LASr was a clinically feasible tool for predicting poor outcomes in patients with asymptomatic hemodynamically-significant chronic AR. It provides incremental prognostic value in addition to traditional factors and guideline-recommended LV parameters. Therefore, LASr may be incorporated into clinical decision making process to identify asymptomatic AR patients at high risks of death.

**A NOVEL SCORING SYSTEM COMBINING PHENYLALANINE AND LEUCINE PREDICTS 30-DAY MORTALITY OF PATIENTS WITH HEART FAILURE IN CARDIAC CARE UNIT BETTER THAN TRADITIONAL SCORES**

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**Background:** Risk scores such as Acute Physiology and Chronic Health Evaluation II (APACHE II) score are used to predict the outcome in critically ill patients. However, traditional risk scores do not provide information for intervention. In the cardiac care unit (CCU), the levels of phenylalanine and leucine amino acids (PLA) both have prognostic value as shown in previous studies. We combined these two amino acids to develop PLA score to investigate the performance of 30-day mortality prediction in CCU and to evaluate whether these measurements could help interpret the nutritional phases of critical illness.

**Methods:** We recruited 537 patients with a diagnosis of heart failure(American Heart Association stages B to D), and APACHE II scores **≥**15 in CCU for the initiation cohort. The value of PLA was determined by the phenylalanine and leucine concentrations. We then build up the PLA score for 30-day mortality prediction and compare it with traditional risk scores and factors. A multicenter validation cohort with 437 patients was recruited to confirm the consistency.

**Results:** In the initiation cohort, we found the higher mortality rate was associated with phenylalanine **≥**88.5 mM (indicating metabolic disturbance) and leucine <68.9 mM (indicating malnutrition). PLA score was developed based on different levels of phenylalanine and leucine. In multivariable analysis, PLA scores predicted 30-day mortality independent of traditional risk scores and factors (hazard ratio=1.46, 95% confidence interval=1.33-1.60, p <0.001). Patients were classified into low, intermediate, high, and very-high risk categories according to the PLA score, with observed mortality rates of 9.0%, 23.8%, 45.6%, and 81.8%, respectively. Kaplan-Meier curves demonstrated that PLA scores differentiated the mortality rates of each risk group (log-rank=167.8, p <0.001). The prognostic value of PLA score was validated in the multicenter cohort. Based on the area under receiver operating characteristic curves, PLA scores predicted mortality better than traditional scores.

**Conclusions:** By combining phenylalanine and leucine concentrations, PLA score predicts 30-day mortality in CCU patients with heart failure better than APACHE II score and traditional risk factors. It also provides information of the nutritional phase and has a potential to improve the outcome of CCU patients by PLA-guided nutritional intervention.

**OUTCOMES WITH T-WAVE DISCORDANCE OF LEFT BUNDLE BRANCH BLOCK AND PRESERVED OR MILDLY REDUCED EJECTION FRACTION**

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Aims: Left bundle branch block (LBBB) is associated with an increased risk of adverse outcomes for patients with heart failure. The prognosis of LBBB in patients with a preserved ejection fraction (EF) remains controversial. This study investigated the predictive value of T-wave discordance for the prognosis of patients with LBBB and preserved or mildly reduced EF.

Methods: We enrolled 707 patients with complete LBBB and left ventricular EF ≥40% observed using electrocardiogram (ECG) and echocardiogram between January 2010 and December 2018. Their serial ECGs were reviewed during the follow-up period. The T-wave pattern was classified as discordant LBBB (dLBBB) or concordant LBBB (cLBBB) according to the 12-lead ECG T-wave morphology. The primary outcome was the composite of cardiovascular death or hospitalization for heart failure during a median follow-up period of 3.1 years. Multivariable Cox regression analysis was used to evaluate the independent predictors of the primary outcome.

Results: Patients with dLBBB had more comorbidities, higher heart rate, longer QRS and QTc duration, larger left ventricle (LV) end-systolic volume and left atrial dimension, lower LVEF, and higher mitral E/A ratio and E/e’, compared to those with cLBBB. Older age (p = 0.023), history of heart failure (p = 0.001), chronic kidney disease (p = 0.008), larger LV end-systolic volume (p = 0.002), lower LVEF( p =0.001), and presence of dLBBB (HR = 1.63, 95% CI = 1.011-2.628, p = 0.032) were independent predictors of the primary outcome in patients with LBBB and LVEF ≥40%. The discordant or concordant T wave morphology of LBBB could transform from one subtype to the other in up to 23% of the study population during the follow-up period, and individuals with persistent or transformed dLBBB faced an increased risk of cardiovascular death or nonfatal heart failure hospitalization.

Conclusions: In patients with LBBB and EF ≥40%, dLBBB serves as an independent predictor of a higher risk of cardiovascular death or nonfatal heart failure hospitalization.

**ASSOCIATION OF VISIT-TO-VISIT GLYCEMIC VARIABILITY WITH RISK OF ADVERSE CLINICAL OUTCOMES IN DIABETIC PATIENTS WITH HEART FAILURE WITH PRESERVED EJECTION FRACTION**

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**Background**

Previous studies have shown associations between glycemic variability (GV) and cardiovascular outcomes in patients with type 2 diabetes. However, the impact of GV on outcomes in patients with heart failure with preserved ejection fraction (HFpEF) has not been well investigated.

**Method**

Between 2014 and 2019, we conducted a retrospective cohort analysis using the electronic medical records of a tertiary medical center in Taiwan. Diabetic patients with HFpEF were enrolled. Each individual's coefficient of variability of fasting glucose (FGCV) was determined and the FGCVs were categorized into tertiles. Multivariable Cox regression models and the Kaplan-Meier with log-rank test were used to assess the association between the FGCV and the risk of hospitalization for heart failure (HHF), atrial fibrillation (AF), cardiac mortality, and overall mortality.

**Results**

In a cohort comprising 74,835 individuals diagnosed with diabetes, a subset of 753 patients was identified with HFpEF and measurement of FGCV. The median follow-up duration 38.1 months. In the model of full adjustment, the third FGCV tertile was significantly associated with an increased risk of HHF compared to the first tertile (hazard ratio [HR] = 1.32, 95% confidence interval [CI] 1.03-1.50, p = 0.026). Likewise, the highest FGCV tertille was associated with an increased risk of death (HR= 1.64, 95% CI: 1.15 to 2.26; p = 0.006) while it was not associated with increased of AF and cardiac mortality. Kaplan-Meier analyses revealed a significant association between FGCV and both HHF and overall mortality (log-rank p = 0.022 and <0.001, respectively).

**Conclusion**

Our study highlights a significant association between increased GV and a higher incidence of HHF as well as an elevated overall mortality rate in individuals with diabetes and HFpEF.

**THE IMPACT OF INTRAVASCULAR LITHOTRIPSY ON CLINICAL OUTCOMES IN COMPLEX CALCIFIED CORONARY LESIONS: A RETROSPECTIVE SINGLE-CENTER STUDY**

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Introduction:

Despite the increasing adoption of intravascular lithotripsy (IVL) for severely calcified coronary lesions, patient-level data on the safety and effectiveness of IVL in Taiwan are lacking.

Methods:

A retrospective study from Cheng Hsin General Hospital included patients undergoing coronary IVL between November 5, 2022, and January 5, 2024. Analysis focused on postprocedural major adverse cardiac events (MACE) within one month, assessing baseline demographics, procedural success, complications, and clinical outcomes, including all-cause mortality, myocardial infarction (MI), target lesion revascularization (TLR), and MACE (composite of cardiac death, target vessel myocardial infarction [TVMI], and TLR).

Results:

Among 86 patients (mean age 72.7 ± 9.5 years), 105 moderately to severely calcified coronary lesions underwent IVL. Diabetes mellitus (61.6%, n=53 patients) and end-stage renal disease (18.6%, n=16 patients) were prevalent. Acute coronary syndrome occurred in 29.1% (n=25 patients), stable angina in 70.9% (n=61 patients). De novo lesions constituted 85% (n=89), and in-stent restenosis 15% (n=16). Intravascular imaging was used in 97% (n=84 lesions), with upfront IVL in 25.7% (n=27 lesions) and bailout procedures for the rest. Rotational atherectomy ("RotaTripsy") was performed to facilitate the crossing of IVL balloon in 26.7% (n=28 lesions). Procedural success reached 91.4% (n=96 lesions). Complications included type A or B coronary dissection in 15.2% (n=16 lesions), without perforation or emergency coronary artery bypass graft surgery. TVMI occurred in 8.1% (n=7 patients ), with no TLR or mortality one month post-IVL. MACE rate was 8.1% (n=7 patients).

Conclusion:

 This study provides crucial insights into IVL's real-world use, showing high procedural success and low MACE rates. IVL emerges as highly effective and safe for managing severely calcified coronary lesions, demonstrating a short learning curve and favorable outcomes. Challenges, particularly in nodular calcification and stent under-expansion, necessitate further investigation. Extensive prospective studies are crucial to refine IVL strategies across diverse lesion characteristics.

**CAN TRANSCATHETER VALVE IN VALVE IMPLANTATION TREAT CONCOMITANT PARAVALVULAR LEAK OF SURGICAL BIOPROSTHESES?**

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**Introduction**

There were sporadic case reports of using transcathetervalve-in-valve (ViV) implantation to treat concomitant paravalvularleak (PVL) in failed surgical bioprostheses. However, the feasibility of this approach has never been well studied.

This study sought to evaluate the feasibility and clinical outcomes of transcatheterViVimplantation for concomitant bioprostheticvalve failure (BVF) and PVL.

**Methods**

We retrospectively reviewed the 165 patients underwent transcatheter mitral (n=104) or aortic (n=61) ViV implantation at a single center from 2014 Feburary to 2023 June.

The severity of PVL was quantified by the vena contracta (VC) width and the clinical outcomes were evaluated by 3D-TEE and according to the Valve Academic Research Consortium-2 criteria.

**Results**

26 of the 165 (15.7%) patients undergoing ViV implantation were identified with both BVF and mitral (n=16) or aortic (n=10) PVL. The procedural success rate of ViV was 100%. However, significant residual PVL≧moderate degree, defined as the VC width of PVL≧3mm, were evident in 5 of the 26 (19.2%) patients.

Among the 13 patients with VC width of PVL<3mm, the PVL became trivial or disappeared after ViV implantation. For the 7 patients with VC width of PVL between 3-5mm, the severity of PVL remained unchanged after ViV. Transcatheter occluder was needed in 4 patients with larger VC width of PVL≧5mm. One patient presented persistent hemolysis and/or heart failure during follow-up.

**Conclusions**

Our data suggest that ViV implantation to treat concomitant BVF and mild PVL is feasible with good echocardiographic and clinical outcomes. For patients with VC width of PVL≧3mm, the severity of PVL remains unchanged after ViV. Moreover, transcatheter occluder repair may be needed in those patients with larger VC width of PVL≧5mm.

**CAN TRANSCATHETER VALVE IN VALVE IMPLANTATION TREAT CONCOMITANT PARAVALVULAR LEAK OF SURGICAL BIOPROSTHESES?**

Chun-Ting Liu1, Yung-Tsai Lee1,2, Tien-Ping Tsao1,3, Kuo-Chen Lee1,3, Ming-Chon Hsiung1, Wei-Hsian Yin1,4  
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**ASSOCIATION OF FRAILTY AND CRITICAL LIMB ISCHEMIA IN PATIENTS ON MAINTENANCE HEMODIALYSIS: A PROSPECTIVE COHORT STUDY**

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**Background and hypothesis.** Critical limb ischemia (CLI), the most severe manifestation of peripheral arterial disease, is a prevalent yet unpredictable complication among hemodialysis patients. Frailty is a multidimensional syndrome linked to adverse outcomes in this patient population. In this study, we aimed to examine the influence of clinical factors on vascular events in patients undergoing hemodialysis.

**Methods.** The Hsinchu VA study was a multicenter prospective cohort study that included patients undergoing maintenance hemodialysis since January 2008. The initial cohort for this study consisted of 1,136 patients, of whom 828 successfully underwent a frailty test. Frailty was defined as the following: unintentional weight loss, weakness, exhaustion, low physical activity, and slow gait speed. Patients with one or two of these characteristics were categorized as pre-frail. Major adverse limb events including CLI were recorded at 3-month intervals until December 31, 2022.

**Results.** The mean patient age was 67 years, and 48% were female. Overall, 34% of participants were frail, 38% pre-frail, and 28% not frail. The frailty phenotype was associated with age, female sex, low educational level, diabetes mellitus, and history of stroke. During a median follow-up of 1461 days (interquartile range, 1016–1826 days), 104 patients experienced incident CLI events (not frail, 6.5%; pre-frail, 11%; frail, 20%; P < 0.001). Frail patients had a more than threefold higher risk of CLI than those who were non-frail (hazard ratio [HR] 3.94; 95% confidence interval [CI] 2.22–6.99; P < 0.001). After multivariable adjustment for age and comorbidities, frailty remained significantly associated with CLI (HR 2.98; 95% CI 1.63–5.45; P < 0.001).

**Conclusions.** Frailty is associated with an increased risk of CLI in patients undergoing hemodialysis. These findings highlight the risk of CLI in frail patients undergoing hemodialysis.

**FHL2 AS A TRANSCRIPTIONAL COACTIVATOR OF RUNX2 IN CHRONIC KIDNEY DISEASE-INDUCED ARTERIAL CALCIFICATION**

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Arterial medial calcification (AMC) is a common occurrence among individuals suffering from end-stage renal disease (ESKD) and is an established predictor of cardiovascular morbidity and mortality. AMC is a dynamic process driven by vascular smooth muscle cells (VSMC) that undergo trans-differentiation into an osteogenic phenotype within the vascular wall. This process is regulated by a key transcriptional factor known as runt-related transcription factor 2 (RUNX2). While the pathological role of RUNX2 in AMC has been widely recognized, it has never been used as a therapeutic target due to its indispensable role in mesenchymal cell differentiation and bone development. FHL2 belongs to the LIM-domain only proteins and functions as a molecular transmitter, it links different signaling pathways to transcriptional regulation. In this study, we employed transcriptomic data from human subjects and an in-vivo animal reporter system to demonstrate that FHL2 expression is notably elevated within the cardiovascular system. Specifically, we observed that FHL2 expression is upregulated and translocated to the nucleus of VSMC cells in the aortas of both human subjects with chronic kidney disease (CKD) and mice models of CKD. We further identified that FHL2 interacts with RUNX2 to promote its transcriptional activity within VSMCs. Notably, we observed that depletion of FHL2 expression prevents VSMCs from undergoing trans-differentiation to an osteogenic phenotype, and mitigates aortic calcification in uremic animals. These findings have significant implications for our understanding of the molecular mechanisms that underlie cardiovascular disease progression in the context of CKD, and suggest FHL2 as a potential therapeutic target for preventing or treating aortic calcification in patients with CKD.

**THE CORRELATION BETWEEN SIRTUIN 1 AND SUPPRESSION OF SODIUM-GLUCOSE COTRANSPORTER-2 INHIBITORS ON DIABETES-INDUCED ATRIAL FIBRILLATION**

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Objective:

With the aging population, atrial fibrillation (AF) prevalence is increasing, particularly among diabetic patients. Sodium-glucose cotransporter-2 inhibitors (SGLT2i), a class of oral hypoglycemic drugs, have shown promise in heart failure management and potentially in reducing the onset of AF, according to recent studies. Yet, the mechanisms behind AF reduction by SGLT2i remain unexplored. Notably, SIRT1, a sirtuin pathway enzyme linked to metabolic issues and energy use, could be influenced by SGLT2i. This study aims to investigate the interplay between atrial tachyarrhythmia and cardiomyopathy in diabetic cardiomyopathy, focusing on SIRT1.

Methods and Results:

We established a high-glucose (HG) conditioned cell model (30mM glucose) and administered Dapagliflozin (20 μM), with or without the SIRT1 inhibitor Sirtinol (25 μM). Additionally, a Streptozotocin (STZ)-induced diabetes mellitus (DM) rat model was used to assess the effects of atrial tachyarrhythmia under various conditions: sham control, STZ, STZ plus Dapagliflozin, and STZ with both Dapagliflozin and Sirtinol. The cell model showed increased apoptosis and reactive oxygen species (ROS) levels and decreased SIRT1 expression under HG conditions. Dapagliflozin inhibited these changes, while Sirtinol reversed Dapagliflozin's effects. Calcium channel activity was also modulated in this model. In the rat models, Dapagliflozin upregulated SIRT1 expression. The STZ and STZ plus Dapagliflozin and Sirtinol groups exhibited notable declines in left ventricular function and compliance. Dapagliflozin-treated STZ rats displayed shorter AF durations and reduced inducibility compared to STZ rats, effects partially negated in the STZ rats treated with both Dapagliflozin and Sirtinol.

Conclusion:  
SIRT1 may play a protective role by reducing apoptosis, ROS, and calcium channel activity in cell models. In the STZ rat model, Dapagliflozin decreased AF incidence and duration, potentially through upregulation of SIRT1 levels.

**REVERSAL OF HYPOXIA-INDUCED PULMONARY VASCULAR REMODELING BY IPSC-DERIVED EXOSOMES**

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Chronic hypoxic exposure leads to structural and biochemical changes across the pulmonary vasculature, from the hilum to the peripheral vessels in the alveolar wall. Such alterations contribute to pulmonary arterial hypertension (PAH), a disease characterized by occlusive vascular remodeling. While current treatments for PAH improve quality of life, they fail to address the underlying structural abnormalities in the pulmonary vasculature. Stem cell-derived exosomes have emerged as a promising therapeutic avenue, yet their mechanisms of action remain partially understood. This study explores the potential of induced pluripotent stem cell-derived exosomes (iPSC-Exo) to ameliorate vascular remodeling in the pulmonary arterioles of a hypoxia-induced rat PAH model, focusing on the medial smooth muscle layer of arterioles. Murine iPSC-Exo were administered intraperitoneally to male Sprague-Dawley rats (aged 4-6 weeks) daily following hypoxia exposure. Additionally, hypoxia-induced rat primary pulmonary artery smooth muscle cells (PASMCs) served as an in vitro model to elucidate the mechanisms through which iPSC-Exo modulate vascular remodeling. Treatment with iPSC-Exo mitigated histopathological alterations in pulmonary artery muscularization and reduced right ventricular systolic pressure in rats with hypoxia-induced PAH. iPSC-Exo also counteracted the hypoxia-induced proliferative, pro-migratory, senescent, and apoptosis-resistant phenotypes in PASMCs. Our findings demonstrate the efficacy of iPSC-Exo in reversing pulmonary hypertension by attenuating pulmonary vascular remodeling, suggesting a potential iPSC-free therapy for PAH treatment.

**FGF21 ALLEVIATES LPS-TRIGGERED INFLAMMATION IN HUMAN MACROPHAGE AND PROMOTES M2 MACROPHAGE POLARIZATION**

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Objectives:

1.Investigate the effect of FGF21 on inflammation pathways of human monocyte-derived macrophages.

2.Examine how FGF21 influenced the polarization of human monocyte-derived macrophages.

Methods: Human THP-1 cells were differentiated into macrophages by culturing in RPMI supplemented with 50 nM PMA (phorbol 12-myristate 13-acetate), and flow cytometry detection of CD68 was conducted to confirm differentiation.1 µg/mL LPS was used to trigger inflammation in macrophages, and different concentrations of FGF21 (0, 25, 50, 100, 200, 400 ng/mL) were also added. CD36, CD9, NF-κB, and JNK were analyzed by western blot. Macrophage polarization was analyzed by flow cytometry detection of M1 marker CD86 and M2 marker CD206. Also, caspase-1 inflammasome assay was done to determine inflammasome activation in macrophages.

Results: Western blotting results indicated a decrease in lipid and scar associated macrophage markers CD36 and CD9 after FGF21 treatment. Also, NF-κB and JNK activation were decreased. M2 macrophage markers including CD163 and TGM2 were increased after FGF21 treatment on LPS-stimulated macrophages, showing an M2-polarizing trend. Also, FGF21 reduced caspase 1 activity in LPS-treated macrophages.

Conclusion: Our findings suggest that FGF21 has a general anti-inflammatory effect, while also driving M2 polarization of macrophages under LPS stimulus. The anti-inflammatory effect of FGF21 might be mediated by inhibiting NF-κB and JNK signaling pathway activation. In conclusion, FGF21 might alleviate inflammatory cardiovascular disease involving M1/2 macrophage imbalance.

**IDENTIFYING THE ROLE OF GDF2 VARIANTS IN PULMONARY ARTERIAL HYPERTENSION THROUGH WHOLE EXOME SEQUENCING AND PATIENT-SPECIFIC IPSC-DERIVED ENDOTHELIAL CELL ANALYSIS**

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Since 2000, significant advances have been achieved in understanding the genetics and genomics of Pulmonary Arterial Hypertension (PAH), yet much remains to be uncovered. This study explores novel genetic variants related to PAH using Whole Exome Sequencing (WES) in cohorts of Asian idiopathic and heritable PAH at Kaohsiung Veterans General Hospital. Among 69 patients, the most frequent variants were identified in the BMPR2, ATP13A3, and GDF2 genes. Notably, GDF2 has not been previously associated with PAH. To ascertain the critical role of GDF2 in PAH, we generated induced Pluripotent Stem Cells (iPSCs) from a patient carrying the GDF2 V403I variant (iGDF2) and differentiated these iPSCs into endothelial cells (iGDF2-ECs). Functional analyses of endothelial cells from both control iPSCs and iGDF2 individuals were conducted, focusing on the uptake of acetylated-LDL and lectin binding through direct fluorescent staining. Results indicated that iPSC-ECs from all subjects exhibited similar endothelial cobblestone morphology, acetylated LDL uptake, and UEA-1 lectin binding. Intriguingly, iGDF2-ECs displayed enhanced proliferative and migratory phenotypes compared to control iPSC-ECs, alongside promoted angiogenesis. Collectively, our findings suggest that GDF2 variants may play a significant role in PAH, potentially offering an alternative therapeutic strategy for specific PAH patients in the future.

**PREDICTORS OF SURVIVAL TO DISCHARGE IN PATIENTS WITH OUT-OF-HOSPITAL SUDDEN CARDIAC ARREST AFTER RETURN OF SPONTANEOUS CIRCULATION**

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Background: The early predictors of survival to discharge and neurological outcome in patients with out-of-hospital sudden cardiac arrest after return of spontaneous circulation remain unclear.

Methods: Between June, 2012 and September, 2021 at Far Eastern Memorial Hospital, 4365 consecutive non-traumatic out-of-hospital cardiac arrest patients were sent to emergent department. The patients with return of spontaneous circulation were enrolled. Coronary angiography was performed in all the patients. Baseline characteristics, electrocardiography characteristics, coronary lesion, treatment methods, hematology and biochemistry data were analyzed.

Results: A total of 282 consecutive return-of-spontaneous-circulation patients were enrolled. The mean age was58.11 ± 13.10 (mean ± SD) with male 235 patients. 208 patients (73.8%) were diagnosed of acute myocardial infarction, confirmed by coronary angiography. The overall survival rate was 64.9%. Univariate analyses showed shockable rhythm, sinus rhythm and faster heart rate post resuscitation, patent coronary arteries, ST-segment elevation myocardial infarction, higher sodium level, total cholesterol/ low-density lipoprotein/ high-density lipoprotein, and left ventricular ejection fraction were associated with survival. However, older age, acute myocardial infarction, history of diabetes, prolonged cardiopulmonary resuscitation duration, transient wide QRS, junctional rhythm, atrial fibrillation, complete right bundle branch block post resuscitation, higher blood sugar, HbA1C, potassium level, and GPT were associated with mortality. The combination of transient complete right bundle branch block with non-sinus rhythm (atrial fibrillation or junctional rhythm) post resuscitation was highly associated with mortality. Multivariate analyses showed older age (year) (OR: -0.076; 95% CI: -0.120〜-0.033; p=0.001), acute myocardial infarction (OR: -1.786; 95% CI: -3.352〜-0.219; p=0.025), higher lactate (mg/dl) (OR: -0.145; 95% CI: -0.226〜-0.063; p=0.001), and transient CRBBB with non-sinus rhythm post resuscitation (OR: -6.424; 95% CI: -8.858〜-3.991; p<0.001) were independent negative predictors of survival to discharge.

Conclusions: In patients with non-traumatic sudden cardiac arrest after return of spontaneous circulation, older age, acute myocardial infarction, higher lactate, and transient complete right bundle branch block with non-sinus rhythm (atrial fibrillation or junctional rhythm) were negative predictors of survival on discharge. Transient complete right bundle branch block with non-sinus rhythm was the most powerful and unique early predictor of mortality.

**USING ECHOCARDIOGRAPHY BASED DEEP LEARNING TO EARLY DETECT THE INFARCT RELATED ARTERY IN PATIENTS WITH ACUTE CORONARY SYNDROME**

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**BACKGROUND:** In patients with acute coronary syndrome (ACS), echocardiography detected regional wall motion abnormalities (RWMA) facilitates the recognition of ischemic heart disease and infarct related artery. Nevertheless, the differentiation of RWMA relies on the experiences of performers. Notably, in ACS patients without transmural infarction, RWMA may not be visible upon naked eyes.

PURPOSE: This study aims to investigate whether the application of 3D Convolution Neural Network could assist clinicians to differentiate patients with and without ACS based on echocardiography detected RWMA.

**METHODS:** From 2018 to 2021, we collected echocardiographic imaging in 796 patients without ACS (Normal Control; NC), 759 with ACS and detectable RWMA (RWMA) and 267 with ACS but not detectable RWMA (uncertain; UC). The diagnosis of ACS was defined by the obstructive coronary arterial disease (CAD) in coronary angiography. Apical four, two and long chamber viewer were acquired and RWMAs were defined by cardiologists. Cardiac-Echo Net consists the techniques of 3D Convolution Neural Network and 3D MaxPooling.

**RESULTS:** After exclusion echocardiographic imaging not qualified for analysis, we collected 40813 and 5928 images for establishing the model of Cardiac-Echo Net. In the final model, areas under the receiver operating characteristic curve are 98.9 and 89.2% for the training and validation, respectively. In the external validation dataset, the sensitivity was 81.8% and specificity was 81.6%. Notably, compared with cardiologists, Cardiac-Echo Net showed a superior accuracy in differentiating NC from RWMA (0.89 v.s. 0.815). Likewise, in differentiating NC from UC, Cardiac-Echo Net has a persistently higher accuracy than cardiologists (0.87 v.s. 0.65).

**CONCLUSIONS:** Superior to previous deep learning models, this novel one combined several neural-networking from different fields. Cardiac-Echo Net could spontaneously detect the subtle myocardial ischemia in ACS patients without eye-catching RWMA while further external validation is necessary.

**INTRADIALYTIC FOOT TEMPERATURES PREDICT THE PRESENCE OF SEVERE LOWER EXTREMITY ARTERIAL DISEASE**

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**Aims**

Patients with end stage renal disease (ESRD) are at high risks of lower extremity arterial disease (LEAD), with worsened prognosis. Diagnosing LEAD is challenging in these patients due to atypical symptoms, limited accessibility to specialist care, and severely calcified vessels that often lead to falsely elevated ankle brachial indices (ABI). We aimed to establish a highly accessible means of detecting LEAD in patients with ESRD through recording intradialytic foot temperatures.

**Methods**

This is an observational, prospective cohort study enrolling patients with ESRD who are under maintenance hemodialysis. Patients underwent a doppler ultrasound (DUS) of the lower limbs and an ABI measurement at baseline, and foot temperature recordings during hemodialysis. The presence of severe LEAD was confirmed by DUS, defined by monophasic or absent flow in the distal posterior tibial artery (PTA) and distal anterior tibial artery (ATA). For intradialytic foot temperature measurement, skin contact thermometers were placed on the instep and the sole of the feet, and one on the wrist contralateral to the hemodialysis access. ABI and skin temperatures were compared between patients with and without severe LEAD, and the power of skin temperature parameters in detecting severe LEAD were evaluated by receiver operating characteristic (ROC) curve analysis.

**Results**

A total of 85 patients were recruited, including a total of 170 limbs. The ABI of limbs with severe PTA disease was 0.93±0.26, and 0.93±0.27 in limbs with severe ATA disease. The area under curve (AUC) of the ROC of the difference between wrist and instep temperatures at the end of the hemodialysis session was 60.9% in predicting severe PTA disease, 63.5% in predicting severe ATA disease, and 64.9% in predicting an ABI of <0.9. The AUC-ROC of the difference between wrist and sole temperatures at the end of the hemodialysis session was 62.3% in predicting severe PTA disease, 64.6% in predicting severe ATA disease, and 61.7% in predicting an ABI of <0.9.

**Conclusions**

In patients with ESRD under regular hemodialysis, measuring intradialytic foot temperatures may be a highly accessible and reproducible means of detecting severe LEAD that can be integrated into telehealth systems and distributed to resource-poor settings.

**RELATIONSHIPS AMONG INTENSIVE BLOOD PRESSURE CONTROL, MORNING BLOOD PRESSURE SURGE AND 24-HOUR BLOOD PRESSURE VARIABILITY – POST HOC ANALYSIS OF THE SPRINT DATA**

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**Objectives:**This study aimed to investigate the relationships between cardiovascular outcomes and blood pressure (BP) reduction, 24-hour BP variability (BPV), morning BP surge (MBPS), and visit-to-visit BPV.

**Methods:**This study involved 897 participants from the Systolic Blood Pressure International Trial (SPRINT), for whom biochemical and demographic data, medication history, and various blood profile information were collected. Univariate and multivariate linear regression models were used to assess the impact of intensive BP management on 24-hour BPV and the MBPS.

**Results:**Among the 897 participants (average age 71.48 ± 9.42 years; 640 males), 453 received intensive treatment. Those in the intensive group were more likely to be on ACE inhibitors, CCBs, or diuretics. This group showed significant reductions in 24-hour systolic and diastolic BP, as well as in both short-term and long-term BPVs, such as average real variability (ARV) and MBPS. The analysis demonstrated strong associations between intensive BP management and improvements in various BPV measures, including systolic ARV, diastolic BP ARV, and cumulative BP loads. A significant correlation was also found between intensive BP lowering and the primary composite outcome of the SPRINT study.

**Conclusions:**This study highlighted the significant associations between intensive BP management and reductions in the MBPS and 24-hour BPV, which affect both short-term and long-term health outcomes. The primary benefits of intensive BP control appear to stem from direct BP reduction rather than changes in BPV. However, further extensive randomized controlled trials are needed to confirm these findings.

**EXPLORING PERICORONARY ADIPOSE TISSUE DIFFERENCES BETWEEN CALCIFIED AND NON-CALCIFIED PLAQUES ON CORONARY CT ANGIOGRAPHY WITH FAT ATTENUATION INDEX AND RADIOMICS ANALYSIS**Te-Wei Shieh, Yu-Tung Tsai, Jia-Yu Yan, Yu-Ting Sun, Cheng-Han Chang, Wen-Jeng Lee, Tzung-Dau Wang  
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**Introduction**

**Coronary CT angiography (CCTA)-derived pericoronary adipose tissue (PCAT) is a novel marker for coronary inflammation. The relationship between the extravascular PCAT and the vascular lesion itself is particularly important to establish its use in monitoring the disease status. Past studies investigated lesion-level PCAT differences in vessels with acute myocardial infarction. However, lesion-level PCAT differences between calcified and non-calcified plaques in patients with stable coronary artery disease is not well understood. In this study, we investigated the differences between PCAT adjacent to calcified plaques and PCAT adjacent to non-calcified plaques using fat attenuation index (FAI) and radiomics analysis.**

**Material and Methods**

**In a cohort of 100 consecutive patients who underwent CCTA, 446 plaques were identified in 89 patients. Among these plaques, 213 were calcified plaques and 49 were non-calcified plaques, with the rest classified as mixed by our expert cardiologist and radiologist. The PCAT of these 262 plaques were then extracted. Following prior literature, adipose tissue was defined as voxels with attenuation ranging -190 to -30 HU. PCAT was extracted within 3 mm distance from the vessel wall, along the middle 10 mm of each plaque. FAI was defined as the mean attenuation of the PCAT volume. For radiomics analysis, we used PyRadiomics and the random forest classifier from Scikit-Learn to distinguish PCAT volumes of calcified and non-calcified plaques. Patient-level 5-fold cross validation was used to develop the classifier.**

**Result**

**FAI were higher in PCAT adjacent to non-calcified plaques compared to PCAT adjacent to calcified plaques (-81.4 [IQR: -91.8 to -73.5] vs -86.7 [IQR: -94.1 to -78.4], p=0.008). The classifier using radiomics features  achieved better discrimination (AUROC=0.738 [0.715-0.761]) than FAI alone (AUROC=0.607 [0.605-0.609]).**

**Conclusion**

**Significant differences in PCAT characteristics were found between calcified and non-calcified plaques either with direct comparison of FAI or radiomics analysis. Radiomics analysis revealed further discrimination between PCAT characteristics other than FAI may exist between calcified and non-calcified plaques. It is also important to note that FAI of calcified plaques could be affected by beam hardening. Further investigation is needed to explore the underlying cause of such differences and their clinical implications.**

**AGING MATTERS: ADDRESSING THE CHALLENGES IN SCREENING OF PRIMARY ALDOSTERONISM IN ELDERS**Hao-Yun, Loa,b, Zheng-Wei Chena,c, Vin-Cent Wua, Yen-Hung Lina, on behalf of the TAIPAI Study Group  
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**Background**:

    The aldosterone-to-renin ratio (ARR) is considered the most reliable and accessible screening test for detecting primary aldosteronism (PA). The decline in accuracy of ARR with increasing age presents a critical challenge in identifying PA in elderly individuals. This study aimed to clarify the effect of aging on PA screening and explore ways to enhance the accuracy of PA screening in the elderly population.

**Methods and Results**:

    We prospectively enrolled 1586 PA patients and 1493 essential hypertension (EH) patients, which were further divided into elderly (≥ 65 years, 292 PA and 484 EH) and non-elderly (< 65 years, 1294 PA and 1209 EH) groups respectively. Elderly patients had higher plasma aldosterone concentration (PAC), ARR and lower plasma renin activity (PRA) compared with non-elderly patients in both PA and EH groups. Restricted cubic spline method revealed a trend towards higher log- PAC/ARR and lower log-transformed PRA with increasing age. However, the results of the confirmation tests (captopril challenge test and salt loading test) showed no significant difference in PAC, PRA, and ARR between the elderly and non-elderly groups. Using standard ARR cut-off value (35), compared to non-elderly group, elderly group have similar sensitivity (elder: 84.93% versus non-elder 85.47%) but decreased specificity (elder: 54.93% versus non-elder 66.58%).

    Receiver operating characteristic curve analysis was performed in elderly and non-elderly groups to determine the optimal ARR cut-off value for PA screening. The results indicated the best cut-off value was 35.81 (sen:85.01%, spe:67.08%) in non-elderly and 50.3 (sen:79.11%, spe:65.49%) in elderly groups based on the maximum Youden's index. If ARR > 35 was adopted to screen PA in elderly patients, an additional criterion with PAC > 20 ng/dL as indicated by Youden's index, should be added to achieve similar accuracy (sen:78.08%, spe:65.14%).

**Conclusion**:

    The ARR levels were higher in elderly patients (both PA and EH), leading to decreased accuracy of ARR if using the standard cut-off value for PA screening. Both raising the ARR cut-off value to 50 or adding a second step of PAC > 20 ng/dL improved the screening accuracy of ARR in elderly population.

**PROGNOSTIC SIGNIFICANCE OF BLOOD PRESSURE FLUCTUATIONS IN THE MORNING AND EVENING FOR CARDIOVASCULAR OUTCOMES**Chi-Chen Cheng, Wei-Lun Chang, Shao-Yuan Chuang, Yu-Hsuan Lee, Chi-Jung Huang, Hao-Min Cheng, Chern-en Chiang, Chen-Huan Chen, Kei Asayama, Takayashi Ohkubo, Teemu J Niiranen, José Boggia, Jan A. Staessen  
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**Objective**

Independent of office blood pressure (BP) levels, home BP is associated with the occurrence of cardiovascular events. Abnormal diurnal variations in BP have been linked to cardiovascular disease (CVD). However, data on diurnal patterns of home BP, the morning and evening BP difference (MEdiff), are scarce. This study looked into the relationship between all MEdiff indices and cardiovascular outcomes.

**Design and method**

The association was assessed using data from the International Database of Home Blood Pressure in Relation to Cardiovascular Outcome (IDHOCO), which comprised 6753 patients from five cohorts. We found 1254 patients who had taken home blood pressure readings every morning and every evening for more than 14 days. The MEdiff over a 13-day period, omitting the first day, for each patient were used to calculate the average (MEdiff\_sbp\_avg), minimum (MEdiff sbp\_min), and maximum value (MEdiff\_sbp\_max). MEdiff is defined as morning (6AM-8AM) minus evening BP (8PM-10PM) measures. The prognostic values of these parameters were assessed using Cox proportional hazard models. Factors adjusted in the base model included age, sex, body mass index, total cholesterol, high-density lipoprotein cholesterol, smoking status, prevalence of diabetes, history of cardiovascular disease and antihypertensive drug treatment.

**Results**

Only MEdiff\_min\_sbp strongly predicted CVD(183 events; mean follow-up time of 13.4 years), but MEdiff\_avg\_sbp [0.906(0.087-1.018 per SD)], MEdiff\_max\_sbp 0.949(0.846-1.064 per SD) did not. Spline regression analysis with thresholds of

-17mmHg and -4mmHg revealed a U-curve association between MEdiff\_min\_sbp and the risk of CVD outcomes. Three group were identified by MEdiff\_min\_sbp : lower than  -17 mmHg (Low, n=574), between -17 and -4 mmHg (medium, n=568) and higher than -4 mmHg (high, n=112). The high group (1.353 (95% CI 1.052-1.739)) had significantly higher risk of CVD, compared to the low group, in the models with adjustment for the mean of the morning and evening SBP values, and MEdiff\_avg\_sbp or various BP variability indices.

**Conclusions**

MEdiff\_min\_sbp was strongly associated with an increased risk of cardiovascular outcomes, suggesting that it could be a useful BP index for describing abnormal BP diurnal variations for CVD risk prediction.

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**IMPACT OF ALBUMINURIA ON ARTERIAL STIFFNESS AND ITS REVERSAL AFTER TARGETED TREATMENT IN PATIENTS WITH PRIMARY ALDOSTERONISM**Ting-Wei Kao1, Che-Wei Liao2, Cheng-Hsuan Tsai3, Yi-Yao Chang4, Chien-Ting Pan5, Chin-Chen Chang6 , Bo-Ching Lee6 , Wei-Chieh Huang7 , Kuo-How Huang8 , Ching-Chu Lu9 , Tai-Shuan Lai10 , Chieh-Kai Chan11, Jeff S Chueh8 , Vin-Cent Wu10 , Chi-Sheng Hung3 , Yen-Hung Lin3 , Zheng-Wei Chen5  
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Primary aldosteronism (PA) has been associated with atherosclerosis beyond the extent of essential hypertension, but the impact of albuminuria remains unknown. The aim of the study was to investigate the effect of concomitant albuminuria on arterial stiffness in PA. A prospective cohort study was conducted to evaluate the association of albuminuria (>30 mg/g in morning spot urine) with arterial stiffness, as measured noninvasively by pulse wave velocity (PWV) in patients with PA. Propensity score matching (PSM) with age, sex, systolic and diastolic blood pressure, creatinine, potassium, number of antihypertensive medications, and hypertension history was used to balance baseline characteristics. The effects of albuminuria and urine albumin-creatinine ratio (UACR) on PWV before and 1 year after treatment were analyzed. A total of 840 patients with PA were enrolled, of whom 243 had concomitant albuminuria. After PSM, there were no significant differences in baseline demographic parameters except alpha-blocker and spironolactone use. PWV was greater in the presence of albuminuria (P=0.002), and positively correlated with UACR. Multivariable regression analysis identified albuminuria, age, body weight, systolic blood pressure, hypertension duration, and calcium channel blocker use as independent predictors of PWV. As for treatment response, only PA patients with albuminuria showed significant improvements in PWV after PSM (P=0.002). The magnitude of improvement in PWV increased with UACR, and reached plateau when UACR exceeded 100 mg/g according to restricted cubic spline analysis. In conclusion, concomitant albuminuria in PA was associated with greater arterial stiffness and more substantial improvement after targeted treatment.

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**FATTY ACID-BINDING PROTEIN-3 AND RENAL FUNCTION DECLINE IN PATIENTS WITH CHRONIC CORONARY SYNDROME**  
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**Background:** Renal function decline is frequently encountered in patients with coronary artery disease. Due to the shared vascular pathogenesis between the two conditions, novel biomarkers such as the fatty acid-binding protein-3 (FABP-3) have been proposed for diagnosis and prognosis prediction. This multicentre prospective cohort study investigates the association between FABP-3 and renal dysfunction.

**Methods:** A cohort with the chronic coronary syndrome were studied. Patients were classified into three groups based on the initial serum FABP-3 levels. Three commonly-employed formulas [the Modification of Diet in Renal Disease (MDRD), CKD Epidemiology Collaboration (CKD-EPI), and Cockcroft-Gault (CG) equations] were used to estimate the patient’s renal function. Renal events were defined as >25% and >50% decline in estimated glomerular filtration rate (eGFR). Multivariable analysis with Cox regression was employed to delineate the correlation between FABP-3 and renal dysfunction.

**Results:** A total of 1606 subjects were included. During a mean follow-up of 35.9 ± 23.2 months, there were 274, 239, and 286 patients with eGFR >25% reduction and 60, 60, and 58 patients with eGFR >50% reduction according to MDRD, CKD-EPI, and CG equations, respectively. In the Kaplan-Meier survival curve and log-rank test, increased levels of FABP-3 were significantly correlated with eGFR >25% reduction (P < 0.001 for 3 equations) and >50% reduction (P < 0.001 for 3 equations). Multivariate analysis showed that the group with the highest level of serum FABP-3 has a significantly increased risk with a hazard ratio (HR) of 2.643, 3.054, and 2.843 for >25% reduction, and 9.769, 4.838, and 8.404 for >50% reduction in eGFR according to MDRD, CKD-EPI, and CG equations, respectively.

**CORONARY MICROVASCULAR DYSFUNCTION, CARDIAC MALADAPTATION AND POOR CLINICAL OUTCOMES IN NON-ISCHEMIC HEART FAILURE WITH PRESERVED EJECTION FRACTION: A PROSPECTIVE DYNAMIC SPECT STUDY**

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**Background:** Although studies have demonstrated clinical utility and accuracy of quantitative dynamic single-photon emission computed tomography (SPECT), there were still insufficient data regarding its application in patients of non-ischemic heart failure with preserved ejection fraction (HFpEF).

**Methods:** We prospectively enrolled 103 individuals undergoing dynamic SPECT imaging with (99m) Tc-sestamibi dipyridamole stress (MyoFlowQ, Taiwan), followed by standard gated myocardial perfusion imaging (Siemens, USA) from April 2019 to December 2020 in a single medical center and all were proven without obstructed coronary arteries via invasive angiography or coronary computed tomography angiography before. Absolute quantitation of myocardial blood flow (MBF) and myocardial flow reserve (MFR) were derived. Of them, 55 individuals were HFpEF, and 48 were control. The prognostic value was evaluated by Kaplan-Meier analysis.

**Results:** Among them, the mean age was 65.5 years and 52.4% were men. Patients of HFpEF were older (68.5±10.9 years), had higher N terminal pro B type natriuretic peptide level (386.0±490.9 pg/ml), higher prevalence of atrial fibrillation (20.4%) whereas lower estimated glomerular filtration rate (68.0±23.2 ml/min/1.732), serum albumin concentration (4.1±0.4 mg/dl), compared with the control group (p<0.05). Compared with control group, patients of HFpEF were associated with worse global left ventricular (LV) / left atrial (LA) deformational indices, lower post-stress MBF (2.37±0.78 vs 33.0±0.94 ml/min/g, p<0.001), higher global rest MBF (1.11±0.55 vs 0.89±0.19 ml/min/g, p=0.008), and resulting significantly lower global MFR (2.34±0.84 vs 36.8±1.04, p<0.001). Lower MFR (<2.5) was independently associated with worse global LV longitudinal strain and LA strain, Coef: -0.59 (95% confidence interval [CI]: -0.86 ~ -0.32) and 2.20 (95% CI: 1.14 ~ 3.27) in the multivariate models. The results were similar in regional MFRs by vascular based analysis. The median follow-up was 2.2 (IQR: 1.9, 3.0) years. Patients with HFpEF and global MFR <2.5 had an increased risk of all-cause mortality and hospitalization, as the reference as controls with global MFR ≥ 2.5 (26.3 % vs. 2.4 %, Log rank p=0.005).

**Conclusion:** Coronary microvascular dysfunction, assessed by dynamic SPECT is frequently observed in patients with non-ischemic HFpEF and correlates with abnormal atrioventricular mechanics. Comorbidities with HFpEF and coronary microvascular dysfunction worsen individual’s prognosis.

**SUPRANORMAL LEFT VENTRICULAR EJECTION FRACTION, CONCENTRIC REMODELING, AND LONG-TERM SURVIVAL: A HOSPITAL-BASED COHORT STUDY**

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**Background**

Supranormal left ventricular ejection fraction (LVEF) by echocardiography has been conferred a paradoxically higher risk of mortality in the general population. However, its underlying mechanism remains unclear. In this study, we aim to investigate the prognostic implication of supranormal LVEF and its interaction with concentric remodeling among clinically referred Asian population.

**Methods**

Consecutive participants who underwent echocardiography with LVEF≥ 60% in a tertiary medical center (between April 2005 and December 2021) were included. Concentric remodeling was defined as LV relative wall thickness (RWT) of more than 0.42. The association between LVEF categories and long-term all-cause mortality was assessed using adjusted Cox model, and subgroup analysis was performed by the stratification of concentric remodeling. Study endpoint was all-cause mortality by linkage to the National Death Registry.

**Results**

This study included a total of 67,108 participants (age 60.5 ± 17.2, men 44.6%), and there were 7,029 deaths (10.5%) observed during a mean follow-up of 60.4 ± 47.9 months. In the adjusted Cox model, subjects within the highest quartile (Q4) of LVEF had a significantly higher risk (vs. Q1) for all-cause mortality (adjusted hazard ratio, 1.13; 95% confidence interval, 1.06-1.21; P< 0.001), after adjusting for potential confounders. A significant interaction was found between LVEF and concentric remodeling (P for interaction< 0.001) such that the higher mortality risk of supranormal LVEF could be only observed among those with concentric remodeling but not among those without concentric remodeling.

**Conclusions**

Supranormal LVEF is also significantly associated with a higher risk for all-cause mortality in the Asian population. The presence of concentric remodeling may elucidate the underlying mechanism of the poor prognosis of the subjects with supranormal LVEF.

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**EPIGENETIC REGULATION OF MYOCARDIAL SERCA2A AND CALCIUM HOMEOSTASIS BY LONG NONCODING RNA LNC-SYNPO**

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**Background**: Abnormal sarcolemmal Ca2+ handling and impaired contractile function owing to sarcoendoplasmic reticulum calcium ATPase (SERCA2a) downregulation are pathognomonic hallmarks of heart failure (HF). However, no clinically proven HF therapy that targets SERCA2a dysregulation directly. Therefore, we aim to identify a long noncoding RNA (lncRNA) as a novel HF therapeutic target to rescue abnormal cardiac function by regulating SERCA2a expression.

**Results**: Myocardial *lnc-SYNPO*, a lncRNA derived from the 3' untranslated region of *SYNPO2* gene, was significantly upregulated during HF and showed a strong negative correlation with that of SERCA2a in both mouse and human left ventricle. Knockdown of *lnc-SYNPO* in human induced pluripotent stem cell-derived cardiomyocytes (hiPSC-CMs) and P1 neonatal mouse cardiomyocytes (P1CM) increased, whereas overexpression of *lnc-SYNPO* reduced SERCA2a expression, Ca2+ homeostasis and myocyte contractile function. Mechanistically, ATAC-Seq revealed that knockdown of *lnc-SYNPO* induced open chromatin at the SERCA2a promoter region. RNA pull-down assay showed that *lnc-SYNPO* directly bound to SERCA2a promoter and EZH2, the core components of the polycomb repressor complex 2 (PRC2). In addition, CUT&RUN-qPCR assay demonstrated that *lnc-SYNPO* recruited EZH2 and induced H3K27me3 occupancy at the SERCA2a promoter, leading to epigenetic silencing of SERCA2a. Furthermore, *lnc-SYNPO* expression was induced by endothelin-1 (ET-1) treatment, a neurohumoral factor upregulated in the failing heart, and could be blocked by calcineurin inhibitor. Additionally, luciferase reporter assay showed *lnc-SYNPO* transcriptional activity was activated by calcineurin overexpression. EMSA revealed that NFAT1 transcriptionally regulated *lnc-SYNPO* via physical interaction with *lnc-SYNPO* promoter. These data showed that *lnc-SYNPO* expression was regulated by calcineurin/NFAT1-dependent transcriptional control. In animal studies, cardiomyocyte-specific *lnc-Synpo* knockout mice (αMHC-Cre/ERT; *lnc-Synp*o fl/fl) preserved LV contractile function and prevented LV dilation following myocardial ischemia reperfusion injury (IRI) induced by transient left anterior descending coronary artery (LAD) ligation.

**Conclusion**: Taken together, our results unveiled a previously undiscovered epigenetic regulatory mechanism of SERCA2a by *lnc-SYNPO*, contributing to impaired myocardial Ca2+ homeostasis and contractile dysfunction observed with HF. Targeting *lnc-SYNPO*, therefore, could be a novel therapeutic approach to improve Ca2+ homeostasis and contractile function in the human failing heart.

**ASYMPTOMATIC LEFT VENTRICULAR DYSFUNCTION IN PATIENTS WITH OR WITHOUT TYPE 2 DIABETIC MELLITUS: A POPULATION-BASED COHORT STUDY**

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Background

Type 2 diabetes mellitus (T2DM) has been related to ventricular systolic and diastolic dysfunction and subsequent heart failure.The study aimed to investigate the prevalence of asymptomatic left ventricular dysfunction (aLVD) in subjects with T2DM, and its predisposing factors. 

Methods

Subjects without existed cardiovascular disease were eligible for this study. Left ventricular ejection fraction (LVEF), mitral E/Aratio, peak mitral annulus tissue velocity (e’), left atrial dimension (LAD), peak tricuspid regurgitation velocity (TR Vmax) were obtained by a standard echocardiographic study. Asymptomatic left ventricular diastolic dysfunction(ALVSD) was defined as LVEF< 50% and the asymptomatic left ventricular diastolic dysfunction (ALVDD) was defined as having three or more of the four parameters: septal e' < 7, septal E/e’>15, LAD > 40mm, and TR Vmax> 2.8 m/s. All-cause mortality up to 2 years was obtained by linking to the National Death Registry.

Results

A total of 27684 subjects (59.6±17.4 years, 46.2% men, 17.7%diabetes) were included in this study(Table 1). The ALVSD patients were older, male dominant, more prevalent with hypertension, diabetes Mellitus and coronary artery disease, and had higher BMI, lower hemoglobin, and estimated glomerular filtration rate. ALVSD and ALVDD were more prevalent in patients with T2DM than those without (4.9% vs 8.2%, P<0.001)(15.4% v.s.32.1%) (Figure 1). Age (odds ratio and 95% confidence interval: 1.04, 1.03–1.04), male gender(2.24, 1.99-2.52), T2DM (1.28, 1.11-1.46), coronary artery disease (1.38,1.14-1.68), hemoglobin (0.95, 0.91-1.00), CKD stage 3(2.02, 1.63-2.50), and CKD stage 4-5(3.95,3.06-5.09) were all independently correlated toALVSD(Table 2a). On the other hand age (odds ratio and 95% confidence interval: 1.07, 1.07–1.08), BMI (1.12, 1.11-1.13), T2DM (1.34, 1.24-1.46), hypertension (1.31, 1.20-1.43), hemoglobin (0.88, 0.85-0.91), and CKD stage 4-5(3.11, 2.58-3.76) were independently correlated to ALVDD(Table 2b). During a mean follow-up of 5.3 ± 3.6 years, there were 2625 deaths (9.5%). The Kaplan–Meier curve analysis revealed diabetic subjects were associated with the worst survival rate than those with either T2DM or ALVSD/ALVDD  (Figure 2).   
  
Conclusions  
ALVD has a high disease burden, and its prevalence is higher in patients with T2DM, which was associated with an excessive mortality rate.

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**N-CADHERIN SERVES AS A NOVEL THERAPEUTIC TARGET ON CARDIAC REGENERATION FOLLOWING CARDIAC INJURY THROUGH β-CATENIN DEPENDENT MECHANISM**

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**Rationale:**Cardiovascular diseases are frequently associated with loss of cardiomyocytes (CMs) which may lead to heart failure (HF) and high mortality. One of the major hurdles to reverse cardiac dysfunction is the lack of regenerative capacity in adult human heart following injury. Mammalian heart, including mouse and human, do have the potential to regenerate; this, however, occurs only within a narrow window at young age. The molecular determinants undunderlying the disparate regenerative capacity remain incompletely understood.  
**Methods and results:**Exploring a comparative bulk transcriptome analysis in regenerative neonatal vs. non-regenerative adult mouse CMs, we identified gene encoding N-Cadherin, Cadherin-2 (Cdh2) could contribute to the mitotic activity of CMs. The expression of Cdh2/N-Cadherin exhibited a spatial-temporal correlation with age and its response to cardiac injury, revealing an important role of Cdh2/N-Cadherin in cardiac regeneration. Knocking down Cdh2in neonatal mouse primary CMs culture as well as human induced pluripotent stem cell-derived CMs (hiPSC-CMs) reduced, whereas overexpression of Cdh2 increased, the proliferative activity and cell cycle gene expression. Meanwhile, N-Cadherin directly interacted with b-Catenin to promote CMs proliferation, and mediated the b-Catenin expression in a post-translational dependent manner. Restricted proliferating capability could be facilitated while the β-Catenin was ectopic expressed in Cdh2 knockdown mouse and human CMs revealing that N-Cadherin/β-catenin, indeed, contributes to the regulation of CMs proliferation. Intriguingly, targeting N-Cadherin by utilizing a mouse line with inducible, cardiac-specific deletion of N-Cadherin led to incomplete cardiac repair/regeneration and increased fibrotic scar deposition at the injured site, supporting the functional role of N-Cadherin in regulating CMs regeneration following injury. Conversely, enhancing N-Cadherin expression in adult mouse heart exhibited protective effects against left anterior descending artery occlusion-induced cardiac hypertrophy, reduced fibrosis and markedly improved left ventricle function. Furthermore, boosting de novo CMs, divided from existing CMs were the center of the cardiac regeneration.  
**Conclusion:**Cdh2/N-cadherin played critically in promoting CM regeneration by post-translational stabilization of pro-mitotic transcription factor, β-catenin. Enhancing cardiac expression of N-cadherin, therefore, could be a potential novel therapeutic approach to promote cardiac regeneration and restore cardiac function in adult heart following injury.  
**Keywords:**cardiac regeneration, myocardial infarction, heart failure, N-Cadherin, Bulk RNASeq

**THE ASSOCIATION BETWEEN LEFT VENTRICULAR EJECTION FRACTION AND LONG-TERM SURVIVAL: A LARGE HOSPITAL-BASED COHORT STUDY IN TAIWAN**

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**Background**

Left ventricular ejection fraction (LVEF) is the most commonly used metric for quantifying left ventricular function. A U-shaped relationship between LVEF and long-term survival with a nadir between 60-70% has been reported in the western, regardless of age, sex, or prevalent heart failure. However, the distribution of mortality risk across the spectrum of LVEF among the Asian population remains unclear.

**Methods**

In this large hospital-based cohort study, consecutive participants who underwent echocardiography (2002-2021) in a tertiary medical center in Taiwan were included. LVEF was measured by echocardiography using biplane Simpson’s method. All LVEFs were categorized into intervals of 5% from <20% to ≥70%. The National Death Registry was linked to identify mortality and causes of death. Cox proportional hazards regression was used for analyses with adjustment for age, sex, body mass index, and comorbidities.

**Results**

A total of 139,526 participants (age 61.4 ± 17.5, men 50.5%) were included. During a mean follow-up of 58.5 ± 47.0 months, 17,293 subjects (12.4%) died. Overall, adjusted hazard ratios (HR) for all-cause mortality showed a U-shaped relationship between LVEF and long-term survival with a nadir of risk at LVEF of 60-70%. Compared with the reference group (LVEF: 60-65%), subjects with LVEF> 70% had an HR of 1.21 (95% confidence interval [CI]: 1.12-1.31), and those with LVEF of 50-55% had an HR of 1.28 (95% CI: 1.22-1.35). Similar results with a nadir at 60-70% could also be observed when stratified by the age of 65, sex, comorbidities of hypertension, or diabetes.

**Conclusions**

A U-shaped relationship between LVEF and long-term survival with a nadir at LVEF of 60-70% can also be observed in the Asian population. This result may support the use of LVEF> 70% to define supranormal LVEF in Asian subjects.

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**PROPER SIZING MAY IMPROVE PROCEDURAL OUTCOMES WITH BOTH BALLOON AND SELF-EXPANDABLE VALVE FOR THE TREATMENT OF BICUSPID AORTIC VALVE STENOSIS**Huan-Chiu Lin, Yung-Tsai Lee, Tien-Ping Tsao, Wei-Hsian Yin, Kuo-Chen Lee, Ming-Chon Hsiung, Jeng Wei  
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**Background:**

Previous data comparing the performance of new-generation self-expandable (SE) versus balloon-expandable (BE) transcatheter heart valves (THVs) in bicuspid aortic stenosis (BAS) demonstrated that a higher rate of moderate-severe paravalvular leakage (PVL) was observed in the SEV, whilst patients treated with BEV had a higher rate of annular rupture. We aimed to study whether proper sizing may improve procedural outcomes with both SEV and BEV for the treatment of BAS.

**Methods:**

The complementary approach of supra-annular sizing to conventional annular sizing method (Wei's Method) developed by our team is useful in providing guidance to perform safer THV implantation in BAS patients undergoing transcatheter aortic valve replacement (TAVR). We applied the sizing method in 66 consecutive patients who underwent TAVR using new-generation Evolut R/PRO or Sapien 3 valves in BAS.

**Results:**

A total of 66 patients (n=41 [62%] treated with Sapien 3 and n=25 [38%] treated with Evolut R/PRO) were included. Mean age was 70 ± 13 years and mean Society of Thoracic Surgeons Predicted Risk of Mortality was 4.4±4.0%. Valve Academic Research Consortium-2 device success was similar between Sapien 3 and Evolut R/PRO (95% versus 88%; P=NS). No patients experienced annular rupture in both SEV and BEV group. No differences in the rate of permanent pacemaker implant were observed. The echocardiographic follow-up after TAVR demonstrated significant higher rate of moderate-to-severe PVL among patients treated with SEV (SEV 12% versus BEV 2%, P<0.05). Although estimated aortic valve area were similar in both groups, however, mean trans-aortic valve pressure gradient was significantly higher among patients treated with BEV (SEV 6.7±3.5mmHg versus 11.0 ± 5.0mmHg, P<0.05).

**Conclusions:**

Our study confirms that proper sizing may improve procedural outcomes with both BEV and SEV for the treatment of BAS, but higher rate of moderate-to-severe PVL among patients treated with SEV is still a concern.

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**LONG TERM EFFECT OF TREATMENT IN PATIENTS WITH CAROTID ANGIOPLASTY AND STENTING IN-STENT RESTENOSIS**Sheng-Fu Liu1,2,3, Ying-Hsien Chen2,4, Mao-Shin Lin2,4, Ching-Chang Huang2,4, Chi-Sheng Hung2,4, Chih-Fan Yeh2,4, Cheng-Hsuan Tsai2,3,4, Hsien-Li Kao2,4  
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**Objectives:**

Carotid angioplasty and stenting (CAS) is one of the mainstream treatments for extracranial carotid artery stenoses. Carotid angioplasty and stenting in-stent restenosis (CAS ISR) is associated with an increased rate of recurrent cerebrovascular events. The treatment strategy for patients with restenosis after carotid angioplasty and stent placement is rather ambiguous, and whether patients will receive clinical benefit after treatment is also questionable. The purpose of this study is to investigate the long-term prognosis of patients with carotid angioplasty with stenting restenosis after endovascular intervention.

**Methods:**

We conducted a retrospective cohort study of consecutive CAS from 1997 to 2021 and enrolled subjects with CAS ISR ≥50% who were divided into two groups according to whether the patients received endovascular intervention or not. We analyzed baseline demographic data, and angiographic and procedural characteristics. The primary endpoint was the composite of all-cause mortality, ipsilateral stroke, and myocardial infarction. Cox proportional hazards regression models were used to estimate the incidence of the primary endpoint in different groups.

**Results:**

A total of 106 patients with CAS ISR ≥50% were enrolled, where 63 (59.4%) subjects received endovascular intervention and 43 (40.6%) subjects were not. Median follow-up for the cohort was 1,044 days (330-1,847). There were significant differences between the arms with sex (p = 0.045), degree of ISR (p < 0.05), ipsilateral CCAS (p = 0.038), total occlusion (p = 0.024) and multiple stents (p = 0.010). In Cox regression analysis, there were no statistically difference among the endovascular intervention and nonintervention groups for the composite of all-cause mortality, ipsilateral stroke, and myocardial infarction. By Kaplan-Meier analysis, there were no statistically difference between the both groups with respect to death / stroke / MI as a composite at last follow-up (p = 0.5).

**Conclusions:**

For the patients with CAS ISR ≥50%, there were no significant difference between endovascular intervention and nonintervention groups in long term outcomes. Endovascular intervention failed to improve long term death / stroke / MI relative to nonintervention in current study.

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**PRIMARY RESULTS FROM APOLLO-B, A PHASE 3 STUDY OF PATISIRAN IN PATIENTS WITH TRANSTHYRETIN-MEDIATED AMYLOIDOSIS WITH CARDIOMYOPATHY**WEN-CHUNG YU1; MATHEW S. MAURER2; MARIANNA FONTANA3; JOHN L. BERK4; FINN GUSTAFSSON5; MARCOS SIMÕES6; MARTHA GROGAN7; FÁBIO FERNANDES8; ROBERT L. GOTTLIEB9; MILOS KUBANEK10; STEEN POULSEN11; THIBAUD DAMY12; IGOR DIEMBERGER13,14; NOBUHIRO TAHARA15; W.H. WILSON TANG16; LAURA OBICI17; ALEJANDRA GONZÁLEZ-DUARTE18; YOSHIKI SEKIJIMA19; MATTHEW T. WHITE20; ELENA YURENEVA20; PATRICK Y. JAY20; JOHN VEST20; JULIAN D. GILLMORE21  
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Background:

Patisiran, an RNAi therapeutic that inhibits TTR synthesis, is approved for the treatment of hATTR amyloidosis with polyneuropathy. Exploratory analyses of a cardiac subpopulation in the APOLLO study demonstrated the potential for patisiran to improve cardiomyopathy manifestations in patients with hATTR amyloidosis with polyneuropathy.

Objective:

To investigate safety and efficacy of patisiran in patients with ATTR amyloidosis with cardiomyopathy in the APOLLO-B study.  
Methods:

Patients were 18–85 years old with evidence of cardiac amyloidosis by echocardiography, and  ATTR amyloid deposition on tissue biopsy or fulfilling nonbiopsy diagnostic criteria for ATTR amyloidosis with cardiomyopathy. Medical history of HF due to ATTR amyloidosis with ≥1 prior HF hospitalization, or current clinical evidence of HF, was required. Patients were randomized 1:1 to patisiran IV 0.3 mg/kg or placebo Q3W for 12 months. Primary endpoint: change from baseline in 6-MWT at Month 12 (M12) with patisiran vs placebo. Secondary endpoints at M12: effect of patisiran vs placebo on health status and QoL (KCCQ-OS); 3 composite endpoints included death, hospitalizations, and urgent HF visits.

Results:

APOLLO-B enrolled 360 patients (patisiran, n=181; placebo, n=179): median (range) age at screening, 76.0 (41, 85) years; male, 89%; wtATTR, 80%; receiving tafamidis at baseline, 25%. At M12, patisiran demonstrated significant clinical benefits vs placebo in functional capacity (6-MWT, meters) (median [95% CI] change from baseline: -8.15 [-16.42, 1.50] [patisiran]; -21.35 [-34.05, -7.52] [placebo]; Hodges-Lehmann estimate of median difference [95% CI]: 14.69 [0.69, 28.69]; p=0.0162)and KCCQ-OS (LS mean [SEM] change from baseline: 0.30 [1.26] [patisiran]; -3.41 [1.28] [placebo]; difference [SEM], 3.71 [1.80]; p=0.0397). For the composite endpoint of all-cause mortality, frequency of CV events, and change from baseline in 6-MWT, the win ratio was 1.27 (95% CI: 0.99, 1.61; p=0.0574),  favoring patisiran. All-cause deaths were observed in 4 (2.2%) patisiran vs 10 (5.6%) placebo (HR [95% CI]: 0.36 [0.11, 1.14]). Exploratory biomarker analysis favored patisiran. Patisiran demonstrated an acceptable safety profile, including no cardiac safety concerns or drug-related deaths.

Conclusion:

In the 12-month study, patisiran demonstrated statistically significant and clinically meaningful benefits on functional capacity (6-MWT) and health status and quality of life (KCCQ-OS) compared to placebo.

**ACUTE IMPROVEMENT IN ARTERIAL-VENTRICULAR COUPLING AND LV MECHANICAL EFFICIENCY AFTER TRANSCATHETER AORTIC VALVE IMPLANTATION WITH SEVERE AORTIC STENOSIS**Shen-Che Lin, Jer-Shen Chen, Jih-Hsin Huang, Kuan-Ming Chiu, Chih-Yao Chiang  
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**Purpose:**We evaluated the acute effects of TAVI on VA coupling and LV mechanical efficiency by doppler echocardiography. **Methods:** Forty-seven patients (male/female 21/26, age 83±6.7) underwent isolated TAVI using CoreValve, doppler echocardiography before and after TAVI in same hospitalization. We compared the changes in aortic valve and LV geometry and hemodynamics included aortic valve, LV geometry, valvular load, arterial load and global LV load. Load-independent index of contractility Ees and index of afterload Ea. We determined ventricular-arterial coupling, stroke work and LV mechanic efficiency. **Results:** Reduction of valvular load MPG (45.6 ± 19.7; 8.2±4.8 mmHg P<0.0001). The stroke work loss also decreased (25.7±9; 6.1 ± 3.7% P<0.0001). With parallel with increased indexed effective orofice area (0.46±0.13; 1.28±0.43 cm2m-2 P<0.0001), Energy loss Index (0.53 ± 0.17 cm2 m-2; 2.00 ±1.04 P<0.0001) and Reduction of vascular load, systemic arterial compliance (0.82±0.32; 1.07±0.48 ml m-2mmHg-1P<0.0001), Systemic vascular resistance (1.4±0.4; 1.2±0.4 dyne s cm-5, P=0.0003). The global LV load ( Valvulo-arterial impedance, Zva) (3.98±1.28 ; 2.56±0.63 mmHg ml-1 m2P<0.0001). A significant reduction of end-systolic meridional stress (85.2±40.7; 49.3±17.5Kdyne cm-2 P<0.0001). LV Ejection Fraction (61±14 %; 65±10 %; P=0.0123). Fractional shortening (33.5±9.5; 36.3±7.0 % P=0.264). Ea (3.0±0.9; 1.7±0.5 mmHgml-1 P<0.0001), Ees (5.7±4.7, 4.6±2.4 mmHg ml-1 P=0.1143). Normalization of VA coupling (0.71±0.45; 0.45±0.23, P=0.0003). With regard to left ventricular mechanical efficiency, a significant reduction of stroke work (8676±2953; 6112±2300 mmHg ml, P<0.0001) and potential energy (4026±2491; 2382±1431 mmHg ml P<0.0001), increase work efficiency (69±13; 73±8% P=0.04). **Conclusions:** TAVI procedure determined acute improvement of the global LV hemodynamic load, allowing a better mechanical efficiency. Further follow-up investigations are needed to evaluate the long-term results in prolonged observation.

**SHOCKWAVE INTRAVASCULAR LITHOTRIPSY FOR SEVERELY CALCIFIED CORONARY ARTERY DISEASE: FIRST CLINICAL EXPERIENCE IN TAIWAN**Tien-Ping Tsao1, Wei-Hsian Yin2, Jeng Wei3, Yu-Cheng Kao4  
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**Background:**

     Severely calcified coronary lesions usually caused stents under-expansion, which was recognized as the major cause of stent restenosis, thrombosis, and target lesion failure. In the past, interventionists might apply rotational atherectomy, cutting balloon or NC balloon in extremely high pressure. Nevertheless, these procedures sometimes bring higher risks, such as perforation, bradycardia, hypotension, no-reflow, distal embolization and severe dissection. Shockwave intravascular lithotripsy (SIVL), a novo weapon for calcified lesions modification, was relatively safer to perform for most calcified lesions. We present our SIVL clinical experience that is the first real world experience in Taiwan.

**Methods:**

     There were 6 patients enrolled since November to December in 2022. Total 12 severely calcified lesions were identified on the coronary angiography of each patient. Except the newly implanted stent, we did not exclude other lesion characteristics, such as stent restenosis, vessel size, lesion length, tortuosity, bifurcation, or total occlusion. Technical success is defined as successful delivery the SIVL balloon and stents, residual stenosis < 20%, TIMI 3 blood flow and no edge dissection. Procedural success is defined as achieving technical success without periprocedural or in-hospital complication.

**Results:**

     There were total 12 lesions in 6 cases intention to do SIVL procedures. The technical success rate was 91.7% while the procedural success rate was also 91.7% because of no periprocedural complication. The ability to deliver a used and deflated SIVL device should be one of the concerns in planning. For the eccentric calcification and calcium nodule, focally using the SIVL emission ≧ 40 pulses was one of the choices to improve the lesion preparation and stent expansion without carrying the high risk of vessel dissection or rupture at normal intima side. We performed rotational atherectomy then SIVL for some lesions preparation, so called the Rotatripsy. There was much less risk for vessel injury while emitting ultrasonic energy and only 4 to 8 atm dilation pressure than continually enlarging burr size with numerous ablation cycles in large or tortuous vessels.

**Conclusions:**

     The SIVL procedures, inclusive of Rotatripsy, could be a safer, simpler, and effective PCI strategy for severely calcified coronary artery disease.

**THE IMPACT OF TOTAL STENT LENGTH ON ALL-CAUSE MORTALITY AMONG DIABETES MELLITUS AND NON-DIABETES MELLITUS PATIENTS, A RETROSPECTIVE SINGLE-CENTER STUDY**  
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**Background**:

    Percutaneous coronary intervention (PCI) is the current standard treatment of coronary artery disease (CAD). In the previous studies, the longer stent length was shown to be correlated to adverse outcomes in patients with CAD. Besides, Diabetes Mellitus(DM) is a risk factor for cardiovascular disease and has been associated with 2- to 4- fold higher morality. This study aims at investigating the impact of stent length on mortality among DM and non-DM patients who underwent PCI.

**Methods**:

   The patients who underwent PCI between 2003 to 2013 were eligible for the study. Total stent length was defined as the total length of stenting in all three coronary arteries in the first PCI procedure of the patient. The patients were divided into two groups by stent length (< 48 mm, and ≥ 48 mm, according to stent distribution. All-cause mortality up to 10 years after discharge was obtained by linking to the National Death Registry.

**Results**:

   Among a total of 7404 participants (68.73 ± 12.52 years, 81.9% men). There were 4686 (63.2%), and 2718 (36.8%) subjects in non-DM and DM subjects, respectively. The BMI was in randomized distribution. In the Kaplan-Meier analysis, a significantly higher mortality rate was demonstrated among the DM subjects between the group of stent length ≥ 48 mm compared to those <48 mm total stent length (Log-Rank p = 0.003). On the other side, there was no significant difference in mortality among the non-DM subjects between the group of stent length ≥ 48 mm and < 48 mm. (Log-Rank p = 0.303). Multivariate cox models adjusting age, sex, renal function, cholesterol level, triglyceride level, the neutrophil-lymphocyte ratio (NLR), congestive heart failure, hypertension revealed that among the DM subjects comparing with total stent length <48 mm, total stent length over 48mm was significantly associated with all-cause mortality (P<0.001, HR= 1.153-1.659), whereas no significant among the non-DM group. (P=0.659, HR= 0.897-1.187)

**Conclusions**:

   In the present study, we demonstrated that total stent implantation over 48 mm at one time was an independent predictor of long-term mortality among DM patients instead of non-DM patients.

**APPLYING AN ARTIFICIAL INTELLIGENCE–ENABLED ELECTROCARDIOGRAPHIC SYSTEM FOR REDUCING MORTALITY: A PRAGMATIC RANDOMIZED CLINICAL TRIAL**

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**Background**

Intensive care helps to reduce mortality in patients with critical illness. However, early identification the vulnerable patients poses a substantial challenge in clinical practice. We aimed to evaluate the effectiveness and outcome of an artificial intelligence (AI) enabled electrocardiogram (ECG) for identifying patients with high risk of mortality.

**Methods**

In this multi-center single-blind patient-level randomized controlled trial (NCT05118035), we recruited 39 attending physicians and their patients from emergency department and inpatient department. The AI-ECG intervention included: (1) an AI report in patients’ electronic health record; and (2) an active warning message to physicians for patients with high-risk of mortality. Patients were allocated by HIS system. The primary outcome was all-cause mortalityin 90 days. The secondary analyses included followed-up medical behavior changes and causes of death.

**Results**

15,965 patients (N = 8,001 intervention; N = 7,964 control) with a mean age of 61±18 years were used for data analysis. The cumulative proportion of death was significantly reduced [hazard ratio (HR): 0.83, 95% confidence interval (CI): 0.70-0.99] in the intervention group (3.6%) compared to the control group (4.3%), majorly in the high-risk cases identified by AI-ECG (N = 709 intervention; N = 688 control) with reducing 31% of mortality (16.0% in the intervention arm versus 23.0% in the control arm, HR: 0.69, 95% CI: 0.53-0.90; p for interaction of intervention and AI-ECG risk = 0.026). More intensive care unit admissions, arrhythmia interventions, echocardiographic examinations, and electrolyte examinations were performed in the intervention group with high-risk ECGs, which may contribute to the significant risk reduction on cardiac death (0.2% in the intervention arm versus 2.4% in the control arm, HR: 0.07, 95% CI: 0.01-0.56).

**Conclusion**

An AI-ECG recognizing perilous patients to instigate intensive care endows the reduction of all-cause mortality risk.

**COMPUTED TOMOGRAPHY PULMONARY ANGIOGRAPHY DETECTS CLINICAL DETERIORATION IN PATIENTS WITH ACUTE PULMONARY EMBOLISM**

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**Objective**

Acute pulmonary embolism (PE) is a dynamic disease which the severity may evolve over time. The simplified pulmonary embolism severity index (sPESI) is a generally accepted index to evaluate the disease severity, but lack the ability to predict the progression of the disease. An imaging model to facilitate the prediction of possible clinical deterioration of pulmonary embolism is lacking.

**Methods**

This retrospective study included 210 patients (age, 65 ± 16 years; percentage of men 40%) diagnosed with acute PE from 2008 to 2019. The pulmonary artery (PA) was divided into 27 segments based on CT pulmonary angiography (CTPA), and the level of obstruction was determined. The primary outcome is clinical deterioration, which is defined as death from PE, resuscitation, the need of mechanical ventilation, shock, and the need of thrombolysis. Models were designed based on the location (central versus peripheral) and the degree (partial versus total) of obstruction, as well as variables deducted by multivariate logistic regression analysis; and the integrated discrimination index (IDI) and net reclassification index (NRI) were utilized to test the improvement of the predictive ability of the models compared with sPESI alone. External validation was performed using 109 patients (age, 64 ± 17 years; percentage of men 43%) from another tertiary medical center.

**Results**

The level of obstruction on CTPA can predict clinical deterioration (AUC 0.62-0.68). The combination of image severity with sPESI increases the predictive ability of clinical deterioration compared with sPESI alone (NRI 0.09-0.12; IDI 0.05-0.09). The summation of the obstruction of all 27 PA segments provides the best predictive value, while calculating nearly totally obstructed 20 peripheral segments provides similar predictive ability (AUC 0.78 versus 0.77). All models combined with sPESI increased the predictive ability in the external validation.

**Conclusion**

The severity of obstruction on CTPA in combination with sPESI increased the ability to predict clinical deterioration compared to sPESI. Calculating nearly totally obstructed 20 peripheral segments together with sPESI can aid in predicting clinical deterioration.

**MAINTAIN ADEQUATE SERUM VITAMIN D3 LEVEL CAN REDUCE THE RISK OF ATHEROGENIC DYSLIPIDEMIA IN YOUNG ADULTS**

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**Abstract**

**Background:** Atherogenic dyslipidemia is a significant risk factor for CVDs. Limited studies have suggested a link between low levels of vitamin D3 and dyslipidemia. However, more research is needed to fully understand the association between vitamin D3 deficiency and novel biomarkers of atherogenic dyslipidemia.

**Methods:** We recruited 976 young adults between 2011 and 2019 to investigate the association between vitamin D3 levels and markers of atherogenic dyslipidemia. We measured serum levels of vitamin D3 and lipid profile markers, including low-density lipoprotein cholesterol (LDL-C), low-density lipoprotein triglycerides (LDL-TG), and small-dense low-density lipoprotein cholesterol (sdLDL-C) as novel biomarkers of atherogenic dyslipidemia.

**Results:** Our study results suggested that vitamin D levels were significantly associated with lower levels of LDL-C, LDL-TG, sdLDL-C, non-high-density lipoprotein cholesterol (non-HDL-C), triglycerides, and total cholesterol after controlling for potential confounding factors using multivariate linear regression. Additionally, using multivariate logistic regression, the group with vitamin D3 levels above 25 ng/mL had a significantly lower risk (risk ratio of LDL-TG ≥ 75th percentile: 0.51, 0.32-0.89; RR of sdLDL-C: 0.46, 0.22-0.95) of developing atherogenic dyslipidemia compared to the group with levels lower than 15 ng/mL. The sensitivity test did not show any significant differences among subgroups of different sexes, ages, BMIs, smoking statuses, or physical activity levels.

**Conclusions:**  Our study demonstrated that vitamin D3 deficiency was associated with atherogenic dyslipidemia among young Taiwanese adults. These results also suggested that keeping serum 25(OH)D levels above 25 ng/mL could significantly decrease the risk of developing atherogenic dyslipidemia, particularly novel biomarkers of sdLDL-C and LDL-TG.  
  
**Keywords:** atherogenic dyslipidemia, LDL-TG, sdLDL-C, vitamin D3, cardiovascular disease

**THE ASSOCIATION BETWEEN THE RESPONSE OF LIPID-LOWING AGENTS AND MONOGENIC VARIANTS OF FAMILIAL HYPERCHOLESTEROLEMIA**

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Purpose

The presence of familial hypercholesterolemia (FH) variants (LDLR, APOB, or PCSK9) may theoretically impact the effectiveness of lipid-lowering drugs in reducing low-density lipoprotein cholesterol (LDL-C) levels by affecting their pathways. The response to lipid-lowering drugs in individuals with these variants is currently unclear in clinical practice. Our study is to assess the relationship between the response of lipid-lowering drugs and FH variants.

Methods

This was a single-center retrospective cohort study that initially included 2353 patients with coronary artery disease (CAD) who underwent DNA microarray genetic analysis between August 28, 2018, and May 13, 2022. We identified FH-related pathogenic or likely pathogenic variants according to the ClinVar database. The dose of statin and ezetimibe for carriers of FH variants was converted to the equivalent dose of atorvastatin. Patients with atorvastatin use were included among the noncarriers. The multivariate linear regression model evaluated the response to lipid-lowering drugs in reducing LDL-C levels for both FH variant carriers and noncarriers as the main outcome.

Results

Our study comprised 22 carriers of FH variants, all associated with LDLR variants (81.8% male; average age 65.6 years) and 1053 non-carriers (82.2% male; average age 64.3 years). The FH variants carriers had less effectiveness in reducing LDL-C levels than noncarriers (*β*, standard error [SE], -43.70 [4.63]; adjusted P < 0.001). Additionally, there was no significant difference in achieving LDL-C treatment goals with a CAD below 70 mg/dL between carriers and non-carriers (77.3% vs. 83.2%; P = 0.401). Carriers had a higher prevalence of receiving coronary artery bypass graft surgery compared to non-carriers (18.18% vs. 3.89%, with an odds ratio of 5.47 [95% CI: 1.29-17.64]; P=0.012).

Conclusions

Carriers of FH variants have reduced lipid-lowering effects. Therefore, when an initial prescription for heterozygous FH, more intensity lipid-lowering agents should be considered.

**THE ROLE OF CARDIAC FIBROBLAST IN HEART REGENERATION**

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**RATIONALE:** Cardiovascular disease is a leading cause of death worldwide, and few treatments focus on promoting cardiac regeneration. Myocardial infarction, a common cardiovascular disease, results in the loss of cardiomyocytes, in contrast to neonatal mouse and zebrafish hearts, which can fully regenerate after injury without scarring. Adult mouse hearts lose this regenerative capacity and subsequently experience adverse remodelling and reduced cardiac function. Previous studies have shown that zebrafish fibroblasts express factors that support cardiomyocyte proliferation, but this mechanism has not been confirmed in mice. This study aims to test the hypothesis that cardiac fibroblasts are essential for cardiomyocyte regeneration in neonatal mice.

**RESULTS:** We first re-analysed the RNA-Seq data (GSE153480) and to focus scRNA-Seq on fibroblasts, we analysed the clusters of cells after injury. We found that clusters of fibroblasts characterised by the expression of Col1a2 were highly expressed at the peak of regeneration. To test whether cardiac fibroblasts influence heart regeneration, we ablated Col1a2-expressing cells using a Col1a2-Cre/ERT2;iDTR neonatal mouse. We showed that Col1a2-iDTR neonatal mice significantly increased the area of fibrosis by Masson's trichrome and also decreased cardiomyocyte proliferation. We tested whether fibroblast deletion affected heart size or apical resection survival in neonatal mice. These data show that 42% of DT-treated mice died within 3 weeks of AR. Significant differences were also found in the heart-weight-to-body weight ratio. We used bulk RNA sequencing to identify signalling pathways between baseline neonatal and adult cardiac fibroblasts. Differential expression analysis in adult and neonatal CF showed strong enrichment in genes involved in the cell cycle.

**CONCLUSIONS:** Our study provides evidence that fibroblasts, specifically those expressing Col1a2, play a crucial role in heart regeneration. Our findings indicate that the ablation of these cells leads to an increase in fibrosis and a decrease in cardiomyocyte proliferation, ultimately affecting heart size and survival in neonatal mice. The bulk RNA sequencing data also supports the importance of fibroblasts in the regenerative process by showing a strong enrichment of genes involved in the cell cycle. These results suggest that targeting fibroblasts may have therapeutic potential for the treatment of heart disease.

**DETECTING PULMONARY HYPERTENSION IN TAIWANESE POPULATION WITH MACHINE LEARNING MODELS**

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Background:

Pulmonary hypertension (PH) is commonly found in patients with heart disease and has major prognostic implication. Early detection and further precise diagnosis are essential. We therefore investigated the diagnostic accuracy of ECG for PH and prognostic value in Taiwanese population.

Methods:

We enrolled a total of 67493 patients received ECG and echocardiography at cardiology out-patient department visit were prospectively enrolled during 2005-2016. Clinical evidence of pulmonary hypertension was defined as right ventricular systolic pressure more than 40 mmHg found on echocardiography. Machine learning algorithm based on hold-out testing with training set 80%, test set 10% and validation set 10%, and 5-fold cross validation were applied. The causes and date of death for the participants were confirmed via review of hospital charts and death certificates, and linkage of our database with the National Death Registry through unique personal identification number given to every Taiwan citizen.

Results:

The accuracy, F1 score and AUC of the AI algorithms were XGBOOST model (82.9%/0.84), CATBOOST model (89.2%/0.89/0.95). Feature selection was complete to identify significant parameters, which showed QRS terminal magnitude, amplitude of R wave in lead V2 and V6; P area in V1 and aVL of great importance.

Conclusions:

The machine learning algorithms provided robust performance on detecting PH. Through feature selection, we may identify potentially important factors of PH on EKG.

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**THE INDISPENSABLE ROLE CD36+-TISSUE RESIDENT MACROPHAGES IN CARDIAC REGENERATION**  
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Heart failure (HF) is a major cause of death, owing to the inability to replenish lost cardiomyocytes following cardiac injury. Both human and mouse heart are known to fully regenerate in response to injury at the neonatal stage. This capacity, however, sharply declined after birth. Previously, although cardiac tissue-resident macrophages (cTMs) have been shown to be required for cardiac regeneration, the detailed mechanisms remain understood. Herein, we aim to identify distinct macrophage populations that are required for the regenerative capacity and determine the underlying mechanisms. First of all, bulk RNA sequencing (RNA-seq) revealed an entirely different gene profile in the neonatal (P1-3) vs. adult (8 weeks) mouse heart on day 10 post-myocardial infarction (MI) surgery, which oxidative phosphorylation and EIF2 signaling were up-regulated in neonatal, whereas Wnt signaling, inflammation and fibrogenic activities were activated in adult, cardiac macrophages. Next, single-cell RNA-seq analyzed 4 of the 10 distinct clusters of cardiac macrophages were highly enriched in neonatal, but not in adult. One of the neonatal cTMs clusters, CD36hi cTMs, exhibit phagocytic, anti-inflammatory and pro-reparative transcriptional signatures. Moreover, the temporal dynamics demonstrated that CD36+ cTMs were sharply declined on D1 post-MI, but quickly replenished in neonatal, but not adult, *Cx3cr1-GFP* mouse hearts on D3 to D21 post-MI. Functional blockade of CD36 using anti-CD36 antibody suppressed cardiomyocyte proliferation and resulted in excessive scarring in neonatal mice following apical resection (AR) surgery. Furthermore, targeted deletion of CD36 in cTMs (*CD36cKO*) significantly abolished the regeneration capacity in neonatal hearts. More importantly, the adoptive transfer of *CD36+* cTMs from neonatal mice hearts improves cardiac function in adult mice hearts following MI surgery. Taken together, these results demonstrate the critical role of CD36 in cTMs to promote cardiac repair and regeneration, which could be a potential therapeutic strategy to facilitate myocardial recovery in MI and HF.

**RESTORING DAMAGED MYOCARDIUM BY MICRORNA AND G-ACTIN MONOMER BINDING PROTEIN**  
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Myocardial infarction causes massive loss of cardiomyocytes, leading to high mortality rate and heart failure. Cardiac regeneration is thought to have therapeutic potential for myocardial infarction and heart failure.

We recently found that Nkx2.5+ cardiac myoblasts exist in declining number in the hearts of postnatal mice. The Nkx2.5+ cardiac myoblasts reactivate after myocardial infarction. They reside mostly in the subepicardium. Using inducible Nkx2.5 enhancer-Cre/ROSA26 reporter double transgenic mice to lineage trace the fate of activated Nkx2.5+ progenitor cells, we documented that the activated Nkx2.5+ cardiac myoblasts proliferate and differentiate into mature cardiomyocyte *in vivo*. We also documented that Nkx2.5+ cardiac myoblasts originate from the embryonic epicardial cells.

Thymosin ß4 is a G-actin monomer binding protein, involved in cell proliferation, migration, and differentiation. We hypothesized that Thymosin ß4 would promote Nkx2.5+ cardiac myoblasts, an embryonic epicardial-derived cells, mobilization and cardiomyogenesis, after myocardial injury.

Various microRNAs (miR) have been reported to control cardiac development and promote cardiac regeneration. We have transfected neonatal Nkx2.5+ cardiac myoblasts with various microRNAs and found miR-590 markedly promotes cardiomyogenesis capacity of cardiac myoblasts.

We have proved that miR-590 plus Thymosin ß4 markedly improved murine cardiac function after myocardial infarction. In the treatment group with miR-590 plus Thymosin ß4, LVEF improved from 45% (Day 0) to 77% (Day 7), to 83% (Day 28), to 85% (Day 56) with no LV regional wall motion abnormality and minimal aneurysm formation. In comparison, the control group with miR-mimic plus PBS, persistent hypokinesia at apex and anterior wall with impaired LV contractility and larger aneurysm was revealed. Heart section of the post-MI mouse receiving miR-590 plus Thymosin ß4 showed minimal fibrosis in the outer layer of myocardium. In contrast, large fibrotic area was noted in the no treatment group. Survival benefit was also seen in the treatment group. Enhanced cardiomyocytes proliferation was documented at treated post-MI heart section.

        The study demonstrated administration of miR-590 plus Thymosin ß4 markedly enhance cardiac repair/ regeneration. We anticipate the result would promote cardiac regeneration in clinical use.

**REVERSAL MECHANISM OF HIS BUNDLE PACING ON MYOCARDIAL REMODELING IN PACING INDUCED CARDIOMYOPATHY FROM A PIG MODEL**  
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**Background**

Pacing-induced cardiomyopathy (PICM) is induced by left ventricular (LV) electrical or mechanical dyssynchrony. Although several ongoing clinical study try to evaluate the effect of physiologic pacing on preventing and treatment of PICM, it lacks pathological mechanism to support that.

**Purpose**

Creating a big animal model to assess the possible reversal mechanism of His-bundle pacing on PICM

**Method**

There were three animal groups created in this study, which were (1) sham control, (2) RV pacing group (RVOT pacing for six months), and (3) His bundle pacing group (i.e. RV pacing for six months followed by 3 months of His bundle pacing). Echocardiographic study was performed to assess the grossly LV function and dyssynchrony. Lipid metabolic staining, Western blot and qPCR were performed to assess the genetic and protein changes associated with lipid metabolism between groups.

**Results**

RV pacing group had significantly more LV mechanical dyssynchrony than sham control and His bundle pacing groups. In addition, the LV systolic dysfunction induced by 6 months of RV pacing could be reversed by switching to 3 months of His bundle pacing. In terms of lipid metabolism, triglyceride and diacylglycerol (DAG) were accumulated in the cardiomyocytes in the RV pacing group. The expression of LIPE gene was lower in the RV pacing group compared to the His pacing and sham control groups. Moreover, the expression of ER stress was significantly higher in the RV pacing group than the sham control and His pacing groups. Of note, the metabolic pathway related to lipid disorder could be reversed by His bundle pacing due to reactivation of LIPE. Moreover, the expression of PDK4 was significantly lower in the RV pacing group than the sham control group and consequently.

**Conclusion**

Our study showed that PICM was possibly induced increased intracellular triglyceride and DAG expression, and eventually increased ER stress. His bundle pacing could reverse those altered regulatory genes related to lipid metabolism and meliorated the ER stress induced by RV pacing and might account for the beneficial effects of His bundle pacing in reducing the incidence of PICM in patients needed dependent pacing.

**APPLICABILITY AND PROGNOSTIC IMPLICATION OF THE COMPASS CRITERIA AND THE ARC-HBR CRITERIA IN PATIENTS WITH PERIPHERAL ARTERY DISEASE**

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**Background**

In the COMPASS trial, low-dose rivaroxaban in addition to aspirin reduced ischemic events in patients with peripheral artery disease (PAD). This study aimed to evaluate the applicability of the COMPASS trial and the Academic Research Consortium High Bleeding Risk (ARC-HBR) criteria in real-world treatment of chronic PAD.

**Methods**

We applied the COMPASS enrollment criteria and the ARC-HBR criteria to identify 12,440 chronic PAD patients from the Chang Gung Research Database between 2001 and 2019. The COMPASS-eligible and COMPASS-excluded patients were compared for major adverse cardiovascular events (MACE; cardiovascular death, myocardial infarction, and stroke), major adverse limb events (MALE; lower limb revascularization and major amputation for arterial disease), and Bleeding Academic Research Consortium (BARC) type 3 or 5 bleeding. The COMPASS enrichment criteria and the ARC-HBR criteria were evaluated for their prognostic impact on the COMPASS-eligible patients and the overall PAD patients, respectively.

**Results**

A total of 3,569 patients (28.7%) were considered COMPASS-eligible, with HBR being the main reason for exclusion (n=8,837; 71%). At a mean follow-up of 4.3 years, the COMPASS-excluded patients had remarkably heightened risks of MACE (26.3% vs. 14.8%; adjusted hazard ratio [aHR] 1.44; 95% confidence interval [CI] 1.29–1.60), MALE (14.4% vs. 9.2%; aHR 1.31; 95% CI 1.14–1.5), and BARC type 3 or 5 bleeding (12.6% vs. 6.2%; aHR 1.81; 95% CI 1.54–2.12) compared with the COMPASS-eligible patients. An increased number of the COMPASS enrichment criteria or the ARC-HBR criteria was generally associated with incremental ischemic and bleeding risks. The COMPASS-eligible patients with high-risk limbs (amputation, critical limb ischemia, and peripheral vascularization) or multiple enrichment criteria had disproportionately higher rates of ischemic events than bleeding compared with those without these features.

**Conclusions**

In this multi-institutional cohort of chronic PAD, the prevalence of HBR was 71% and the eligibility rate for COMPASS was 28.7%. Both the COMPASS enrichment criteria and the ARC-HBR criteria facilitated risk stratification of patients with PAD. The COMPASS-eligible patients who had multiple enrichment criteria or high-risk limbs may be good candidates for intensified antithrombotic therapy with favorable risk–benefit tradeoffs.

**SEX-DIFFERENCES IN OUTCOMES OF CHRONIC AORTIC REGURGITATION IN ASIANS: A MULTICENTER STUDY**

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**Objective:** To examine clinical-differences between Asian men and women with hemodynamically-significant chronic aortic regurgitation (AR) and compare findings from westerners.

**Methods:** Consecutive patients with ≥moderate-severe AR (n=1305) undergoing echocardiograms were retrospectively identified from 2008 through 2022 from 3 tertiary referral centers. Endpoints included aortic valve surgery (AVS), all-cause death (ACD) and cardiovascular death (CVD).

**Results:** At baseline, compared to men (63±17 years; n=968), women (69±16 years; n=337) were older, more symptomatic, had more comorbidity, larger indexed aorta size, and larger left ventricular end-systolic dimension index (LVESDi) (all<.001). LVESDi>20mm/m2 was noted in 90% asymptomatic women versus 71% asymptomatic men (P<.001). The correlation of symptomatic status and degree of LV remodeling was better in men. Median follow-up was 3.9 (IQR:1.3-7.1) years. Women were independently associated with less AVS (P≤.0001); overall 10-year survival for ACD and CVD was better in men than women (P≤.002). However, 10-year post-AVS survival was similar between sexes (P=0.9). AR-progression related LV remodeling was similar between sexes (P=0.86). Multivariable independent determinants of ACD and CVD were age, symptoms, indexed aorta size, LV parameters, and Taiwanese (all P≤.04) but not female-sex; AVS was protective. Adjusted mortality cutoffs for LV ejection fraction, LVESDi, LV end-systolic volume index (LVESVi) and indexed aorta size in women are 53%, 26mm/m2, 44ml/m2 and 25mm/m2, respectively; the corresponding cutoffs in men are 53%, 23.5mm/m2, 50ml/m2 and 23mm/m2, respectively.

**Conclusions:** Sex-differences in Asian AR patients did exist and the survival condition was similar to western cohort. Women had survival penalty due to disadvantages they carried, including older age, advanced symptoms, more comorbidity, and less AVS, but not due to sex per se. It was encouraging that women had similar post-AVS survival versus men, suggesting that to close survival-gap in female patients, taking abovementioned sex-specific mortality-cutoffs into account for early surgical referral is important.

**CONSENSUS-CONCORDANT ANTITHROMBOTIC THERAPY IS ASSOCIATED WITH IMPROVED OUTCOMES IN PATIENTS UNDERGOING TRANSCATHETER AORTIC VALVE IMPLANTATION**

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**Background:** A consensus on management of antithrombotic therapy in patients undergoing transcatheter aortic valve implantation (TAVI) was published in 2021 by the European Society of Cardiology (ESC). However, the relationship between consensus-concordant care and patient outcomes is unknow. The present study aims to determine whether consensus-concordant care is associated with improved outcomes.

**Methods:** The study sample consisted of 458 consecutive patients (male/female = 217/241; average age 78 years) from 2013 to 2020 who received successfully TAVI. One-year survival free of net adverse clinical events (NACE) in terms of a composite outcomes of mortality, ischemic events (recurrent nonfatal stroke or myocardial infarction) and major bleeding (Bleeding Academic Research Consortium type 2-4) were compared between patients who received care concordant with ESC consensus recommended antithrombotic therapy and patients who received discordant care.

**Results:** Care concordant with the ESC consensus recommendations was associated with improved 1-year NACE-free survival, compared with consensus-discordant care (88.0 % vs.78.7%; log-rank P=0.006). In addition to consensus-discordance (HR 1.892, P=0.007), age (HR 1.068, P<0.001) and chronic kidney disease (HR 5.150, P<0.001) are independent predictors of NACE by Cox proportional hazard analysis. Multivariate Logistic Regression analysis identified that the presence of diabetes mellitus (OR 0.658, P=0.037) and pre-existing atrial fibrillation (OR 1.830, P=0.006) were associated with discordant care.

**Conclusions:** Care as outlined in the ESC consensus on antithrombotic therapy after TAVI is associated with improved 1-year outcomes. These findings support the use of an evidence-based approach to guideline development and assessment of quality of care in patient undergoing TAVI.

**Application of Artificial Intelligence in the Diagnosis and Treatment of Hypertension**

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Hypertension is a major cause of cardiovascular death and affected 1.39 billion people throughout the world. However, almost half of people are unaware that they have hypertension. It is therefore important to improve the diagnosis and monitoring of hypertension for better management of blood pressure and to reduce the risk of developing future cardiovascular diseases. Hypertension requires transformative solutions that can help reduce the global burden of the disease. Artificial intelligence and machine learning, which have made a substantial impact on our everyday lives over the last decade may be the route to this transformation.

**Interaction between lipid-lowering agents**

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Lipid-lowering agents, especially 3-hydroxy-3-methylglutaryl–coenzyme A inhibitors (statins), are widely used in the treatment and prevention of atherosclerotic disease. The benefits of statins are well documented. However, lipid-lowering drugs may cause liver function impairment or myopathy, the risk of which is increased by drug-drug interactions. We will review drug-drug interactions between lipid-lowering agents

Tricuspid regurgitation: Choice of management

成大醫院心血管外科　胡祐寧

The tricuspid valve has historically been a forgotten valve, and tricuspid regurgitation (TR) has been less emphasized compared to left-sided valvular heart disease. The consensus in the past, and even in current guideline, is that tricuspid valve surgery should be reserved for TR patients who are refractory to medical treatment but have not developed right ventricular dysfunction. However, recent literatures have shown that TR itself affects long-term survival rates and that the severity of TR is significantly correlated with survival. Furthermore, it has been found that secondary TR is not just a result of left heart failure or pulmonary hypertension, but can also exacerbate left heart dysfunction. Currently, interventional cardiology is aggressively studying transcatheter tricuspid valve repair and replacement. In this context, the ideas and approaches of cardiac surgeons towards TR should keep up with the times. This report presents several cases of TR in National Cheng Kung University Hospital and summarizes recent studies on tricuspid valve management.

**VALIDATION OF A NEW METHOD FOR ATRIAL FIBRILLATION DETECTION USING RR INTERVALS**Kuan-I Lee1, Chen-Chin Lee2  
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Background: AF is characterized by the irregularly irregular RR intervals (RRIs) in ECG recordings. Previous studies have attempted to use RRIs for AF classification. However, the presence of premature beats (PBs) is a major cause of AF misclassification because they increase the irregularity of RRIs. Identifying PBs accurately is essential for ECG recordings with irregular RRIs. In order to improve AF classification, we developed a rule-based algorithm to detect PBs. The "PhysioNet/CinC Challenge 2017" training set, containing 8,528 ECG recordings, was used for validation. These recordings included 5,076 normal sinus rhythms (NSRs), 2,415 other arrhythmias (OAs), 728 AFs, and 279 noises. Noise ECGs were excluded in this study. For this study, NSRs and OAs were collectively classified as non-AF ECGs.

Methods and Results: Our method calculates the relative difference of RRIs for all non-noise recordings. Two distinct PB patterns—PBs with compensatory pauses and PBs without compensatory pauses—were applied to identify isolated PBs. For segments with successive irregular RRIs, an additional algorithm was applied to detect the regular irregularities of possible mixed PBs.

Regular irregularities, if compatible with mixed PBs, would be considered PBs. Regularity scores were calculated from the RRIs from regular beats and PBs. Based on these scores, the system classified each ECG recording as either AF or non-AF and compared the results with the ground truth labels in the database. Our algorithm achieved a sensitivity, positive predictive value (PPV), and F1-score of 0.86, 0.73, and 0.79 for AF detection, and 0.98, 0.99, and 0.98 for non-AF detection. The combined F1-score was 0.88.

Conclusions: This proposed method is simple, efficient, and clinically interpretable. Its ability to detect PBs addresses a significant challenge in AF classification for single-lead ECGs. Furthermore, its low computational requirements and streamlined data transmission make it well-suited for remote, long-term monitoring in diverse healthcare settings.

**A NEW METHOD TO DETECT PAROXYSMAL ATRIAL FIBRILLATION USING THE RR INTERVALS**Kuan-I Lee1, Chen-Chin Lee2  
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Background: With the advancement of digital health technology, paroxysmal atrial fibrillation (PAF) can be detected using prolonged ECG monitors. AF is characterized by the irregular irregularities of the RR intervals (RRIs). An accurate and effective algorithm facilitates screening for asymptomatic PAF. Previous studies have used RRIs for AF detection. However, the presence of premature beats (PBs) increases the irregularity of RRIs and cause AF misclassification. RR intervals (RRIs) of PBs might be regularly irregular. Therefore, we developed a new method to detect PBs based on the RRIs. It improves PAF classification for ECG recordings with irregular RRIs.

Materials: The "PhysioNet/MIT-BIH Atrial Fibrillation Database" was used for validation. There were 35 ECG recordings with durations ranged between 9 hours 13 minutes 20 seconds to 10 hours 13 minutes 43 seconds. Each recording was segmented into one minute intervals and classified as non-AF or AF successively.

Methods and Results: For each segment of ECG, we calculated the relative difference of the RRIs. Two RRI patterns, PBs with or without compensation, were applied for identification of isolated PBs. ECG segments with successive irregular RRIs were analyzed by another algorithm to find the possibility of mixed PBs. Regularity scores were calculated from the regular RRIs and the PBs. Each ECG segment was classified into non-AF or AF based on the regularity scores. The result was compared with the label in the database. The overall accuracy by our algorithm was 0.98. For AF detection, the sensitivity was 0.96, the specificity was 0.99, the PPV was 0.98, and the F1-score was 0.97.

Conclusions: In this study, we proposed a rule-based expert system for PB detection and AF classification. It is simple, efficient, and clinically explainable for detection of PAF. Furthermore, its low computational requirements makes it easy for data transmission in remote, long-term monitoring in healthcare setti

**PREDICTION AND PREVENTION OF LEFT VENTRICULAR DYSFUNCTION USING ARTIFICIAL INTELLIGENCE-ENABLED ELECTROCARDIOGRAMS IN PATIENTS WITH NORMAL LEFT VENTRICULAR EJECTION FRACTION**

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**Background:** Left ventricular dysfunction (LVD) is a major contributor to heart failure and is associated with increased mortality. Artificial intelligence-enabled electrocardiogram (AI-ECG) accurately detects LVD, and its misclassification of LVD in patients without LVD has been proposed as a potential risk factor for future LVD. However, the predictive value of AI-ECG for future LVD and its role in guiding preemptive treatment remain insufficiently investigated.

**Methods:** This retrospective study included adult patients with a left ventricular ejection fraction (LVEF) ≥ 50% who underwent ECG at a regional and a tertiary hospital between January 2016 and December 2024. A previously developed ECG algorithm stratified patients into high-risk and low-risk LVD groups. Comorbidities, laboratory data, echocardiographic findings, and medication records were extracted from electronic health records.

**Results:** Among 38,624 included patients, 30,681 were stratified as low-risk and 7,943 as high-risk for LVD based on AI-ECG. Over a mean follow-up of 856 days, incident LVEF ≤ 40% occurred in 247 (0.8%) low-risk and 344 (4.3%) high-risk patients. AI-ECG demonstrated the strongest predictive ability (hazard ratio [HR], 5.78; 95% confidence interval [CI]: 4.91–6.81), surpassing chronic kidney disease (HR, 3.77), atrial fibrillation (HR, 3.07), acute myocardial infarction (HR, 2.79) and other comorbidities. This association remained significant after multivariable adjustment. Among high-risk patients on antihypertensive therapy, those receiving angiotensin-converting enzyme inhibitors (ACEi) or angiotensin II receptor blockers (ARB) had a lower risk of LVEF ≤ 40% (HR, 0.75; 95% CI: 0.56–0.99) and all-cause mortality (HR, 0.64; 95% CI: 0.48–0.84) compared to other antihypertensive treatments. This protective effect of ACEi/ARB was not observed in the low-risk group.

**Conclusion:** In patients with normal LVEF, AI-ECG effectively identified those at high risk for future LVEF decline, while ACEi/ARB therapy was associated with reduced adverse events. Further large-scale studies are warranted to establish a causal relationship between AI-ECG-detected LVD risk and the protective effects of ACEi/ARB therapy.

**AUTOMATED CARDIAC FUNCTION ASSESSMENT IN ACUTE MYOCARDIAL INFARCTION PATIENTS USING DEEP LEARNING MODELS ON CORONARY ANGIOGRAM VIDEOS**

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**Background:**  
Left Ventricular Ejection Fraction (LVEF) is a crucial measure for evaluating cardiac function in individuals with cardiovascular disease. Cardiac function can vary during cardiac catheterization, making real-time assessment essential. In acute myocardial infarction (AMI) cases, echocardiographic evaluation may not be readily available during the procedure. Typically, skilled clinicians estimate cardiac function qualitatively by analyzing the cardiac silhouette in coronary angiograms. This study aims to develop a deep learning model for the automated measurement of LVEF, particularly for patients experiencing AMI.

**Methods:**  
Coronary angiograms were collected from 6,088 patients from China Medical University Hospital. A total of 55,202 cine loops were randomly divided by case into training (64%), validation (16%), and test (20%) sets. We employed the SlowFast model architecture with the Deep Evidential Action Recognition (DEAR) method to classify LVEF as either ≥40% or <40%. Model performance was evaluated using AUC, sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV). We tested the model for clinical validation on an independent set of 10 AMI cases.

**Results:**  
The proposed model achieved an AUC of 0.8371, sensitivity of 0.8047, specificity of 0.7361, PPV of 0.4459, and NPV of 0.9346. In the clinical validation test, the model demonstrated an accuracy of 80%, with one true negative and one false positive.

**Conclusion:**  
Our deep learning-based classification model shows promising performance in predicting LVEF from coronary angiography videos. These findings suggest the potential utility of automated AI-assisted LVEF assessment in clinical practice in real time. Further validation in larger cohorts is warranted.

**GENERATIFE AI IN DIAGNOSIS OF PULMONARY HYPERTENSION: IMPACTS OF INVASIVE MEASURES**Jui-Tzu Huang, Shih-Hsien Sung  
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Background: Pulmonary hypertension is commonly found on patients with heart disease and has major prognostic implication. The gold standard for diagnosis is obtained through right heart catheterization. Early detection and possibly non-invasive diagnostic tool are essential.  
Methods: We enrolled 53069 patients received ECG and echocardiography. Pulmonary hypertension was defined as right ventricular systolic pressure more than 40 mmHg found on echocardiography, and around 1612 patients received right heart catheterization. Machine learning algorithm based on training set 80% and test set 20% with 5-fold cross validation.

Results: AI algorithms with CatBoost, XGBoost, LightGBM, and a multi-layered deep learning model (ResNet1D18) which reported the training accuracy and AUC as (0.850, 0.850), (0.846, 0.842), (0.849, 0.851) and (0.967, 0.953); the validation accuracy and AUC as 0.838, 0.842. Implement of generative AI layres with variational autoencoder generative adversarial network was added for model enhancement, probability of risk prediction also calculated.  
Conclusion: The machine learning algorithms provided robust performance on detecting PH. We may identify potentially important factors of PH on EKG through further feature selection.

**EARLY RISK STRATIFICATION OF ATRIAL FIBRILLATION AND ITS ASSOCIATION WITH LONG-TERM CARDIOVASCULAR OUTCOMES IN A TAIWANESE COHORT**

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**Objective**

Early identification of individuals at risk for atrial fibrillation (AF) is crucial for timely intervention and improved clinical outcomes. The increasing burden of AF underscores the need to identify high-risk individuals for targeted AF screening and primary prevention strategies.

**Methods**

The FIND-AF algorithm, developed using a random forest approach, was externally validated using data from National Taiwan University Hospital (2014–2019). The data were randomly split (80:20) for model evaluation. The predictive performance of FIND-AF-Taiwan, CHA₂DS₂-VASc, and C₂HEST models was assessed using the area under the receiver operating characteristic curve (AUROC), calibration slopes, and Brier scores. Risk groups were stratified based on the Youden index, and Kaplan-Meier analysis and Cox regression were used to examine the association between predicted high-risk status and long-term outcomes.

**Results**

Among 103,321 individuals, 4,544 (4.4%) developed incident AF. Patients with AF were older and had a higher prevalence of hypertension, heart failure, and ischemic heart disease. The AUROCs for 5-year incident AF were similar for FIND-AF-Taiwan (0.678 [95% CI: 0.661–0.695]), CHA₂DS₂-VASc (0.679 [95% CI: 0.661–0.697]), and C₂HEST (0.679 [95% CI: 0.662–0.697]). Calibration slopes were 0.972, 0.940, and 0.959, respectively. High-risk individuals identified by FIND-AF-Taiwan had significantly higher risks of AF hospitalization (HR: 8.25 [95% CI: 5.65–12.03]), heart failure hospitalization (HR: 55.29 [95% CI: 41.99–72.81]), ischemic stroke/TIA (HR: 10.53 [95% CI: 9.08–12.20]), CKD progression (HR: 3.24 [95% CI: 3.07–3.43]), cardiovascular mortality (HR: 5.73 [95% CI: 4.50–7.29]), and all-cause mortality (HR: 1.81 [95% CI: 1.68–1.95]) (all p < 0.001). Kaplan-Meier analysis confirmed significant differences in event-free survival (log-rank p < 0.001).

**Conclusions**

The FIND-AF algorithm demonstrated robust predictive performance in Taiwanese data, comparable to existing risk scores for incident AF. Stratification into risk groups effectively identified individuals at higher risk for adverse long-term outcomes, underscoring the importance of early AF risk stratification to guide preventive interventions.

**DYNAMIC TRANSITION OF CARDIAC FIBROBLAST CONTRIBUTES CRITICALLY TO CARDIAC REGENERATION**

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**BACKGROUND:**

Cardiovascular disease is the leading cause of death globally, with myocardial infarction (MI) often causing heart failure due to irreversible cardiomyocyte loss. While adult mammalian hearts have limited regenerative capacity, species like zebrafish, newts, and neonatal mice (within 7 days after birth)—can regenerate heart tissue through cardiomyocyte proliferation. Cardiac fibroblasts (CF), critical for ECM production, tissue repair, and angiogenesis, have been shown to be essential for cardiac regeneration in zebrafish, though their role in mammals remains unknown. This study aims to investigate the roles of CF in mammalian myocardial regeneration.

**METHODS & RESULTS:**

We investigated the role of CF in heart regeneration using neonatal Col1a2-CreERT2; Rosa26RCAG-tdTomato;iDTR mice. By ablating Col1a2-expressing CF and subjecting the mice to apical resection (AR) and myocardial infarction (MI) at post-natal day 5, we observed impaired cardiac regeneration. CF ablation led to reduced cardiomyocyte proliferation and paradoxically increased myocardial fibrosis, evidenced by extensive scar formation on Masson’s trichrome staining. CF depletion adversely affected survival, with mortality rates of 42% after AR and 55% after MI, compared to near 0% in controls. Lineage tracing confirmed that CF do not directly transdifferentiate into cardiomyocytes. Bulk RNA sequencing on CF isolated from neonatal and adult mouse hearts showed neonatal CF had elevated expression of genes related to cell cycle progression and cardiogenesis. Further insights were gained from single-cell RNA sequencing analysis on mouse cardiac cells (GSE153480), which revealed that in regenerative neonatal mouse hearts, CF populations transitioned from predominantly the FB4 (Col1a2+ S100a8+) cluster on day one post-injury to the FB3 (Col1a2+ CD36+) cluster by day three—populations that were scarcely present in non-regenerative hearts. These findings highlight a potential critical role for FB3 and FB4 in cardiac regenerative process.

**CONCLUSION:**

Our findings reveal that Col1a2-expressing cardiac fibroblasts are critical for neonatal heart regeneration. Their ablation severely impairs regeneration and increases scarring in the neonatal mouse heart. While bulk RNA sequencing demonstrates enhanced cell cycle and cardiogenesis gene expression in neonatal CF, scRNA-Seq reveals dynamic shifts from FB4 to FB3 populations during regeneration, highlighting potential novel therapeutic targets for heart failure.

**PROTEOMIC SIGNATURES TO DETECT PRIMARY ALDOSTERONISM IN HYPERTENSIVE PATIENTS**

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**Backgrounds:**Primary aldosteronism (PA) is a major cause of hypertension and cardiovascular disease; however, diagnosing PA remains challenging. We investigated whether deep proteomic analyses could be used to diagnose PA in hypertensive patients.

**Methods:** We enrolled 52 patients with unilateral PA and 46 with essential hypertension (EH) and divided them into training and validation cohorts. Plasma samples were collected at baseline from all patients and again from PA patients after adrenalectomy. Deep proteomic analysis was performed to identify potential peptide signatures. These proteomic features were utilized to develop a classification model for distinguishing PA from EH in the training cohort. The classification model was subsequently validated in the validation cohort and post-adrenalectomy PA patients.

**Results:** After proteomic analysis, six peptide features including HBB, FIBA, Complement CO7, ALBU, C4BPA and A2AP were selected to generate risk scores and develop a classification model for distinguishing PA from EH. Risk scores were significantly higher in PA patients compared to those with EH. The classification model had a sensitivity and specificity of 80.5% and 83.3%, respectively, for diagnosing PA in the training cohort, and 81.8% and 80.0% in the validation cohort. The model demonstrated strong performance with an area under the curve (AUC) of 0.92 for distinguishing hypertensive patients with or without PA. Post-unilateral adrenalectomy, the risk scores showed a significant decrease.

**Conclusions:**Proteomic analyses can identify signatures that distinguish PA from EH. These findings may support the utility of proteomics in the diagnosis and treatment monitoring of PA without discontinuing medication.