Taiwan Society of Cardiovascular Interventions

臺灣介入性心臟血管醫學會

87期 會訊

2022年6月

2022_06_18 Carotid Stenting Certificating Course大合照

臺灣介入性心臟血管醫學會會訊（第八十七期，June，2022）

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目　錄

各位親愛的會員平安：

在五、六月疫情肆虐間，本會仍然積極召開線上理監事會及各委員會事務之推動。

5 月 28 日 Intervention Complications 之教育訓練，邀請了 8 位優秀的講者分享有關 Stent Dislodge、Coronary Fistula、Aortocoronary Dissection、Rotablator & TAVI Complication、Coronary Perforation and CTO Retrograde Dissection 等議題，會員參加很踴躍，討論也非常深入精采。

6 月 18 日與 TSOC 合辦 Carotid Stenting Training Course。另 6 月 25 日週邊委員會也舉辦了 PAD Update 教育訓練，所以這季從頸動脈、冠狀動脈、週邊動脈及瓣膜等議題皆有深入的探討。接下來七月即將有精彩的介入藥物討論會，也會主辦介入專科醫師的 Board Review Courses，當然最重要的是 7 月 30-31 日的台中金典酒店舉辦的 2022 夏季會，歡迎各位會員踴躍報名參加。

在六月份我們介入學會也有了自己永久的會址並做了喬遷，讓學會秘書處各項工作能更穩定的規劃發展。

在此要感謝各委員會的貢獻，安排有經驗的主持人及講者，也感謝會員踴躍的參與。

值此 Covid-19 慢慢趨緩之際，必要時我們會用 Hybrid 的方式來舉辦會議，方便大家能擴大參與，在防疫與學習皆能並進。

祝福大家

健康 平安 喜樂

教育委員會主委 洪大川

2022.6

公 告
會址喬遷啟事

為了給予會員更好的服務，讓學會運作更順利，

本學會已於民國 111 年 6 月進行喬遷，

歡迎各位蒞臨參觀指教！

臺灣介入性心臟血管醫學會

新會址：台北市忠孝西路一段 50 號 18 樓之 34

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臺灣介入性心臟血管醫學會 入會申請表

填表日期： 年 月 日

姓 名		性 別	□男 □女	貼相片處 (實貼一張)
英文姓名		身分證 號 碼		
出生日期	年 月 日	出生地	省(市) 縣(市)	
最高學歷	學校			科系（所）
現任醫院		單位/職務	/	
戶籍地址		電話（必填）	O:	
			H:	
通訊地址	□ 同戶籍地址 □ 通訊地址 _____		M:1. 2.	
E-mail(必填)	@		Fax:	
最近一年介入性工作經歷	(1) 醫院：_____ 期間： ___ 年 ___ 月至 ___ 年 ___ 月 醫師主管姓名：_____ 列印後主管簽名：_____ (2) 醫院：_____ 期間： ___ 年 ___ 月至 ___ 年 ___ 月 醫師主管姓名：_____ 列印後主管簽名：_____ (3) 醫院：_____ 期間： ___ 年 ___ 月至 ___ 年 ___ 月 醫師主管姓名：_____ 列印後主管簽名：_____			
推薦會員（1）	姓 名：_____ 列印後簽名：_____	推薦會員（2）	姓 名：_____ 列印後簽名：_____	

審查結果 (此欄由審查人員填寫)	□ 同意入會 □ 不同意入會 審查人員：	會員類別	□ 普通會員 □ 準會員 □ 名譽會員 □ 贊助會員	會員證號 碼	

6 會務活動-入會申請書

本人茲遵照　貴會章程之規定，申請加入　貴會為會員，遵守 貴會一切章程、簡則、決議等，謹此檢具各項證件，敬希　鑒核准予入會。

此致　臺灣介入性心臟血管醫學會

申請人：　　（簽章）

中　華　民　國　　年　　月　　日

繳驗資料：

□1. 入會申請表一份（共兩面）
□2. 本人二吋照片共三張
□3. 身分證正反面影本一份
□4. 最高學歷畢業證書影本一份
□5. 醫師會員 -- 心臟專科醫師證書影本一份（若無，請附醫師證書影本一份）
　　醫事會員 -- 師級醫事人員資格證書（護理師或放射師或醫檢師）影本一份
□6. 服務（在職）證明正本一份

注意事項

一、準會員申覆為普通會員：

1. 請在入會申請表左上角自行加註「準會員申覆普通會員」字樣。
2. 證明從事介入性心臟血管醫學實務工作滿一年，須由現職主管簽章。

二、列印入會申請表格，填寫完整後，將紙本資料備齊全，郵寄至學會進行審查。

三、介入性工作經歷

1. 醫師準會員指真正從事介入性工作日起算，醫師普通會員指取得心臟專科證書起算。
2. 醫事人員指真正從事介入相關工作日起算。

四、醫師申請入會之兩位推薦會員，必須為本會之**普通會員**。

五、介入性工作經歷須由**現職之醫師主管在「最近一年介入性工作經歷」欄位親自簽名**。

臺灣介入性心臟血管醫學會 秘書處
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臺灣介入性心臟血管醫學會
第九屆第二次醫事人員委員會會議紀錄

一、時　　間：111 年 5 月 16 日（星期一）18：30

二、地　　點：線上會議

三、出席人員：主　委：郭宜蘭
副主委：曾欽煇
委　員：王鳳花、何智仰、吳莉娟、李庚原、李素珠、林宜慶、林莉萍、林瓊枝、邵雅芬、陳良維、陳橙葦、黃漢龍、黃銘玲、潘龍發、蔡其峰

四、請假人員：無

五、列席人員：理事長：李文領
秘書處：黃啓宏秘書長；秘書：賴瑋儀、陳詠潔、劉子瑄 (記錄)

六、報告事項：

七、議程：

提案一：討論 2022 夏季會醫事人員教育訓練課程安排。

說明：1. 2022 夏季會預計於 7 月 30-31 日於台中舉辦，醫事人員課程時段為 7 月 31 日。

2. 第一次醫事人員委員會會後投票表決之 2022 夏季會課程主題方向如下：

(1) Structural Heart Disease Update

(2) Complication Troubleshooting

(3) OCT 及 IVUS 在 PCI 應用之優點

(4) Functional Studies (iFR/DFR) 種類，臨床的運用與選擇

(5) 低溫治療 for OHCA/IHCA

3. 2022 夏季會課程安排如附件一。

※決議：決議通過，將於學術委員會上追認後進行邀請。

提案二：討論 TTT 2023 醫事人員教育訓練課程安排。

說明：參考過去節目規劃：2021 TTT 及 2022 TTT 醫事人員繼續教育課程節目表。

※決議：將至下次委員會時再進行討論。

提案三：討論下次召開會議日期。

※決議：會後將於醫事人員委員會群組中進行調查。

八、臨時動議

九、散會

社團法人臺灣介入性心臟血管醫學會
第九屆第二次理監事聯席會會議紀錄

時　　間：2022 年 5 月 18 日（星期三）下午 6 點

地　　點：線上會議

出席人員：【主　席】李文領
【理　事】高憲立、方慶章、詹世鴻、曹殿萍、洪大川、許榮城、劉世奇、鄭正忠、王光德、盧澤民、陳冠宇、陳俊吉、郭風裕、黃群耀、傅雲慶、王宇澄
【監　事】顧博明、黃偉春、任勗龍、王怡智

請假人員：【監　事】張其任

列席人員：【秘書處】李政翰主委、郭宜蘭主委
秘書長：黃啓宏
秘　書：陳詠潔（紀錄）、劉子瑄、賴瑋儀、黃玉卉

一、主席致詞

各位理監事、好朋友大家好，秘書處原本已經安排盛宴讓大家齊聚一堂討論會務。但是因為疫情，所以臨時改為線上會議，飯店的部分我們保留到下一次。我先向大家報告一個好消息，就是關於 TAVI 給付的年齡限制，昨天我跟高憲立理事在蘇峻弘委員的牽線下，跟林靜儀立法委員以及健保署官員進行溝通，這是非常好的開始，大致決議就是回歸手術本質，取消年齡限制。後續健保署還會有 HTA 評估報告，我們會持續追蹤。那我們就開始今天的會議。

二、秘書處報告

(一) 請確認第 1 次理監事會議紀錄 (參閱附件一)

(二) 2022 年研討會活動規劃

序號	時間	主題	備註
1	3 月 27 日	Interventions Acute Coronary Syndrome	
2	4 月 23 日	Push to Limit (Structure Heart Disease)	
3	5 月 28 日	Intervention Complications	Hybrid
4	6 月 18 日	Carotid Stenting Certificating Course	
5	6 月 25 日	PAD Update	
6	7 月 9 日	介入藥物討論會	
7	7 月 10 日	Board Review Courses	TSCI 主辦

8	7 月 30-31 日	2022 夏季會	台中金典酒店
9	8 月 27 日	醫事人員教育課程 II	
10	9 月 3 日	醫事人員教育課程 I	
11	9 月 17 日	結構教育訓練	
12	10 月 22 日	Basic Skills in Coronary Interventions	
13	11 月 19 日	Peripheral Live 2022	台中榮總
14	12 月 3 日	Calcified Lesions: My Best/ Worst/ Failure Cases	
15	112 年 1 月 7-8 日	TTT 2023	

(三) 指派 5 位新任副秘書長名單及負責工作

副秘書長	負責工作
林俊呈	1. 會訊：第 81、91、95 期 2. 線上會議，行動會議上線建置
賴志泓	會訊：第 88、92、96 期
蘇峻弘	1. 會訊：第 89、93、97 期 2. Live Demo Results 報告規劃
朱俊源	會訊：第 90、94 期
劉俊廷	Live Demo Results 報告規劃

三、各委員會工作進度報告

1、學術委員會 _ 盧澤民主委

目前夏季會已經安排好，詳細各節 Session 內容也有委請相關委員會進行安排，包括週邊 / 結構 / 醫事人員，也都安排得很好，大家可以看一下螢幕分享，結構 Agenda 還沒有出來，我們會再催繳。Case 的部分因為投稿數量不是很多，所以收件延長到 5/30，也請各理監事可以多多鼓勵年輕醫師投稿參加，外週只有收到 2 篇投稿，歡迎大家踴躍投稿。這是 Post-Euro PCR 的 Agenda，是秘書長幫忙安排的，安排得很好，謝謝秘書長幫忙，大家也可以看看有沒有什麼問題。有任何建議都可以提出來。另外也是有稍微確定一下明年 Live Demo 的舉辦醫院，主要由北榮 / 高長 / 國泰三家醫院承辦，TTT2024 則暫定由亞東 / 奇美 / 臺大醫院舉辦，非常謝謝他們。Encore Seoul 也有提出要跟我們 Joint Session，我們已經把節目表安排好。以上是目前學術委員會的工作進度報告，謝謝大家。

2、教育訓練委員會 _ 洪大川主委

各位理監事大家好，我們教育委員會已經把今年的研討會都安排好了，剛剛秘書長已經有稍微報告了時間、主題，都非常精彩，詳細的安排議程前面有條列出來。5 月 28 日舉辦的 Intervention Complications 也快到了，是臨時從 11 月提前到 5

月，秘書處可以放出來給理監事看一下。6 月中的頸動脈認證課程也安排好了。副主委安排的藥物 Agenda 也在安排當中，很快就會出來，公告給大家。近期因為疫情的關係，我個人建議 6 月 30 日以前的活動改成線上，明年的活動主題也都安排好，但是具體舉辦的時間，會在下半年盡快確認讓大家知道，歡迎大家多多參與。

3、結構性心臟病委員會 _ 許榮城副主委

各位理監事大家好，教育訓練今年會有兩場，4 月跟 9 月，另外夏季會也有安排結構的 session。剛剛理監事也有建議一些方向，我們會朝這樣的方向去規劃。另外有一些關於健保的問題，我們也會持續跟健保署交涉，盡量幫大家多爭取一些。大概是這樣，以上報告，謝謝大家。

4、週邊血管介入委員會 _ 李政翰主委

週邊委員會有送兩個案子去健保署，更多爭取 Procedure Fee，但是目前有收到健保署回函要我們補件，我們會繼續整理資料，保持與健保署的溝通協調，有進度再跟大家報告。今年週邊委員會也規劃一些教育訓練，6 月 PAD、11 月 Peripheral Live，以及夏季會/TTT 也都有安排獨立的週邊時段。今年 11 月的 Peripheral Live 會在中榮舉辦，由王奇彥委員協助規劃。明年應該會規劃給一些年輕醫師學習的 Step by Step 的認證課程，細節會在委員會再討論。大家有任何建議，都歡迎提出來，謝謝大家。

5、國際暨兩岸交流委員會 _ 高憲立主委

我們這個委員會因為疫情處於怠速狀態，Encore Seoul 因為已經有規劃安排他們 Joint TTT，所以之後夏季會就不會再邀請他們 Joint，這樣的空缺之後就會安排 APCTO 一起參與，細節會再溝通。同時考量 Encore Seoul 與 TCTAP 之間的關係，也有建議 TTT 大會安排時要把這 2 個 Joint Session 時間稍微錯開。我們今年也先安排一些 APCTO 的人在今年的夏季會上來幫我們暖場，之後每年夏季會就會固定更多的參與。APSIC 部分，目前主席是印度的 Ashok，因為疫情的關係內部有協商讓他連任，所以我也有先寫信邀請他們加入 TTT 年會，對方很快就回應並答應我們了。

6、甄審委員會 _ 曹殿萍主委

各位理監事大家好，甄審委員會主要是舉辦口筆試，與 TSOC 合辦，包含 Board Review Course，另外就是審核一些入會 / 退會的名單。後面議程會有提案要追認的部分，再麻煩各位理監事。Board Review Course 及口筆試時間都已經安排好，各項名單，包括選題 / 口筆試 / 監考等都安排好了，請秘書處放出來給大家看一下。另外就是上一屆理監事有討論到，可能我們題目太簡單了，所以大家有建議將題目分為難 (30%)、中 (40%) 易 (30%) 的等級，也請講師在出題的時候也幫我們標註難易度等級。下次甄審委員會我們會討論是否建立題庫，大家有建議也可

以提出。等一下也有考試加分的提案跟大家討論，不知道各位理監事有沒有什麼Comment？沒有的話報告到這邊，謝謝大家。

7、財務委員會＿王光德主委

各位理監事大家好，我簡單跟大家報告，先看一下111年度預算表，之前已經通過了。主要是看一下110年度的決算表，我們去年決算比預算多了約1500萬，大部分還是廠商捐助，還有一個原因是疫情不能出國，所以結餘比較多。但是會務沒有因為疫情而中斷，我們還是有舉辦很多國內的研討會。感謝上一任的努力，學會買房子了也裝潢好，預計六月可以遷入。大家可以看一下基金的部分，根據人團法的財務管理辦法，我們要提撥一筆錢，就是存起來，未來有需要用的話要經過理監事會議、內政部核備才可以動。我們財務委員會提議提撥5%，等一下提案也有要追認這部分的，大家到時候可以再討論。預計在六月中可以搬入新的會所，算是很順利，謝謝秘書處幫忙監督進度。以上向各位報告。

8、公共醫療政策委員會＿黃群耀主委

理事長好，秘書長、各位理監事大家好，我們委員會在4月11日已經召開了第一次會議，主要是討論這一屆的工作計劃，跟往年比沒有太大的修正，主要是就公共政策與相關主管機關公文往來。也要謝謝各位理事以及許多專家的幫忙，因為很多時候是需要凝聚共識與意見才能對外發言。我們的發文一定是非常謹慎的，在發函之前都有經過理監事或是相關的專家確認內容，才會往外送，所以是十分謹慎的，這點是可以確定的。案件的部分，剛剛週邊委員會提到的Complex PTA、肺動脈瓣膜置換術等，這些已經在第一次會議取得支持與共識後送出，健保署也有來函要求補件，這部份我們會持續努力。另外就是，最近我們有委派柯文欽醫師、鄭正一醫師代表學會參加健保署的會議共識，請大家看一下秘書處現在呈現的會議紀錄，主要是第五案CTO微導管。其實這部份上一屆公共委員會已經有發函表示一些意見，我們經過討論也認為這樣納入全健保給付不太符合大家的利益，當時的建議也跟這次柯文欽委員代表的意見差不多。第二就是關於DEB的部分，相對單純一點。第三項比較特別，我覺得各位理監事也可以討論一下，有點埋伏筆的樣態，健保署請專家評估，也詢問幾家廠商意願，意圖把所有塗藥支架都納入健保。最後的決議主要是在場專家都建議要再研議，財務估算也要再評估。鄭正一委員代表的會議就是理事長開頭說的TAVI問題，感謝高P跟理事長在假日跟TSOC討論，又獲得立法委員的支持，讓我們可以進一步跟健保署溝通，看起來目前是往好的方向發展，我們再努力一下。等一下還有一個Impella的臨時動議，我們也想聽聽各位理監事的意見，再看看怎麼做比較好。謝謝各位委員幫忙。

9、編輯暨登錄委員會＿秘書長代

因為主委臨時有事，所以我代為報告。首先，登錄計劃部分會依照之前的

CHIP、RDN 繼續執行，詳細的收案狀況大家可以看一下螢幕上分享的。再來期刊的部分，第 13 期期刊會在夏季會發行，截稿日期是 6 月 5 日，第 14 期會在 TTT2023 出刊，15 期是明年夏季會，16 期是後年 TTT，基本上都排定好了，撰稿人、審稿人等都已經排定好寫成會議記錄，會依照會議紀錄排定的執行。

10、醫事人員委員會 _ 郭宜蘭主委

謝謝理事長將我們這一屆的委員會編制擴編到 17 人，我們也調整了北中南的比例。今年夏季會的醫事人員課程我們已經排好了，是群組投票產生的講題。以往 Q/A 時間不太夠，所以我們今年有把時間稍作調整，後續也請學會協助邀請作業。另外 TTT 的節目安排，我們會在年中的時候再召開委員會討論。以上報告，謝謝大家。

四、提案討論

提案一：追認事項 (新入會名單)

說明：111 年 3 月 16 日第 9 屆第 1 次甄審委員會通過入會申請名單如下：

◎醫師普通會員入會申請 (7 位)：

北區：王美英

中區：洪毓博、施淳友、蔡鴻義

南區：鐘國瑋、林彤宥、劉宜學

◎醫師準會員入會申請 (2 位)：

北區：林威辰

南區：陳昭佑

◎醫師準會員申請為普通會員 (5 位)：

北區：鍾伯欣、宋思賢

中區：陳科維

南區：黃邦碩、陳則瑋

◎醫事準會員入會申請 (3 位)：

北區：李尉鈞、黃雅雪

中區：陳良維

※決議：照案通過。

提案二：追認事項 (會員退會)

說明：秘書處收到會員主動要求退會信件，名單如下：

1. DN0377 趙嘉倫 (新店耕莘醫院安康院區)，退會原因：近來較少從事介入性心導管的業務。

2. DS0055 呂炎原 (郭綜合醫院)，退會原因：逝世。

※決議：照案通過。

提案三：追認事項：介專筆試加分辦法 (參閱附件二)

說明：1. 第 8 屆第 4 次甄審委員會中臨時動議決議：希望於筆試加分辦法中，醫學期刊後加註兩會雜誌名稱，並於未來筆試加分，投稿部分**僅限制**兩會雜誌發表之介入性醫學相關論文。會後將此決議送與心臟學會討論。
2. TSOC 已於 111 年 4 月 17 日之理監事會議上通過。
3. 預計 112 年度開始實施，提請討論。

※決議：照案通過。

提案四：追認事項：招募同院之普通會員 (參閱附件三)

說明：為鼓勵更多普通會員加入學會，秘書處彙整目前醫師準會員名單，請所有理監事及甄審委員會所有委員協助招募 3 個同院之普通會員。

※決議：照案通過，並將名單依院所分類，Email 予同院之理監事協助招募。

提案五：擬聘任 7 位名譽理事，提請討論。

說明：依本會章程第 24 條辦理，擬聘請：周嘉裕、曾春典、程俊傑、黃瑞仁、吳炯仁、殷偉賢、謝宜璋等共 7 位歷任理事長擔任本屆名譽理事。

※決議：照案通過，依本會【名譽理事聘任辦法】辦理。

提案六：擬定通訊選舉辦法草案，提請討論。(參閱附件四)

說明：1. 第 9 屆第 1 次會員大會已修改章程第十五條，理監事選舉得採通訊選舉。【人民團體選舉罷免法】第 23 條規定：人民團體之理事、監事選舉，得於章程訂定採用通訊選舉，並由理事會於預定開票日一個月前召開會議審定會員（會員代表）名冊，依名冊印製及寄送通訊選舉票。前項通訊選舉，人民團體應訂定相關辦法，載明選舉之通知、選務人員、投票規則及認定、開票、選舉爭議、當選人之通知、公告等事項，提經理事會議通過後實施，並報主管機關備查。

※決議：照案通過，此案報請主管機關核備。

提案七：110 年度財務結算後，從經費收入中提撥 5% 金額做為基金。

說明：2022 年 4 月 15 日第 9 屆第 1 次財務委員會討論通過。

※決議：照案通過，此案報請主管機關核備。

提案八：追認事項：減免本會會員一般教育訓練研討會之報名費，作為會員福利之一，TTT 及認證課程維持收費。非會員維持所有活動收費。**調整後**之收費標準如下：

單位：新台幣

		認證訓練	一般教育訓練研討會	TTT (2 天)
會前報名	**會員 - 醫師**	1,000	**免費**	500
	會員 - 醫事	200	**免費**	200
	非會員 - fellow	200	100	200
	非會員 - 醫師	2,000	600	1,000
	非會員 - 醫事	400	200	400
現場報名	**會員 - 醫師**	2,000	**免費**	500
	會員 - 醫事	400	**免費**	200
	非會員 - fellow	400	100	200
	非會員 - 醫師	4,000	600	1,000
	非會員 - 醫事	800	200	400

* 夏季會之會員 / 非會員報名費全免

說明：此案已於 2022 年 4 月 15 日第 9 屆第 1 次財務委員會討論通過，並於 4 月 23 日理監事 Line 群組取得過半共識，提報本次會議追認。

※決議：照案通過，將此福利於會訊等各管道公告週知。

提案九：將 TTT 2023 移至台中榮民總醫院舉辦，提請討論。(參閱附件五)

李文領理事長補充：有人提出這個建議，說因為國外會議例如 TCT 都會換地方舉辦，中南部也有一定的代表性，所以也應該輪流舉辦。但是也有例如 TSOC 年會固定在台北辦，CCT 也是固定在神戶辦。我個人沒有私心 ，主要以學會以及各理監事意見為主，台北行之有年，動線交通都很熟悉也很方便，所以今天提出來大家討論。我也事先特別請秘書處來中榮實際場勘，確認整體的空間、動線適不適合，有優點也有缺點，大致的狀況就如剛剛秘書長報告的。以上是我簡單的補充。

方慶章常務理事：我發表一下意見，TTT 歷年都在台北辦，以國外的 Guest 來說，台北比較有代表性，其他地方場地如果更大，我們可以把他做的比現在規模更大，那可能也具有代表性；若是

整體空間更小，把整個 TTT 規模辦得比現在小，我是覺得不太適合。以我來說，台北雖然較遠，但是捷運什麼的都很方便，中榮我還要計程車坐一段距離，對我來說不是那麼方便。所以很多因素要考慮，如果真的要換地方我覺得要考慮清楚。所以如果要投票的話，我選台北。

曹殿萍理事：我在哪裡辦是都沒有意見，但是我必須要提醒一下，除了攤位，我們還有一些 Learning Centers, Case Competition, Joint Session 等教室 301 或 401 那些，每間至少也要能容納一百多人，我們要確保新場地有這樣的空間，還有整個攤位區空間規劃，這些基本的結構都要確保有。台北也很好，交通很方便，行之有年大家也很習慣了。但是剛剛提的基本結構這些都要先考慮到。謝謝。

高憲立理事：我也是覺得換到台中也會有一些問題，如方副提出的交通問題要考慮。對於外賓來說，機場到會場，Operator 交通上的往來可能都不是很方便。

李文領理事長：我想這樣聽下來大家 Concern 的都差不多。但是有人提，我們就要正面回應人家。

黃偉春監事：我們在南部地區，所以我支持換地方，高雄也有大型會場，很多國際會議也曾經辦在高雄。對外賓來說，有機會讓他們認識台灣不同的城市也很好。

李文領理事長：我想各種方式或地區都有人支持或是不支持，但實際可不可辦，可能還是要請秘書處到現場去實際評估看看。到高雄辦也可以啊，那我們就請秘書處評估一下，因為秘書處實際執行的經驗比較豐富，對於場地空間、動線的安排抓得比較準確，那我們就請秘書處安排看看。至於 TTT2023 到中榮舉辦與否，我想大家提出的想法跟理由都非常充分，那我們就把今天的討論做成會議記錄來回應我們的會員。

郭風裕理事：Euro-PCR 之前也曾經辦在巴塞隆納，大會有統計過在巴黎辦的參加人數比較多。所以我們應該也可以統計夏季會/TTT 這些會議在不同地點的參加人數，不知道秘書處這邊可以統計數字嗎？大家可以再來評估看看。

賴瑋儀秘書：報告郭理事，本身夏季會就是因為考量 TTT 固定在台北，所以夏季會是中南部輪流舉辦。

郭風裕理事：瞭解，我想還是在台北舉辦參加的人數應該會相對多一點，再

來就是要考慮廠商贊助那些，換地方會不會影響他們的贊助意願，例如可能換地方參加人數變少或是有額外的交通支出，這些也要考慮進去。

李文領理事長：對對對，這也要考慮進去。那我想是不是請秘書處把這段的理監事發言做成會議記錄讓我們會員知道我們的想法與考量。這樣聽起來到中榮辦的支持者應該不多，我想提出的理由都是重點，這個議題我想我們就討論到這邊，如果大家沒有其他意見的話，請秘書長繼續主持。

※決議：考量台中榮總整體空間不敷使用，動線不佳，TTT 2023 維持於台北舉辦。

五、臨時動議

1. 高憲立理事提案：建議向健保署申請新增 Impella 診療項目。

※決議：照案通過，請高憲立理事先協助收集、整理資料，再提至公共委員會討論。

六、散會

臺灣介入性心臟血管醫學會
第九屆第二次學術委員會會議紀錄

一、時　　間：111 年 5 月 24 日（星期二）PM 6：30

二、地　　點：秘書處會議室實體會議 + 線上會議

三、出席人員：主委：盧澤民
委員：方慶章、任勗龍、洪大川、高憲立、張其任、張詩聖、許榮城、郭風裕、陳志成、陳俊吉、劉俊廷、顧博明、蔡政廷

四、請假人員：王怡智、曹殿萍、曹承榮

五、列席人員：理事長：李文領
秘書處：黃啓宏秘書長；秘書：賴瑋儀、陳詠潔、劉子瑄 (記錄)、黃玉卉

六、報告事項：

七、議程：

提案一：討論 2022 夏季會相關細節。

說明：1. 節目規劃 (參閱附件)
2. 2022 夏季會 case 收件情況 (目前統計至 5 月 24 日)：
A. Coronary CTO 5 篇
B. CHIP PCI / Complex PCI / Complications 12 篇
C. EVT 2 篇

※決議：1. 收件至 5 月 30 日，若 EVT 篇數仍然不多，會將此類的 Case 併到 TTT 2023，將 B 類拆成兩組，總共分 3 組做 Case Competition。若 Case 投稿較多篇則可選出前兩名。
2. 結構節目表再請許榮城委員及宋思賢醫師協助新增座長及安排講師 (可邀請台大林茂欣醫師)。

提案二：討論年度國際研討會 TTT 2023 節目及籌備之規劃。

說明：1. 討論節目時段及內容規劃 (參閱附件)
A. 請參閱螢幕 TTT2023 簡表。
B. 討論邀請外賓名單，請參閱螢幕 2021-2022 年邀請外賓名單。
2. 決定 Case Competition 徵稿的主題及初審委員
2021： Coronary CTO、CHIP、Complications、Structural Heart、Image/Physiology、Carotid/EVT

2020：Coronary CTO、CHIP、Complications、Carotid/Structural、Image/Physiology、EVT

2022：Coronary CTO、CHIP、Complications、Structural Heart、Image/Physiology、Carotid/EVT

Case Competition 初審委員：後續再安排

※決議：1. 國內以實體規劃，外賓以 Virtual 為主。

2. Live Demo 聯絡醫師：

參考螢幕

3. Joint Sessions 請下列委員協助參與規劃：

參考螢幕

4. 其餘 Session 由下列委員及委員會安排：

參考螢幕

新增邀請：Teguh Santoso、Kentaro Hayashida

Live Demo 新增邀請 Georg Nickenig (擅長 Mitral，委請台大林茂欣醫師聯繫)

5. TTT 2023 外賓邀請名單，依今日討論的部分先發第一波邀請。

6. 建議可以安排一些區域醫院的 Faculty。

提案三：討論召開 TTT2023 第一次籌備會議日期及出席名單。

說明：1. 確認日期。

2. 確認列席人員名單。

※決議：會後進行討論。

提案四：討論下次召開會議日期。

※決議：8 月 9 日及 8 月 16 日進行調查。

八、臨時動議

九、散會

臺灣介入性心臟血管醫學會
第九屆第二次編輯暨登錄委員會會議議程

一、時　　間：111 年 06 月 13 日（星期一）PM 7：00

二、地　　點：線上

三、出席人員：主　委：王宇澄
副主委：蘇峻弘
委　員：呂信邦、謝明哲、黃建龍、于慶龍、劉俊廷、劉維新、蘇河名、盧怡旭、徐千彝、吳卓鍇 、邱昱偉、王子林

四、請假人員：盧炯睿

五、列席人員：理事長：李文領
秘書長：黃啓宏
秘書處：賴瑋儀、陳詠潔、劉子瑄、黃玉卉

六、報告事項

七、議程

提案一：第九屆雜誌稿件第十三期進度。

說明：第十三期邀稿對象
1. 兩篇 Original Article（陳郁安、黃建龍）
2. 兩篇 Rereview Article（黃國書、黃啟）
3. 三篇 Case Report（蘇貞元、林俊呈、連朕緯）

※決議：1. 已收稿件 4 篇。
2. 成大蘇貞元醫師的部分聯絡催稿。
3. 第十四期開始邀稿。

提案二：學會各項登錄計劃、網路登錄系統之進度。

說明：1. CHIP 計劃進度說明。
2. RDN 計劃進度說明。

※決議：1. CHIP 計劃下期會議時請各 Site 完成所有登錄。
2. RDN 計劃儘量收案。
3. 登錄計劃經費約剩 60 萬，但須再留 CHIP 計劃多收的個案。

提案三：教學醫院評鑑學術性期刊認定。

說明：申請認定共需 6 期雜誌並在時間內出刊，目前醫策會期刊認定期數 3 期。

※決議：滿六期後申請。

提案四：討論本年度預訂召開會議次數、下次召開會議日期及委員們方便出席會議之週間時間。

說明：9 月 12~23 日。

※決議：之後再投票確認日期，依疫情狀況調整會議形式。

八、臨時動議

提案：The Effectiveness and Safety of Reduced dose Prasugrel in Taiwan 登錄計劃（附件一：含廠商回覆之內容）

※決議：相關計劃內容、經費與收案人數再與廠商協調討論。

九、散會

臺灣介入性心臟血管醫學會
第九屆第二次結構性心臟病委員會會議紀錄

一、時　　間：111 年 6 月 20 日（星期一）18：00

二、地　　點：Google Meet 視訊會議

三、出席人員：主　委：傅雲慶
副主委：許榮城
委　員：李應湘、宋思賢、鄭正忠、陳嬰華、劉尊睿、謝明哲、李永在、鍾宏濤、施志遠、鄭錦昌

四、請假人員：羅秉漢、林茂欣、周柏青、邱正安、蔡佳醍

五、列席人員：理事長：李文領
秘書處：秘書：賴瑋儀 (記錄)、陳詠潔、劉子瑄、黃玉卉

六、報告事項：

七、議程：

提案一：7/30-31 夏季會 Transcatheter Mitral Valve Intervention: Steps by Steps 課程內容，節目表詳螢幕。

說明：原規劃肺動脈瓣 TPVR 課程更改至 2022/09/17 舉辦。

※決議：1. 夏季會節目表無異議通過，惟 Topic 需再寫明確，請宋思賢委員修改後再與各講師確定。
2. TPVR 課程節目表會將由主委及鍾宏濤規劃。

提案二：「AS 病友衛教網站」內容確認

說明：https://listentohearts.com/

※決議：同意，需注意網站上文章需與政策及時更新，再請各位委員協助確認文章，若有需修改部分請與秘書處反映。

提案三：討論下次召開會議日期。

※決議：下次召開會議日期以 10/17(一)、10/24(一) 調查，擇多數人可出席的日期。

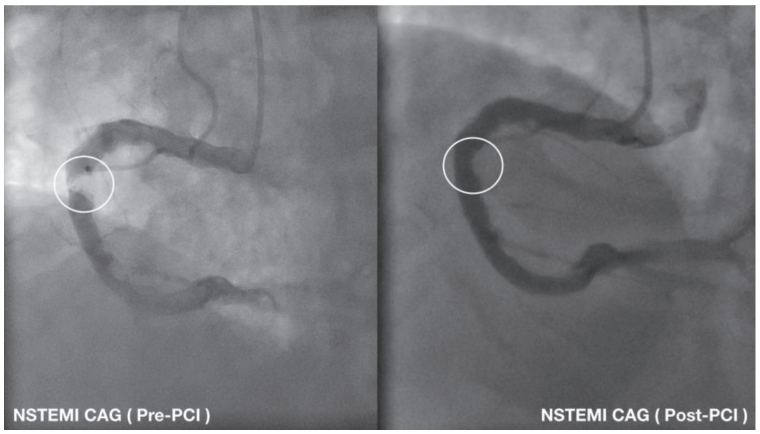
八、臨時動議

九、散會

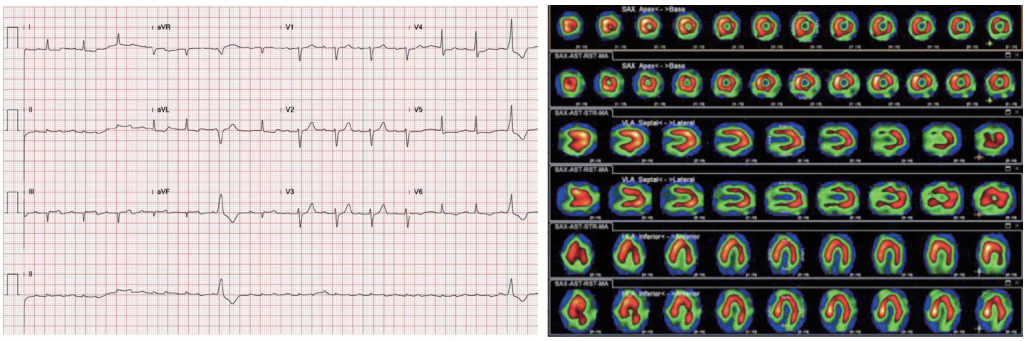
本期案例

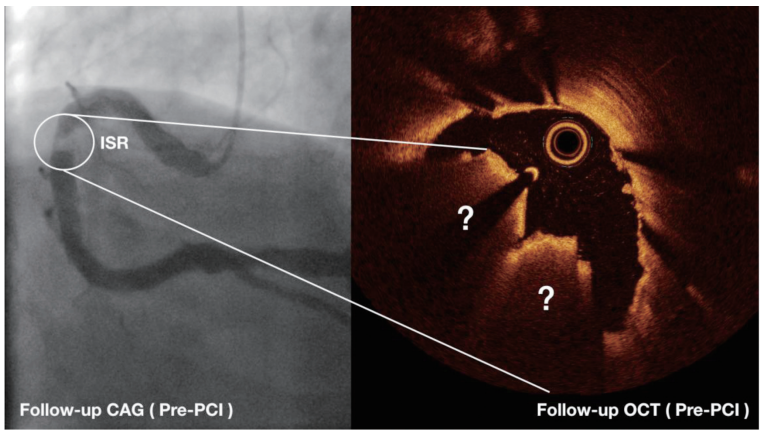
【案例】

66 歲女性患有糖尿病，及長期血液透析之慢性病史。於 2 個多月前因急性心肌梗塞 (Non ST-segment Elevation Myocardial Infarction)，入院接受右冠狀動脈塗藥支架置放術，術後順利出院。



然近日自覺胸部疼痛復發頻繁，回心臟科門診複診後安排心電圖，及核子醫學心肌灌注掃描，並再次入院行心導管併光學同調斷層掃描 (OCT) 複查如下：





【試問】

請問 OCT 影像中造成支架內再狹窄之組成 (Components) 為何？

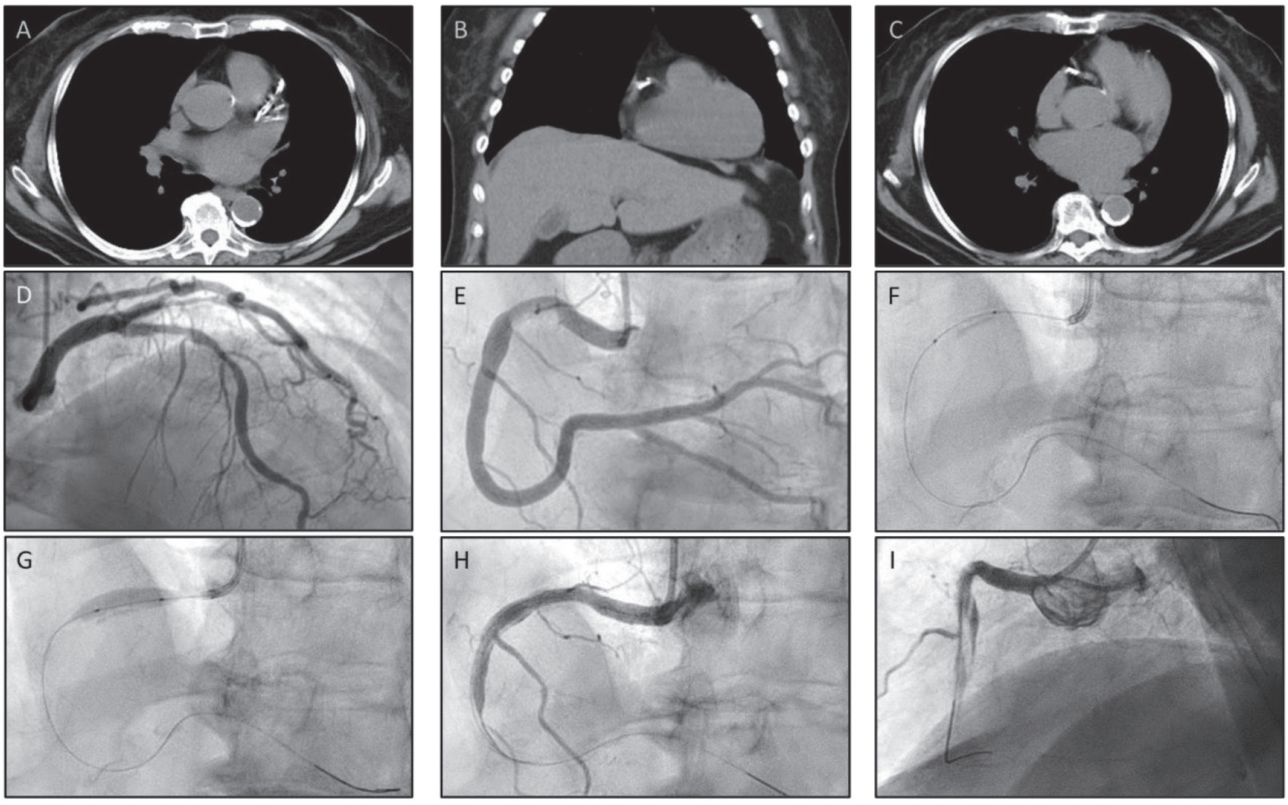
振興醫療財團法人振興醫院 鄧欣一醫師 / 黃建龍醫師

上期解答

【案例】

78 歲女性，有高血壓及高膽固醇病史約 20 年。因喘氣及胸悶症狀數月，至胸腔內科及心臟內科門診評估。期間除一般常規檢查，曾接受胸腔電腦斷層，及鉈-201 (Thallium-201) 心肌灌注掃描檢查。電腦斷層發現左、右冠狀動脈皆有明顯鈣化 (下圖 A, B,C)，心肌灌注掃瞄發現有明顯心肌灌流缺損。診斷為冠狀動脈疾病合併狹心症。因典型症狀加劇，藥物控制失敗，接受心導管檢查及治療。診斷發現三條血管均有顯著鈣化狹窄病灶 (下圖 D, E)，遂接受經皮冠狀動脈介入性治療。

右冠狀動脈前段 70% 鈣化狹窄處，以 3.5/15 mm 非順應性球囊 (NC Balloon) 反覆擴張，最大壓力達 22 atm 後，鈣化處才可完全擴張 (下圖 F, G)，但追蹤造影顯示右冠狀動脈中段全閉塞 (下圖 H, I)，此時病患也發生胸悶症狀，血壓心跳開始下降。



【試問】

此時發生什麼情況？鑑別診斷有哪些？該如何評估？要如何處置？

中國醫藥大學新竹附設醫院 心臟內科 劉俊廷醫師

【解答】

此種狀況為 abrupt vessel closure。鑑別診斷有 dissection, intracoronary thrombus formation, native thrombus (or atheroma) embolization, no reflow, air embolism, vasospasm, other unknown mechanism 等等。

首先要注意 Patient stability。病患不穩定，要進行 hemodynamic support and ischemia relief，可使用血管升壓劑 (vasopressors)，強心劑 (inotropes)，主動脈內幫浦 (intra-aortic balloon pump)，葉克膜 (ECMO)，甚至左心室輔助器 (left ventricular assist device) 等。

評估方式較不建議繼續 antegrade contrast injection，應使用 IVUS (不建議用 OCT) 評估原因，也可考慮利用微導管，在血管封閉處的遠段，小心進行 minimal contrast injection，評估遠端血管情況。

根據此病患的冠狀動脈攝影 (圖 H, I)，最可能是發生嚴重 dissection 及後續的 intramural hematoma (IMH) 阻塞血流。

處理嚴重 dissection 時，應專注於建立或維持血管暢通，保持冠狀動脈導絲在真腔內，避免使 dissection 進一步擴大。

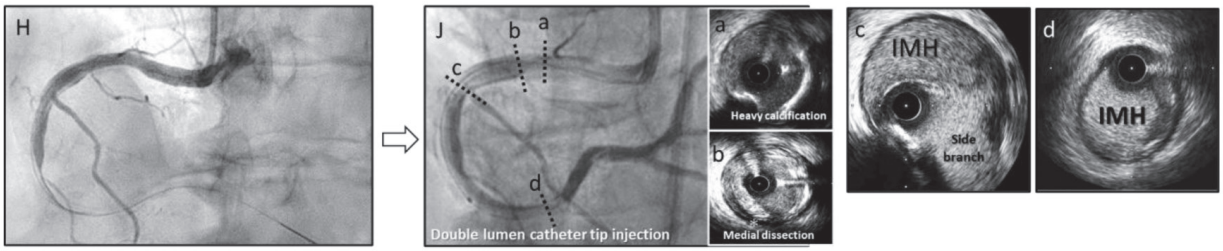
可考慮用小氣球反覆擴張，建立前行血流。要以 IVUS 評估型態，考慮置放支架處理 dissection。若有大塊 intramural hematoma 阻塞管腔，建議使用 cutting balloon 切割擴張，釋放血腫。

如果想要使用支架，處置很長的 dissection，建議先置放在 dissection 的遠端邊緣 (distal edge)。

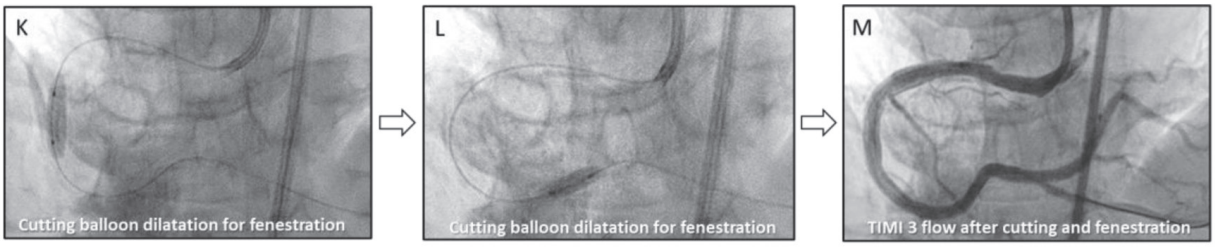
若導絲不慎走位，要重新想辦法進入真腔，以 spring-coil 導絲為主 (較不建議 polymer-jacketed 導絲)，以 IVUS 確定真腔位置，萬一導絲在假腔，無法順利進入真腔，可考慮 ADR, STAR 等方法回到真腔。

萬一無法重建血流，還是要考慮尋求外科協助繞道手術。

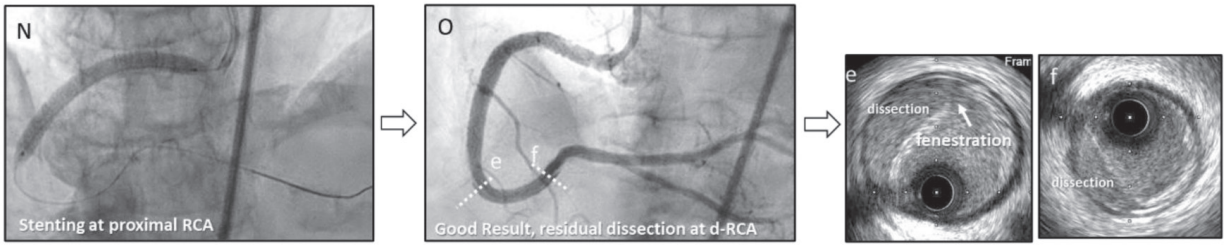
此案例先以 IVUS 確定為 long dissection 及 IMH，也用雙腔微導管在血管封閉處的遠段注射顯影劑造影，確定 IMH 延伸到右冠狀動脈的遠端。



後以 3.75 mm Cutting balloon 進行反覆擴張，讓 IMH 可以 fenestration，釋放血腫壓力，追蹤 IVUS 後證實 IMH 縮小，真腔變大，也重建 TIMI 3 血流。



最後在右冠狀動脈前至及中段置放一支 4.0 mm 長塗藥支架，再以 4.5 mm NC 球囊後擴。最後冠狀動脈血管攝影顯示支架擴張良好，TIMI 3 血流。雖然遠端殘餘 dissection，IVUS 也顯示確有 dissection，但血腫消失，原先血腫空間內高迴聲區域已經不見 (圖 e, f)，真假腔內血流迴聲相近，似有 fenestration 相通，因此遠端 dissection 處未置放支架。其後繼續順利完成左前降枝及左迴旋枝冠脈氣球擴張及塗藥支架置放，病患恢復狀況良好，隔日出院。



Reference:
1. A Practical Approach to the Management of Complications During Percutaneous Coronary Intervention. J Am Coll Cardiol Intv. 2018 Sep, 11 (18) 1797–1810.
2. Management of Percutaneous Coronary Intervention Complications. Circ Cardiovasc Interv. 2020 Jun;13(6):e008962.

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DOI: 10.1002/ccd.28329

CLINICAL DECISION MAKING

WILEY

SCAI clinical expert consensus statement on the classification of cardiogenic shock

This document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019

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[8]The CardioVascular Center, Tufts Medical Center, Boston, Massachusetts

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Abstract

Background: The outcome of cardiogenic shock complicating myocardial infarction has not appreciably changed in the last 30 years despite the development of various percutaneous mechanical circulatory support options. It is clear that there are varying degrees of cardiogenic shock but there is no robust classification scheme to categorize this disease state.

Methods: A multidisciplinary group of experts convened by the Society for Cardiovascular Angiography and Interventions was assembled to derive a proposed classification schema for cardiogenic shock. Representatives from cardiology (interventional, advanced heart failure, noninvasive), emergency medicine, critical care, and cardiac nursing all collaborated to develop the proposed schema.

Results: A system describing stages of cardiogenic shock from A to E was developed. Stage A is "at risk" for cardiogenic shock, stage B is "beginning" shock, stage C is "classic" cardiogenic shock, stage D is "deteriorating", and E is "extremis". The difference between stages B and C is the presence of hypoperfusion which is present in stages C and higher. Stage D implies that the initial set of interventions chosen have not restored stability and adequate perfusion despite at least 30 minutes of

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observation and stage E is the patient in extremis, highly unstable, often with cardiovascular collapse.

Conclusion: This proposed classification system is simple, clinically applicable across the care spectrum from pre-hospital providers to intensive care staff but will require future validation studies to assess its utility and potential prognostic implications.

KEYWORDS
cardiogenic shock, heart failure, hemodynamics

1 | INTRODUCTION

The treatment of acute myocardial infarction (MI) and heart failure (HF) has advanced exponentially over the last 50 years. One of the greatest advances has been the routine use of immediate percutaneous coronary intervention (Primary PCI) for ST segment elevation MI (STEMI) which has reduced mortality and subsequent HF substantially.[1] However, cardiogenic shock (CS) may occur prior to or following reperfusion. Even those who survive acute intervention may later develop CS and the overall 30-day mortality for patients with CS in association with MI is approximately 40–50%. Unfortunately, this incidence has not changed in the past 20 years since the publication of the landmark SHOCK (SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK) trial.[2–5]

The SHOCK trial was conducted when the only percutaneous form of cardiopulmonary support was the intra-aortic balloon pump (IABP). Since then, multiple devices (e.g., left atrial to femoral artery bypass devices [TandemHeart left ventricular assist device, LivaNova, London, UK], axial left ventricular—aorta pumps [Impella, Abiomed, Danvers, MA]), as well as similar devices for right ventricular support and veno-arterial (VA) extracorporeal membrane oxygenation (ECMO) have been developed and studied in the setting of CS.

Unfortunately, despite these efforts, CS mortality remains unacceptably high, and there are no prospective randomized trials showing that percutaneous mechanical circulatory support devices change the mortality in this clinical state.[3–9] It has been difficult to prove therapeutic benefit, in part, because CS patients are a heterogeneous population, and prognosis may vary widely based on etiology, severity of illness and comorbidities. CS encompasses a spectrum spanning from those at high risk of developing shock due to isolated myocardial dysfunction to those critically ill patients with severe multi-organ dysfunction and hemodynamic collapse to those with ongoing cardiac arrest. It is logical to expect that treatments may have widely varying outcomes in different patient subsets, including nonischemic subsets, and therefore a more granular classification of the CS spectrum is urgently needed to guide treatment and predict outcome.

1.1 | Purpose of a new definition

The purpose of the proposed SCAI Classification of CS is to provide a simple schema that would allow clear communication regarding patient status and to allow clinical trials to appropriately differentiate patient subsets. A few guiding principles served to organize the deliberations of the multidisciplinary team. First, the classification must be simple and intuitive without the need for calculation. Next, a new schema must be suitable for rapid assessment. Shock patients often deteriorate abruptly and therefore it is important that the schema be applied rapidly at the bedside upon patient presentation by a wide range of clinicians, as well as allowing reassessment as the patient progresses. In addition, a robust classification should be applicable to retrospective datasets or prior trials to examine whether the different shock categories correlate with definitive patient outcomes. Application of the schema may potentially identify differences between trials and perhaps explain why device-based therapies were or were not beneficial in those trials. This information would potentially inform the development of future trials. The writing group felt it critical that the schema had multidisciplinary applicability. We endeavored to develop a dynamic classification system that would be usable across all clinical settings including emergency departments, intensive care units, catheterization laboratories and others. It was equally important that the new system be actionable. An ideal schema would lead to changes in behavior such as facilitating the "hub-and-spoke" model of shock care, based on recognition of risk of deterioration and further adverse outcomes.[10] Lastly, the schema should have prognostic discriminatory potential. In other words, the different shock groups should reflect different morbidity or mortality rankings.

In the development of a new clinical acuity taxonomy for CS, we took inspiration from the American College of Cardiology/American Heart Association (ACC/AHA) classification of HF and the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) classification.[11,12] The INTERMACS classification is particularly useful due to key "tags" which serve as memorable ways to categorize patients. INTERMACS profile 1 is annotated "crash and burn", 2 is "sliding on inotropes", and profile 3 is "dependent stability". There is a temporary circulatory support modifier, but the INTERMACS classification does not distinguish between patients who were placed on ECMO support for refractory cardiac arrest, those who are stable on multiple inotropes and an IABP and those who received an Impella catheter to improve cardiac output while on inotropes. INTERMACS also does not have a construct to account for stability versus clinical deterioration, having been designed to classify patients at the single timepoint of durable mechanical circulatory support. The heterogeneity of patients described as INTERMACS 1 renders it difficult to compare outcomes across retrospective reports.

1.2 | Methodology

By design, the writing group included multidisciplinary representation reflecting the composition of teams which care for critically ill CS patients including active representation from cardiology (interventional, advanced heart failure, noninvasive), emergency medicine, critical care, and cardiac nursing. Cardiac surgery representation was sought and ultimately involved via peer review of the completed document. Broad involvement of the major professional societies was sought through representation on the writing group and peer review.

In accordance with SCAI Publications Committee policies on relationships with industry and other entities (RWI), relevant author disclosures are included in Supplemental Table S1. Before appointment, members of the writing group were asked to disclose all relevant financial relationships (>$25,000) with industry from the 12 months before their nomination. A majority of the writing group disclosed no relevant financial relationships. Disclosures were periodically reviewed during document development and updated as needed. The work of the writing committee was supported exclusively by SCAI without commercial support.

2 | THE CLASSIFICATION SCHEMA

There are five stages of shock labeled A-E in our proposed schema (Table 1, Figure 1).

Stage A: "At Risk" for CS describes a patient who is not experiencing signs or symptoms of CS but is at risk for its development. The Stage A patient may appear well and may have normal laboratories as well as physical examination. Patients with non-STEMI, prior MI as well as those with decompensated systolic or diastolic heart failure may fall into this classification which is quite broad. In general, anterior wall and large distribution infarcts carry a higher risk of cardiogenic shock but some patients may manifest shock with smaller infarcts in the setting of preexisting left ventricular dysfunction. A recent study notes the increasing incidence of shock in the ICU without myocardial infarction.[13]

Stage B: "Beginning" CS (Pre-shock/compensated shock) describes a patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion. Hypotension is defined as systolic blood pressure (SBP) <90 mmHg OR mean arterial blood pressure (MAP) <60 mmHg or >30 mmHg drop from baseline. Hypoperfusion is defined by clinical signs such as cold, clamped extremities, poor urine output, mental confusion, and the like. The physical exam of the Stage B patient may demonstrate mild volume overload and laboratories may be normal.

Stage C: "Classic" CS is a patient with hypoperfusion that requires an initial set of interventions (inotropes, pressor, mechanical support, or ECMO) beyond volume resuscitation to restore perfusion. These patients typically present with relative hypotension, with the majority manifesting the classic shock phenotype of mean arterial blood pressure (MAP) ≤60 mmHg or systolic blood pressure ≤90 mmHg along with hypoperfusion. The laboratory findings may include impaired kidney function, elevated lactate, brain natriuretic peptide, and/or liver enzymes. Invasive hemodynamics (if available) demonstrates the classic depressed cardiac index that is associated with CS.

Stage D: "Deteriorating" or "Doom" CS describes a patient who has failed to stabilize despite intense initial efforts and further escalation is required. Classification in this stage requires that the patient has had some degree of appropriate treatment/medical stabilization. In addition, at least 30 minutes have elapsed but the patient has not responded with resolution of hypotension or end-organ hypoperfusion. Escalation is an increase in the number or intensity of intravenous therapies to address hypoperfusion, or addition of mechanical circulatory support after the initial period of observation and treatment.

Stage E: "Extremis" CS is the patient with circulatory collapse, frequently (but not always) in refractory cardiac arrest with ongoing cardiopulmonary resuscitation (CPR) or are being supported by multiple simultaneous acute interventions including ECMO-facilitated CPR (eCPR). These are patients with multiple clinicians at bedside laboring to address multiple simultaneous issues related to the lack of clinical stability of the patient.

3 | DOMAINS OF PATIENT CHARACTERISTICS

We also categorized patients in three domains: biochemical (laboratory) findings, clinical bedside findings, and hemodynamics. Our classification does not legislate the presence of a particular number of findings but instead describes the common features that are prototypical of each stage.

3.1 | The arrest modifier-A

Cardiac arrest, however brief, is a significant event and usually worsens the clinical trajectory in ways that may be unforeseen. The $_{(A)}$ modifier is applied to describe patients who have had a cardiac arrest irrespective of duration (treated with chest compressions or direct current cardioversion). Accordingly, a patient may be in stage B_A shock, indicating stage B with a cardiac arrest complicating the clinical picture. This is distinct from the clinical picture of a stage E_A patient with prolonged cardiac arrest, severe clinical instability, often with numerous simultaneous interventions to maintain circulation. Whether a patient who presents with ventricular fibrillation in the setting of AMI and rapidly stabilizes with prompt defibrillation (stage B_A) has a similar or disparate survival as stage E_A will need to be examined in the future. Cardiac arrest and CS frequently occur together and the prognosis for the patient with both is worse than the presence of either cardiac arrest or CS alone.[14]

Two key components are the presence or absence of neurologic recovery and return of spontaneous circulation (ROSC). For example, a patient with out of hospital cardiac arrest (OHCA) intubated and sedated but with ROSC could be Stage A, B, C, D, or E. The prognosis for this patient may depend more on neurologic recovery than on myocardial failure.

TABLE 1 Descriptors of shock stages: physical exam, biochemical markers and hemodynamics

Stage	Description	Physical exam/bedside findings	Biochemical markers	Hemodynamics
A At risk	A patient who is not currently experiencing signs or symptoms of CS, but is at risk for its development. These patients may include those with large acute myocardial infarction or prior infarction acute and/or acute on chronic heart failure symptoms.	Normal JVP Lung sounds clear Warm and well perfused • Strong distal pulses • Normal mentation	Normal labs • Normal renal function • Normal lactic acid	Normotensive (SBP≥100 or normal for pt.) If hemodynamics done • cardiac index ≥2.5 • CVP <10 • PA sat ≥65%
B Beginning CS	A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.	Elevated JVP Rales in lung fields Warm and well perfused • Strong distal pulses • Normal mentation	Normal lactate Minimal renal function impairment Elevated BNP	SBP <90 ***OR*** MAP <60 ***OR*** >30 mmHg drop from baseline Pulse ≥100 If hemodynamics done • cardiac index ≥2.2 • PA sat ≥65%
C Classic CS	A patient that manifests with hypoperfusion that requires intervention (inotrope, pressor or mechanical support, including ECMO) beyond volume resuscitation to restore perfusion. These patients typically present with relative hypotension.	***May Include Any of***: Looks unwell Panicked Ashen, mottled, dusky Volume overload Extensive rales Killip class 3 or 4 BiPap or mechanical ventilation Cold, clammy Acute alteration in mental status Urine output <30 mL/h	***May Include Any of***: Lactate ≥2 Creatinine doubling ***OR*** >50% drop in GFR Increased LFTs Elevated BNP	***May Include Any of***: SBP <90 ***OR*** MAP <60 ***OR*** >30 mmHg drop from baseline ***AND*** drugs/device used to maintain BP above these targets Hemodynamics • cardiac index <2.2 • PCWP >15 • RAP/PCWP ≥0.8 • PAPI <1.85 • cardiac power output ≤0.6
D Deteriorating/ doom	A patient that is similar to category C but are getting worse. They have failure to respond to initial interventions.	***Any of stage C***	***Any of Stage C AND***: Deteriorating	***Any of Stage C AND***: Requiring multiple pressors ***OR*** addition of mechanical circulatory support devices to maintain perfusion
E Extremis	A patient that is experiencing cardiac arrest with ongoing CPR and/or ECMO, being supported by multiple interventions.	Near Pulselessness Cardiac collapse Mechanical ventilation Defibrillator used	**"Trying to die"** CPR (A-modifier) pH ≤7.2 Lactate ≥5	No SBP without resuscitation PEA or refractory VT/VF Hypotension despite maximal support

3.2 | Biomarkers

Biomarkers assist in assessing myocardial dysfunction severity as well as the response of peripheral organs and tissue in the setting of hypoperfusion. While no specific biomarker is diagnostic of shock due to a cardiac etiology, they do serve to support the diagnosis of a cardiac mechanism and provide information regarding the state of the patient at presentation as well as prognostic data as the care of the patient progresses. Frequency of testing will vary depending on the clinical scenario, the availability of rapid testing (or point-of-care testing) and the trajectory of the clinical course.

3.2.1 | Chemistry studies

Measurement of electrolytes, renal function parameters, specifically blood urea nitrogen and creatinine, and liver function tests are markers of vital organ hypoperfusion. Changes in creatinine provide important clinical prognostic features. It may be necessary to utilize the first measured value as previous baseline data may not be available. A creatinine of greater than 1.33 had a significantly higher mortality in the Intra-aortic Balloon Pump in CS (IABP-SHOCK II) trial.[15] Admission hyperglycemia, especially in patients without a known diagnosis of diabetes was also shown in this same trial to have a worse prognosis.[16]

3.2.2 | Creatine kinase and troponin

AMI is a common cause of CS. This complication may occur as a consequence of any type of acute coronary syndrome but occurs most frequently in STEMI.

If AMI is suspected, the diagnosis can be defined further using a variety of serum markers, which include creatine kinase (CK) and its subclasses (CKMB), and troponin (both I and T). Troponin T is an independent prognostic indicator of adverse outcomes and can be used as

FIGURE 1 The pyramid of CS classification

a patient risk-stratifying tool.[17–21] Elevation of troponin in CS may identify patients who present late.

3.2.3 | Lactate

Lactate (whether measured from arterial, venous or capillary blood) is an early marker of mitochondrial dysfunction and cellular hypoperfusion. Since it is commonly available, it has been extensively used in studies regarding the treatment of cardiogenic shock with evidence that increased levels are associated with adverse outcomes, but without consensus on a specific discriminatory value.[16,22–24] In general, arterial lactate is preferable since venous lactate is generally higher than arterial lactate and the 2.0 mmol/L cut-off is best established for arterial lactate. The interval of assessment is uncertain and has not been systematically evaluated but most commonly occurs at 1–4 hours. In stages C or higher patients, hourly or more frequent point-of-care testing may be more appropriate.[25]

3.2.4 | Blood gas measurements

Arterial blood gas determinations of acid–base status and the level of arterial blood oxygenation offer timely assessment of the patient's clinical status. Importantly, severe acidosis has a deleterious effect on myocardial contractility and response to certain vasopressors. A base deficit abnormality correlates with the occurrence and severity of shock. It is also an important marker to follow during resuscitation of a patient from shock to assess response to therapy.[26] Central venous and pulmonary artery oxygen saturations offer insight into tissue oxygen extraction, though pulmonary artery saturation is far preferable.[27–29] Serial evaluations are essential to determine clinical severity and response to therapy.

3.2.5 | Serum bicarbonate

Serum bicarbonate, especially when assessed early in the course of patients at risk of CS may provide information regarding prognosis. In a recent study by Wigger et al[30] serum bicarbonate decreased prior to significant elevation of lactate. A low bicarbonate level was a better predictor of 30-day mortality than the highest recorded lactate level.

3.2.6 | Brain natriuretic peptide (BNP) and emerging biomarkers

Brain natriuretic peptide (BNP) may be useful as an indicator of HF and as an independent prognostic indicator of survival in CS.[31,32] A low BNP level argues against CS in the setting of hypotension; however, an elevated BNP level does not establish the diagnosis as any form of cardiac ventricular or atrial stress may elevate levels of this peptide.

Although a number of biomarkers are under investigation, there are limited data to support their use in the acute evaluation of severity of CS. These include markers of inflammation such as fibroblast growth factor-23 (FGF-23),[33] GDF-15[15] high-sensitive C-reactive

protein (hsCRP), soluble tumor necrosis factor receptor-1 (sTNFR1), and angiopoietin-2.[34] As well, markers of apoptosis including sFas and sFasL, endothelin-1 (marker of neurohumoral axis activation), and procollagen II N-Terminal Pro-peptide (PIINP) as a marker of extracellular matrix turnover are all novel markers under study but not appropriate for routine clinical use.[32]

3.3 | Physical examination

In **Stage A** (*At risk*), patients typically have an unremarkable physical examination often with no signs of volume overload. They are warm, well perfused, with normal mentation. In **Stage B** (*Beginning*), patients have clinical manifestations of elevated right or left sided filling pressures as evidenced by an elevated jugular venous pressure and/or rales on auscultation, or a low BP but preserved end-organ and peripheral perfusion. The hallmark of **Stage C** (*Classic*) and **Stage D** (*Deteriorating / Doom*) is impaired end-organ perfusion. Patients in these categories appear in obvious distress and may exhibit impaired mental status, cold/mottled extremities, volume overload, reduced urine output (<30 mL/h), and/or respiratory failure requiring mechanical ventilatory support. The final **Stage E** (*Extremis*) manifests with cardiovascular collapse with a pulseless (or near pulseless) state and respiratory failure requiring mechanical ventilation.

3.4 | Hemodynamics

3.4.1 | Hemodynamic diagnosis of CS

Although all forms of shock are diagnosed by a relative reduction in systemic blood pressure with tissue hypoperfusion, labeling it *cardiogenic* implies that shock is due to a low cardiac output/index in the absence of hypovolemia. Although CS may be diagnosed clinically, it is often difficult to distinguish it from other forms of shock without invasive hemodynamic monitoring. It is essential to measure intracardiac pressures and cardiac output in patients where the diagnosis of CS is being considered. Intriguing new data suggests that use of PA catheter may be associated with lower mortality in CS patients.[35] Echocardiography may be a valuable adjunct, in particular to identify mechanical complications of myocardial infarction, acute valvular regurgitation and to identify signs of right or left ventricular volume or pressure overload. Other conditions such as pericardial tamponade can also be rapidly identified and may significantly affect management strategies.

3.4.2 | Blood pressure measurements

Systemic hypotension (defined as a sustained systolic blood pressure (SBP) less than or equal to 90 mmHg or a mean arterial pressure at least 30 mmHg lower than baseline) due to CS occurs after a reduction in stroke volume and cardiac output. SBP may be obtained by brachial cuff (cuff measurements in thigh or ankle may be artificially higher or lower), but an arterial line may be preferable to continuously monitor pressure and facilitate frequent arterial blood gas and lactate measurements. However, systolic amplification may occur when measuring arterial pressure in a distal location compared to central aortic pressure. An underestimate of central arterial pressure using a distal arterial line is also possible with peripheral arterial disease or with peripheral vasoconstriction either due to the shock state itself or the vasoactive drugs administered.

3.4.3 | Pulmonary artery catheter measurements

Pulmonary artery (PA) catheters can directly measure right atrial (RA), PA and pulmonary capillary wedge pressures (PCWP), mixed venous oxygenation, cardiac output (CO) and allows calculation of CI, systemic vascular resistance (SVR), pulmonary vascular resistance (PVR), pulmonary artery pulsatility index (PAPI), and cardiac power output (CPO). Recent reviews of the hemodynamics of CS provide further details on the derived values and interpretation of these indices in this setting.[36,37]

Although hemodynamic definitions of CS may vary, the National Cardiovascular Data Registry defines CS as systolic blood pressure ≤ 90 and cardiac index <2.2 L/min/m^2 and/or the requirement for parenteral inotropic or vasopressor agents or mechanical support to maintain BP and CI above these levels.[38] Although classic "cold, wet" CS is associated with low CI and high SVR and PCWP, there are four different common hemodynamic types of CS which are difficult to determine without invasive hemodynamic monitoring, and importantly, the patient may go from one category to another (Figure 2). There are two other uncommon types of CS (approximately 5% of cases): right ventricular shock and normotensive shock.[10]

The use of PA catheters can be critically important to establish the diagnosis of CS versus other causes of shock, unmask normotensive CS in patients with clinical hypoperfusion and SBP >90 mmHg, as well as accurately determining filling pressures. PA catheter hemodynamics are also helpful to assess right ventricular involvement in MI, distinguish classic cardiogenic from a mixed shock picture, assist in choice or titration of vasopressor or inotropic drugs, select patients who may benefit from mechanical circulatory support and guide weaning of pharmacological or mechanical support. Measurement of the saturation of PA blood, as well as CI and CPO are also very helpful to determine prognosis.

Despite these potential benefits, the use of PA catheters remains controversial in the wider setting. A recent analysis of the National Inpatient Sample of 89,718 AMI patients with CS who underwent cardiac catheterization revealed that only 6.1% received a PA catheter.[39] This retrospective report and others have not found a mortality benefit for CS patients who received PA catheters, although interpretation is limited given selection bias to use hemodynamic monitoring in sicker patients. The prospective, randomized ESCAPE trial in patients with decompensated HF showed no benefit, and was stopped early due to safety concerns (infection, ICD firing).[40] However, these patients did not have acute coronary syndromes or CS and all patients were enrolled with clinical equipoise. Accordingly, results of the ESCAPE study do not apply to patients with CS. There is no other randomized trial to evaluate the utility of PA catheters in cardiac patients, especially in those with

		Volume Status	
		Dry	Wet
Peripheral Perfusion	Warm	**Vasodilatory shock** (not CS) Increased cardiac index, low SVRI, low/ normal PCWP	**Mixed CS** Low cardiac index, low / normal SVRI, Elevated PCWP
	Cold	**Euvolemic CS** Low Cardiac index, high SVRI, low / normal PCWP	**Classic CS** Low cardiac index, High SVRI, Elevated PCWP

FIGURE 2 Different hemodynamic presentations of CS

CS and those being supported by mechanical support devices. We recommend the use of a PA catheter to diagnose and/or manage patients with CS, along with consideration of rapid transfer to experienced shock centers in the case of patients who require a higher level of care.

4 | MIXED SHOCK

The underlying cause of CS is by definition failure of myocardial function, and prompt measures to identify and address the underlying cause are of paramount importance. Other hemodynamic forms of shock can contribute to myocardial failure; however, as shock progresses, common pathways emerge leading to tissue and organ dysfunction, often involving inflammation and microcirculatory dysfunction.[41] These pathways can alter the hemodynamic profile of CS.

An analysis of hemodynamics in the SHOCK trial revealed that about 20% of patients had low SVR at the onset of CS.[42] Most of these patients had fever and leukocytosis suggestive of systemic inflammation, but not all of them were proven to have infection.[42] Such vasodilation can further exacerbate impaired systemic perfusion and decreased coronary perfusion pressure resulting from the initial CS state.

Distinguishing infection from systemic inflammation without infection can be challenging. Procalcitonin, an acute phase reactant released in response to endotoxin and other cytokines, is a highly sensitive marker for bacterial infection, and thus low levels may identify patients who do not require antibiotics.[43] Procalcitonin, however, has been shown to be elevated in HF[44] and so elevated levels may not be entirely specific for infection in patients with CS.

The potential for mixed shock emphasizes the importance of invasive hemodynamic monitoring in patients with CS. If patients do not respond rapidly to therapy based on the assumption that CO is low and filling pressures are high, mixed shock merits urgent consideration.

4.1 | Transitions of shock stage

Patients with CS often have dynamic clinical symptomatology and hemodynamics. In designing this classification, the authors acknowledge this and note that a patient may start at a stage B_A (beginning CS with a cardiac arrest) and then worsen over time to a higher stage. Whether transitions to higher or lower grade stages change the prognosis is unknown. For example, a patient who presents with Stage C shock, and rapidly improves following PCI of a proximally occluded left anterior descending artery might regress to stage B, but it is unknown whether the clinical trajectory is the same as a stage B patient who never develops hypoperfusion. Similarly, does the prognosis of a Stage C patient who deteriorates into Stage D but stabilizes on mechanical support and inotropes and can be weaned after 48 hours equal that of a stage C patient who never progressed in this fashion?

It is hoped that the use of the shock classification and application to patient datasets will allow such insights to be gleaned. Clearly validation in clinical datasets will be necessary to establish the utility of this proposed classification schema.

5 | PRACTICAL UTILITY OF SHOCK CLASSIFICATION

The authors recognize that the proposed classification schema presented (like most) is arbitrary and fairly simple. Some may wish for stricter definitions of stages, or to tie stages to laboratory values or some kind of a scoring mechanism. However, we feel that the elegance of the classification resides with its simplicity and that it is designed to be applicable across the care spectrum. The prognostic and therapeutic merits of the proposed classification schema are expected to be retrospectively and possibly prospectively validated.

5.1 | Example case

Mr. SL is a 67-year-old man with diabetes, hypertension, hypercholesterolemia and tobacco use who underwent coronary artery bypass grafting 10 years prior for severe three vessel coronary disease. He presents with vague chest pain which woke him from sleep. Further questioning indicates a crescendo pattern to angina and troponin T measured in the emergency department is positive. His blood pressure is 94/70 mmHg

and heart rate 100 beats per minute (BPM) but he normally has a blood pressure of 140/70 mmHg. He is scheduled to undergo diagnostic catheterization later in the day. In the new classification he would be assessed as **Stage B**. Later that day, in the catheterization laboratory, he is noted to be more tachycardic (heart rate 110 BPM), with reduced urine output. A PA catheter is placed and his cardiac index is 1.8/m^2 with a wedge pressure of 29 mmHg. He would be judged to be **Stage C** at this point. The team considers putting an IABP in but instead decides to intervene on a thrombosed saphenous vein graft to the right coronary artery. During thrombectomy, the patient has ventricular fibrillation and requires a single 200 joule shock by external pads. Now the patient would have the **A-modifier (Stage C_A)**. Low dose inotrope is started and the intervention completed successfully. An IABP is placed at the end of the case. Later that night in the intensive care unit, the patient's urine output continues to decline and the continuous cardiac index assessment remains below 2 L/min/m^2 despite increasing inotropes and IABP 1:1 counter-pulsation. The patient is now in **Stage D_A** and plans are made to escalate percutaneous support.

6 | CONCLUSION

Despite intense study, the mortality of CS in association with MI remains approximately 50% even with the development of percutaneous mechanical circulatory support devices. It is likely that prior trials have not been successful partially because some patients were “too sick” to benefit from the studied intervention. Others may do well with or without an intervention, and in the absence of a standardized classification system, it may be impossible to ascertain which groups may benefit. The schema outlined is a result of a broad multidisciplinary collaboration of experts to define the groups of patients who suffer from CS. The criteria are simple and clinically based, and if validated, this classification may become the “lingua franca” for the field. By having a common language, we hope to support communication at the bedside, in the catheterization laboratory, at the level of shock teams across institutions, and with clinical trialists as new approaches are tested to reduce the high mortality of CS.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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Standards and Guidelines

SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies

This statement was endorsed by the American College of Cardiology (ACC), American College of Emergency Physicians (ACEP), American Heart Association (AHA), European Society of Cardiology (ESC) Association for Acute Cardiovascular Care (ACVC), International Society for Heart and Lung Transplantation (ISHLT), Society of Critical Care Medicine (SCCM), and Society of Thoracic Surgeons (STS) in December 2021.

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Introduction

Since its development and release in 2019, the Society for Cardiovascular Angiography and Interventions (SCAI) shock stage classification for adult patients has been widely cited and increasingly incorporated, owing to its simplicity across all clinical settings, easily understood and visualized framework, and notable endorsement by relevant societies and organizations that manage cardiogenic shock (CS).[1] Ensuing validation studies over the course of the subsequent 2 years documented both its ease and rapidity of use as well as its ability

Abbreviations: ACS, acute coronary syndrome; AMI, acute myocardial infarction; CA, cardiac arrest; CCCTN, Critical Care Cardiology Trials Network; CICU, cardiac intensive care unit; CPR, cardiopulmonary resuscitation; CS, cardiogenic shock; HF, heart failure; LV, left ventricular; MCS, mechanical circulatory support; OHCA, out-of-hospital cardiac arrest; RV, right ventricular; SCAI, Society for Cardiovascular Angiography and Interventions; STEMI, ST-Segment Elevation Myocardial Infarction (STEMI).

Keywords: cardiogenic shock; heart failure; mechanical circulatory support.

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to meaningfully discriminate patient risk across the spectrum of CS, including various phenotypes, presentations, and health care settings. Nonetheless, several areas of potential refinement have been identified to make the classification scheme more applicable across all settings and clinical time points, given that data from validation studies have provided useful information not previously available that could serve to significantly refine the classification. With this background, a clinical expert consensus writing group of all relevant stakeholders was reconvened to re-evaluate and refine the SCAI SHOCK stage classification based on the existing literature and clinician feedback from real-world experience.

Key summary points

1. The SCAI SHOCK stage is an indication of shock severity and comprises one component of mortality risk prediction in patients with CS, along with etiology/phenotype and other risk modifiers; a 3-axis model of risk stratification in CS has been proposed to position the SCAI SHOCK stage in context.
2. Validation studies have underscored the correlation of the SCAI SHOCK stage with mortality across all clinical subgroups, including CS with and without acute coronary syndrome (ACS), cardiac intensive care unit (CICU) patients, and those presenting with out-of-hospital cardiac arrest (OHCA).
3. Progression across the SCAI SHOCK stage continuum is a dynamic process, incorporating new information as available, and patient trajectories are important both for communication among clinicians and for decision-making regarding the next level of care and therapeutics.
4. A hub and spoke model for transfer of higher-risk patients including those with a deteriorating SCAI SHOCK stage has been proposed.
5. Cardiac arrest (CA) as described herein relates to that accompanied by coma, defined as the inability to respond to verbal stimuli, most commonly associated with Glasgow Coma Scale <9, where there is concern for significant anoxic brain injury.
6. The SCAI SHOCK pyramid and associated figure now reflect gradations of severity within each stage and pathways by which patients progress or recover.
7. A streamlined table incorporating variables that are most typically seen, and the revised CA modifier definition, is also provided and incorporates lessons learned from validation studies and clinician experience.
8. The lactate level and thresholds have been highlighted to detect hypoperfusion but may be dissociated from hemodynamics in cases such as chronic heart failure (HF). In addition, patients may demonstrate other manifestations of end-organ hypoperfusion with a normal lactate level, and there are also important causes of an elevated lactate level other than shock.

Development methodology

This statement has been developed as per SCAI Publications Committee policies for writing group composition, disclosure and management of relationships with industry, internal and external review, and organizational approval.[2]

The writing group has been organized to ensure diversity of perspectives and demographics, multistakeholder representation, and appropriate balance of relationships with industry. Relevant author disclosures are included in Supplemental Table S1. Before appointment, members of the writing group were asked to disclose financial and intellectual relationships from the 12 months before their nomination. A majority of the writing group disclosed no relevant, significant financial relationships. Financial and intellectual disclosure information was periodically reviewed by the writing group during document development and updated as needed. SCAI policy requires that writing group members with a current, relevant financial interest are recused from participating in related discussions or voting on recommendations. The work of the writing committee was supported exclusively by the SCAI, a nonprofit medical specialty society, without commercial support. Writing group members contributed to this effort on a volunteer basis and did not receive payment from the SCAI.

Narrative literature searches were performed by group members designated to lead each section, and initial findings were synthesized in section drafts authored primarily by the section leads in collaboration with other members of the writing group. Recommendations were iteratively discussed by the full writing group in a series of virtual consensus meetings until a majority of group members agreed on the text and qualifying remarks. In addition, all recommendations are supported by a short summary of the evidence or specific rationale.

The draft manuscript was peer reviewed in October 2021, and the document was revised to address pertinent comments. The writing group unanimously approved the final recommendations and updated classification. The SCAI Publications Committee and Executive Committee endorsed the document as official society guidance in December 2021.

SCAI statements are primarily intended to help clinicians make decisions about treatment alternatives. Clinicians also must consider the clinical presentation, setting, and preferences of individual patients to make judgments about the optimal approach.

Review of published SCAI SHOCK stage validation studies

Summary of published SCAI SHOCK validation studies

Since the publication of the SCAI SHOCK stage classification in 2019, several groups have produced observational validation studies ranging in size from 166 to 10004 patients that uniformly demonstrate an association between the SCAI SHOCK stage and mortality risk in a variety of

Table 1 Characteristics of studies validating the association between the SCAI SHOCK stage and mortality.

Study	Years included	Population	Design	Patients, n	Primary outcome
Schrage et al 2020[a]	2009-2017	CS or large MI	Retrospective single-center	1007	30-day survival
Baran et al 2020	2019-2020	CS	Prospective single-center	166	30-day survival
Thayer et al 2020	2016-2019	CS	Prospective multicenter[b]	1414	In-hospital mortality
Hanson et al 2020	2016-2019	AMICS	Prospective multicenter[b]	300	Survival to discharge
Jentzer et al 2021[a]	2007-2015	CS	Retrospective single-center	934	30-day survival
Jentzer et al 2019	2007-2015	CICU	Retrospective single-center	10,004	In-hospital mortality
Lawler et al 2021	2017-2019	CICU or CS	Retrospective multicenter	1991	In-hospital mortality
Jentzer et al 2020	2007-2015	CICU survivors	Retrospective single-center	9096	Postdischarge survival
Pareek et al 2020	2012-2017	OHCA	Retrospective single-center	393	30-day mortality

Duplicate data from the same cohort are not shown.
AMICS, CS from acute myocardial infarction; CICU, cardiac intensive care unit; CS, cardiogenic shock; MI, myocardial infarction; OHCA, out-of-hospital cardiac arrest; SCAI, Society for Cardiovascular Angiography and Interventions.
[a] Patients with CS from the Schrage 2020 study were included in the Jentzer 2021 study, so only the nonduplicated patients are reported for the Jentzer 2021 study.
[b] Patient enrollment in these studies was prospective, but the SCAI SHOCK stage was assigned retrospectively.

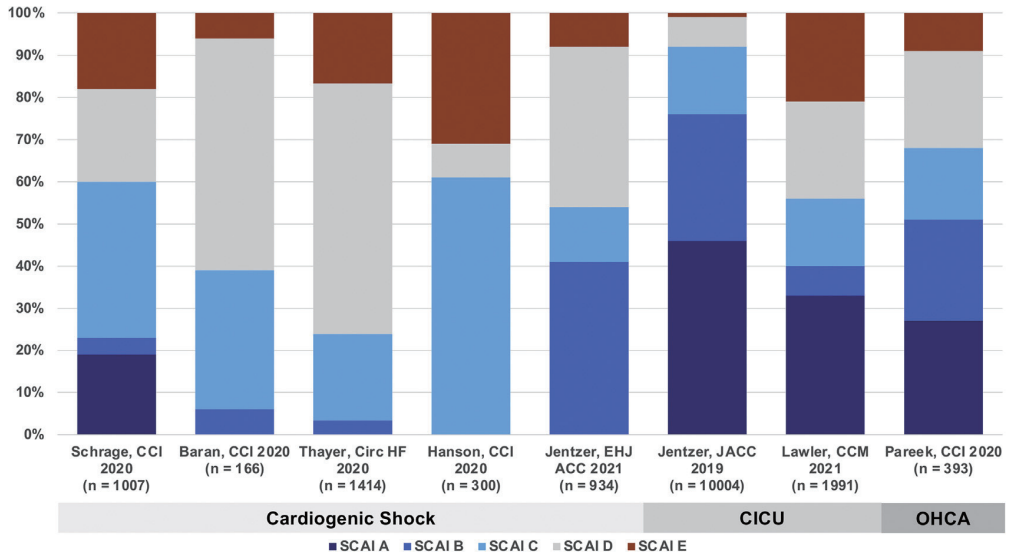

Fig. 1. Distribution of SCAI SHOCK stages in each study. CICU, cardiac intensive care unit; OHCA, out-of-hospital cardiac arrest; SCAI, Society for Cardiovascular Angiography and Interventions.

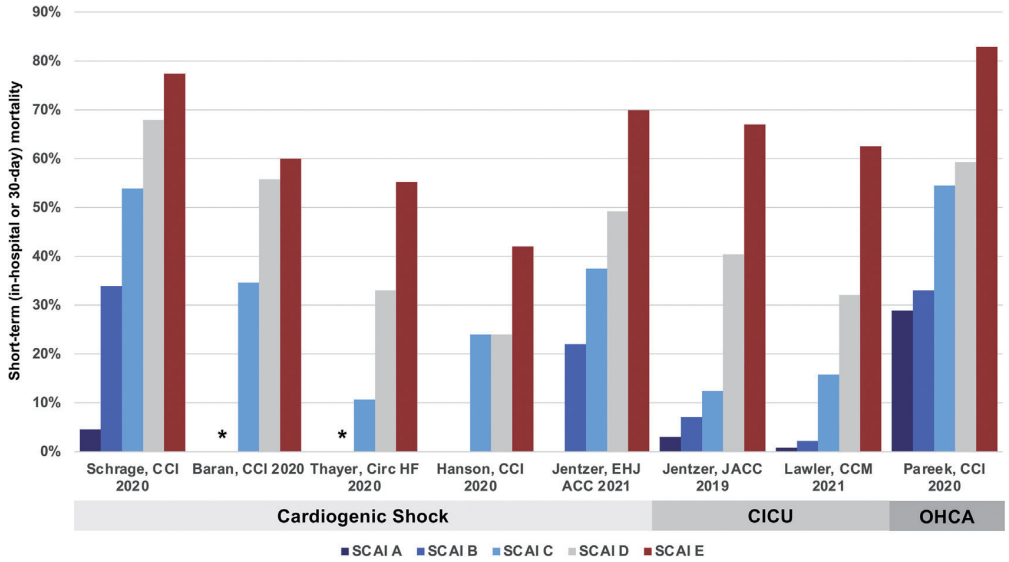

Fig. 2. Short-term mortality as a function of SCAI SHOCK stages in each study. *denotes that no deaths were observed in patients with SCAI stage B in these studies. CICU, cardiac intensive care unit; OHCA, out-of-hospital cardiac arrest; SCAI, Society for Cardiovascular Angiography and Interventions.

populations (Table 1).[1,3-11] Although several studies have focused on patients with CS,[3-7] others have included a broader mix of CICU patients[8-10] or those with OHCA.[11] As expected, the prevalence of each SCAI SHOCK stage varied with the population studied and the definitions used in each study (Fig. 1). The observed short-term (in-hospital or 30-day) mortality also varied depending on the population, and higher SCAI SHOCK stages were consistently associated with higher short- and long-term mortality (Fig. 2).[4,8,11] Furthermore, the SCAI SHOCK stages provided stepwise mortality risk stratification within the subgroups of ACS/acute myocardial infarction (AMI), HF, and those with and without CA.[5,8,11] Most studies classified the SCAI SHOCK stage at a single time point, precluding an analysis of serial changes in stage over time. Importantly, real-time assignment of the SCAI SHOCK stage by the treating team was feasible and allowed for serial assessments.[4] Stratification of mortality risk in the cited studies despite different criteria, populations, and therapies remained consistent, underscoring the strength of the classification scheme.

Variables used to define SCAI SHOCK stages in the validation studies

Each study used different criteria to define the SCAI SHOCK stages (Supplemental Tables S2-S7), including various combinations of clinical variables based on the availability of data.[3-6,8,10,11] Five groups[3,5,8,10,11] developed study-specific SCAI SHOCK stage criteria, whereas two groups[4,6] used physician assessment of the stage without study-specific criteria. Apart from the study by Baran et al which involved real-time prospective assignment of the stage by the treating team, each study assigned the stage retrospectively.

The definitions of the SCAI SHOCK stages used in individual studies range from simple to complex.[5,8] For studies including patients with SCAI SHOCK stage B, this group was defined using vital sign abnormalities (Supplemental Table S4), and there was variability with respect to whether patients receiving vasopressors were classified as SCAI SHOCK stage B or C.[3-5,7,8,10,11] Most studies used elevated lactate levels (≥2 mmol/L) to define hypoperfusion as stage C (Supplemental Table S5); impaired renal function was often used to define hypoperfusion, but few studies distinguished between acute and chronic renal dysfunction. Stage D shock was commonly defined as rising lactate and/or increasing vasopressor or mechanical circulatory support (MCS) requirements (Supplemental Table S6). Definitions of SCAI SHOCK stage E varied (Supplemental Table S7), with criteria including a high lactate level (≥5-10 mmol/L), a low pH (≤7.2), the need for multiple vasopressors/MCS devices, or the need for cardiopulmonary resuscitation (CPR). Despite the importance of physical examination and invasive hemodynamic assessment in defining CS clinically, these variables were not used in most studies because of retrospective data collection. Jentzer et al examined different definitions of shock and preshock in CICU patients and identified that hypoperfusion was associated with mortality to a greater extent than hypotension.[12]

To date, no published study has directly compared the performance of different SCAI SHOCK stage classification schemes in the same population for risk stratification. Importantly, the heterogeneity in mortality in each of the different stages across various studies likely reflects the dissimilar populations and different definitions used; more objective definitions and placing the SCAI SHOCK stage in the context of etiology, phenotype, and other nonmodifiable risk modifiers will help to optimize risk assessment in the future. However, the consistent stratification of risk (using different combinations of variables) suggests that refining and streamlining the criteria for the SCAI SHOCK stage as a categorization of shock severity will facilitate prospective assignment in clinical practice.

SCAI SHOCK validation studies in patients with CS with and without AMI

The National Cardiogenic Shock Initiative reported on 300 patients with CS from AMI (AMICS) and determined the SCAI SHOCK stage by retrospective chart review, assigning the worst shock stage on admission and at 24 hours. The authors found an incremental but strong association between the shock stage and mortality at both time points.[6] Analyses from the Cardiogenic Shock Working Group included a broader group of patients with CS and defined the maximum shock stage during hospitalization, finding a stepwise increase in mortality with a higher shock stage in both patients with AMI and HF.[5] Schrage et al reported on 1007 patients with mixed etiologies of CS and demonstrated mortality risk stratification across the shock stages (including at-risk patients with large AMI).[3] Patients with CS from this cohort were combined with patients with CS from the Mayo Clinic cohort and reported similar findings.[7] Baran et al reported the first prospective validation study in patients with CS by having the treating physician assign the SCAI SHOCK stage in real time based on available clinical data.[4] The studies by Hanson et al and Baran et al demonstrated that a rising or persistently elevated SCAI SHOCK stage was associated with substantially worse outcomes.[4,6]

SCAI SHOCK validation studies in CICU and OHCA patients

Jentzer et al first validated the SCAI SHOCK stages using data from 10004 consecutive CICU patients at the Mayo Clinic, finding that each higher stage was associated with an incrementally higher risk of in-hospital mortality, even after adjustment for known predictors of mortality.[8] Hospital survivors with a higher SCAI SHOCK stage on admission had increased postdischarge mortality.[9] Patients with CA had a higher risk of dying at each SCAI SHOCK stage; both the location in which CA occurred (in-hospital versus out-of-hospital) and the rhythm of CA affected the risk of mortality.[13] A subsequent multicenter study from the CCCTN database in 1991 CICU patients with ACS or HF also demonstrated that the SCAI SHOCK stage was associated with in-hospital mortality; a diagnosis of CS was required for patients in SCAI stages C, D, and E.[10] In a distinct cohort of 393 OHCA patients, Pareek et al found that the observed short-term mortality was higher at each SCAI SHOCK stage than that in other studies, with clear mortality risk stratification as per shock stage.[11]

Studies examining risk modifiers within the SCAI SHOCK stage classification

The SCAI SHOCK stage classification has been leveraged to examine other aspects of mortality risk stratification across the spectrum of shock severity (Table 2). In the Mayo Clinic CICU cohort, age, the presence of systemic inflammatory response syndrome, acute kidney injury, and other noncardiac organ failure, severe acidosis, and echocardiographic findings were found to improve mortality risk stratification beyond SCAI SHOCK stages alone.[14-18] The importance of age as a risk factor for mortality, independent of shock stage, was likewise reported in CS cohorts.[4,7] Thayer et al and Garan et al showed the importance of pulmonary artery catheter use, congestion profile, and invasive hemodynamic data (particularly an elevated right atrial pressure) as risk modifiers independent of the shock stage in patients with CS.[5,19] Worsening shock, either defined by rising shock stage over time or late deterioration, has been consistently associated with higher

Table 2 Studies examining potential risk modifiers on top of the SCAI SHOCK stages for mortality risk stratification.

Study	Population	Design	Patients, n	Variable of interest	Conclusions
Jentzer et al 2019	CICU	Retrospective single-center	10,004	CA	CA and late deterioration were associated with higher mortality
Baran et al 2020	CS	Prospective single-center	166	Change in the SCAI stage	An increasing SCAI stage is associated with higher mortality
Garan et al 2020	CS	Prospective multicenter	1414	Invasive hemodynamics	Higher mortality with higher RAP and HR or lower MAP, lower with PAC
Hanson et al 2020	AMICS	Prospective multicenter	300	Change in the SCAI stage	An increasing SCAI stage is associated with higher mortality
Jentzer et al 2020	CICU	Retrospective single-center	9898	CA type	Non-VF CA is associated with higher mortality
Jentzer et al 2020	CICU	Retrospective single-center	8995	SIRS on admission	SIRS is associated with higher mortality
Padkins et al 2020	CICU	Retrospective single-center	10,004	Age	Higher age is associated with higher mortality
Thayer et al 2020	CS	Prospective multicenter	1414	Invasive hemodynamics	Higher RAP is associated with higher mortality
Jentzer et al 2021	CS	Retrospective multicenter	1749	Age	Higher age is associated with lower survival
Jentzer et al 2021	CICU	Retrospective single-center	5453	Echo hemodynamics	Low SVI and high E/e' are associated with higher mortality
Jentzer et al 2021	CICU	Retrospective single-center	9311	AKI during hospitalization	Worse AKI is associated with higher mortality across SCAI stages
Jentzer et al 2021	CICU	Retrospective single-center	1065	Severe acidosis	Severe acidosis associated with higher mortality across SCAI stages
Zweck et al 2021	CS	Prospective multicenter	1959	Biochemical phenotype	"Cardiometabolic" phenotype associated with higher mortality

Several of these study populations overlap with those presented in Table 1.
AKI, acute kidney injury; AMICS, CS from acute myocardial infarction; CA, cardiac arrest; CICU, cardiac intensive care unit; CS, cardiogenic shock; HR, heart rate; MAP, mean arterial pressure; PAC, pulmonary artery catheter; RAP, right atrial pressure; SCAI, Society for Cardiovascular Angiography and Interventions; SIRS, systemic inflammatory response syndrome; VF, ventricular fibrillation.

mortality.[4,6,8,9] Jentzer et al demonstrated that an increasing number of abnormal markers of hypotension and hypoperfusion was associated with incrementally higher mortality risk in CICU patients; an elevated lactate level or an elevated shock index (the ratio of heart rate to systolic blood pressure) was more strongly associated with mortality.[12] Biochemical phenotypes were identified in a large multicenter registry of patients with CS, highlighting the variability in observed mortality with the different phenotypes across the shock stages.[20] Finally, SCAI SHOCK stages have been used to evaluate the association between certain treatments and outcomes in patients with CS.[21]

Collectively, these studies have demonstrated that higher-risk and lower-risk subgroups exist within each SCAI SHOCK stage, and a higher-risk subgroup within a lower SCAI SHOCK stage might have a mortality risk that exceeds a lower-risk subgroup within a higher SCAI SHOCK stage. Clearly, shock severity assessment is a central component of overall mortality risk stratification in patients with CS, yet other clinical variables modify the predicted mortality risk.[22] Established CS-specific mortality risk prediction scores combine lactate and renal function (markers of hypoperfusion and shock severity) with patient-level variables to provide mortality risk stratification and should be considered distinct from shock severity classification algorithms.[23,24] Recently, a newer CS-specific mortality risk prediction score based on biomarkers has been presented, suggesting that such biomarkers may be integrated into future risk assessment strategies.[25]

Lessons learned from the SCAI SHOCK validation studies

Shortcomings of original classification

The original SCAI SHOCK classification

In brief, SCAI SHOCK stage A is broad and represents the myriad of stable patients who have acute cardiac diagnoses that place them at risk for CS but fail to meet the criteria for preshock (stage B) or shock (stages C-E). Stage B represents patients who have intact systemic perfusion with evidence of hemodynamic instability, such as hypotension or compensatory tachycardia; patients with preserved perfusion despite significantly abnormal invasive hemodynamics (such as reduced cardiac output) are also classified as SCAI stage B. Stage C represents the more classic patients with CS who present with hypoperfusion either untreated or requiring hemodynamic support through pharmacologic or mechanical intervention. Stage D represents the failure of an adequate trial of an initial supportive intervention and, therefore, captures a different shock state than stage C, requiring some element of time. Stage E is reserved for refractory shock with actual or impending cardiovascular collapse despite high and escalating levels of support (including arrest-in-progress). In the opinion of the writing group, SCAI SHOCK stage E is usually a transient state that is easy to recognize in clinical practice (typically a peri-code situation or need to rapidly escalate hemodynamic support) but can be difficult to define retrospectively for the purpose of research.

Retrospective vs real-time classification

The original SCAI SHOCK stage classification was designed to be simple, recognizing that complete clinical information for grading shock severity is not always available. Physical examination, laboratory, and hemodynamic findings were provided to guide the clinician in assigning a specific SCAI SHOCK stage, in the order in which these variables are typically available, while allowing flexibility for application in different care environments. This approach works well for teaching the classification system and for applying it prospectively, but it presents challenges when applying it retrospectively to existing data sets with missing or inappropriately timed data. As a result, the assignment of the SCAI SHOCK stage in published studies has contributed to heterogeneity across studies, and future studies should ideally use consistent staging criteria.

Differentiating preshock (stage B) vs classic shock (stage C)

The distinction between SCAI SHOCK stage B (hemodynamic instability with preserved perfusion, ie, preshock) and SCAI SHOCK stage C (hypoperfusion with or without overt hemodynamic instability, ie, classic shock) is critical and requires the integration of multiple clinical and laboratory data points. Patients with hypoperfusion in the absence of hypotension are at higher risk of dying than patients with hypotension and preserved perfusion.[12] Biomarkers such as lactate are commonly used to detect hypoperfusion but may be dissociated from hemodynamics in cases such as chronic HF where patients may have a normal lactate level with a depressed cardiac index. The authors suggest that a lactate level of >2 mmol/L is consistent with at least SCAI SHOCK stage C, although some patients may demonstrate other manifestations of end-organ hypoperfusion with a normal lactate level, and there are also causes of an elevated lactate level other than shock, such as mesenteric ischemia or compartment syndrome.

Shock classification based on required therapeutic interventions: differentiating stages C and D

As described by the National Cardiogenic Shock Initiative and Cardiogenic Shock Working Group, the intensity of therapies required to achieve hemodynamic stability and restore systemic perfusion can be used to define the SCAI SHOCK stage, but this approach will be most informative when the same escalation strategy is used by clinicians (such as with an institutional CS protocol) given practice variability in MCS patient selection and implantation.[26,27] We suggest that if a patient requires vasoactive drugs or MCS to reverse hypoperfusion or hemodynamic compromise, they should be assigned SCAI SHOCK stage C. If this initial therapy is ineffective, evidenced by the need to add one or more additional vasoactive drugs or MCS devices, then SCAI SHOCK stage D is present. If perfusion cannot be restored using multiple vasoactive drugs and/or MCS devices, or if extremely high vasoactive drug doses are required, then SCAI SHOCK stage E is present. An important limitation of this approach is the variability in vasoactive drug dosing which affects the prognosis. For example, a patient who has stabilized on low doses of two vasoactive drugs may be classified as stage C, whereas a patient who is failing a high dose of a single vasoactive drug might be classified as stage D. Differentiating the CS stage and prognosis based on dose escalation as opposed to additional pharmacotherapy or mechanical support will require further data.

Cardiac arrest modifier clarification

Another area of controversy with the original classification is the "A" modifier representing an episode of CA. Clearly CA events are heterogeneous, and single defibrillation for a brief ventricular arrhythmia without CPR and normal neurologic function does not change the prognosis of a patient with CS.[7] Instead, the two aspects of greatest relevance are the neurologic status (awake or comatose) and physiologic impact of the arrest, as prolonged CA may fundamentally change the patient trajectory if ischemia-reperfusion heralds multiorgan failure. At this time, there is no clearly defined CPR duration that would qualify a patient for the "A" modifier, and we believe that the "A" modifier should refer to patients with potential anoxic brain injury. This may be evidenced by a decreased Glasgow Coma Scale, where a value less than 9 typically defines coma; alternatively, the absence of a motor response to voice (ie, not following commands) is a useful definition.

Whether to include age as a modifier

One of the strongest findings from multiple studies is the effect of increasing age on mortality. Age is a well-known continuous risk factor

for adverse outcomes in patients with CS and was highlighted in the seminal shock trial, the IABP-SHOCK II trial, and subsequent risk scores.[23,24] The ability to overcome the stress of CS declines with advancing age, irrespective of other comorbidities. Older patients are more likely to die, and this added risk is likely to guide clinicians in determining candidacy for specific therapies for CS at each SCAI SHOCK stage.[7,16] A significant challenge with using age as a modifier is that as a continuous variable there is no clearly defined binary threshold of risk. Overall, age functions more as a marker of comorbidities, frailty, and candidacy for (or futility of) therapeutic interventions. Therefore, although age is clearly a major risk factor for adverse outcomes that modifies risk across the SCAI SHOCK stages and should be taken into account by clinicians, too much uncertainty exists regarding how best to apply this prognostic information to incorporate age directly into the SCAI SHOCK classification.

Reasons to maintain a similar classification framework

Although there are reasons to modify the SCAI SHOCK classification as detailed previously, there are also reasons to avoid unnecessarily complex revisions that would make the classification more difficult to use and its distribution globally less rapid. Most importantly, it is simple for multidisciplinary teams to use the existing SCAI SHOCK schema across the spectrum of care in a prospective fashion. Indeed, real-world patient care reflects the fact that not all patients will have the comprehensive information that may be included in a more complex risk score or on a clinical research flow sheet. Moreover, the work of Baran et al shows that real-time prospective assignment of the SCAI SHOCK stage by a team achieves the same predictive value including mortality observed in several retrospective validation studies using complex criteria.[4] Maintaining simplicity and flexibility will therefore allow the SCAI SHOCK stage classification to be used by clinicians with expertise in critical care medicine and emergency medicine, including prehospital clinicians, without losing significant prognostic impact. A system analogous to that used for STEMI may allow patients with shock to be selectively sent to "shock centers", facilitated by a simplified prehospital SCAI SHOCK classification that relies on physical examination alone.[28,29]

Key aspects to emphasize in an updated classification

It is crucial to emphasize the distinction between the grading of shock severity and the prediction of mortality risk. Although shock severity is among the most potent predictors of mortality in patients with CS, numerous other risk modifiers can influence this risk, resulting in lower-risk and higher-risk patients at each SCAI SHOCK stage, as highlighted previously. In addition, the transition between stages is of significant prognostic value. Although the SCAI SHOCK stage can provide mortality risk stratification (particularly when risk modifiers are integrated), its greatest value is in standardization of shock severity assessment to enhance clinical communication and decision-making. In addition, re-evaluation of the clinical stage can guide further treatment options regarding escalation or de-escalation strategies and assist in prognosis. The SCAI SHOCK stage should be reassessed at intervals, the timing of which will differ based on the initial severity and response to therapy. The improvement of the SCAI SHOCK stage by even one category is a powerful favorable prognostic indicator, and conversely, a maintaining or declining SCAI SHOCK is a potent negative marker.[6] Similarly, CA that results in neurologic injury or impacts peripheral organ function is an important concern that impacts mortality and the potential for recovery. Finally, the consideration of age along with the SCAI SHOCK stage is of value to the clinician while planning the next intervention, including the recognition of futility before care is rendered.[28]

To improve care, it will be important to recognize that most sites are not equipped with all modalities for the care of CS, and therefore, some patients will need to be transferred to a primary shock center or "hub" that has the ability and technology to care for all patients.[28,29] However, given capacity constraints, it is important to have a classification system that allows identification of the sickest patients, but also ones where the possibility of survival is greatest; this is where an understanding of the distinction between shock severity and mortality risk is essential, and risk modifiers must be taken into account. Transfers for futile care in unrecoverable high-risk patients do not change outcomes and deny capacity to those who might otherwise benefit. One proposal would be for sites to classify their capabilities and organize into spokes and hubs. The spoke centers with MCS capabilities would manage SCAI SHOCK stage C (most patients) but are triggered to consider referral when progression to SCAI SHOCK stage D occurs (before development of SCAI SHOCK stage E). However, patient candidacy for advanced supportive therapies should always be a central consideration in these decisions, adding a layer of complexity.

Patients with CS represent a heterogeneous population including distinct phenotypes, which are challenging to define and may be independent from shock severity per se.[20] Clinicians must recognize that patients at each SCAI SHOCK stage may appear or behave differently and may present with a spectrum of overall illness severity and mortality risk. Clinical decision-making for patients with CS must integrate not only shock severity but also the etiology of shock (particularly ischemic versus non-ischemic and acute versus acute-on-chronic), the presence and reversibility of organ failure, degree of congestion, mixed or vasodilatory shock states, ventricular involvement (LV, RV, or biventricular dysfunction), and a multitude of factors influencing candidacy for supportive therapies such as age, CA, and important comorbidities. Therefore, using the SCAI SHOCK stages to provide a uniform assessment of shock severity is merely one important component of prognostication and management for these patients, and we believe that a consistent classification system that can be tailored to each care environment is more useful than a comprehensive one.

Updated SCAI SHOCK classification pyramid and table

Framework and criteria for shock stages

The framework emphasizing the domains of physical examination, biochemical, and hemodynamic criteria has been maintained, representing the availability of better data over time that can and should be integrated into assigning the SCAI SHOCK stage. Suggested criteria in each domain to define the SCAI SHOCK stages (Table 3) have been modified to be more succinct and data-driven, with the goal of optimizing sensitivity and specificity to enable increased incorporation into clinical practice. This remains a work in progress that is designed to be flexible and will continue to be refined as more and better data become available. Lactate thresholds have been modified to reflect the available data; although not all studies measured lactate levels routinely, this is important both therapeutically and prognostically and should be adopted as a standard practice going forward.

Table 3 has been modified to characterize diagnostic features as those that are typically included and those that may be included when defining the SCAI SHOCK stage. This was carried out to account for variability in patient presentations and in recognition that the data available to the clinician vary between different care settings, both within a given institution and among different institutions. For example, invasive hemodynamic data are obtainable in the catheterization laboratory and potentially in the critical care unit but are not generally available in the emergency department or the rural community hospital. As a patient moves through the health care system from the first medical contact to the more advanced hospital settings, the quantity and quality of data that become available will increase and the SCAI SHOCK stage assignment will be more accurate.

Clarification of SCAI SHOCK stage D, which is defined as failure to stabilize with initial therapy, may be helpful. In general, the need for more than one vasoactive agent or more than one support device, due to failure of appropriate initial therapy to maintain perfusion, defines a patient in stage D. In addition, either escalating doses of medications or need for higher mechanical support settings over time may represent stage D. As such, patients who need more than one vasoactive agent

Table 3 Descriptors of shock stages: Physical examination, biochemical markers, and hemodynamics.

Stage	Description	Physical examination/bedside findings		Biochemical markers		Hemodynamics	
		Typically includes	May include	Typically includes	May include	Typically includes	May include
A At risk	A patient who is **not currently experiencing signs or symptoms of CS, but is at risk for its development**. These patients may include those with large acute myocardial infarction or prior infarction and/or acute or acute-on-chronic heart failure symptoms.	**Normal JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Clear lung sounds	**Normal lactate**	Normal labs • Normal (or at baseline) renal function	**Normotensive** (SBP ≥100 mmHg or at baseline)	If invasive hemodynamics are assessed: • Cardiac Index ≥2.5 L/min/m^2 (if acute) • CVP ≤10 mmHg • PCWP ≤15 mmHg • PA saturation ≥65%
B Beginning CS	A patient who has **clinical evidence of hemodynamic instability** (including relative hypotension or tachycardia) **without hypoperfusion**.	**Elevated JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Rales in lung fields	**Normal lactate**	Minimal acute renal function impairment Elevated BNP	**Hypotension** • SBP <90 mmHg • MAP <60 mmHg • > 30 mmHg drop from baseline **Tachycardia** • Heart rate ≥100 bpm	
C Classic CS	A patient who manifests with **hypoperfusion and who requires one intervention (pharmacological or mechanical) beyond volume resuscitation**. These patients typically present with relative hypotension (but hypotension is not required).	**Volume overload**	Looks unwell Acute alteration in mental status Feeling of impending doom Cold and clammy Extensive rales Ashen, mottled, dusky, or cool extremities Delayed capillary refill Urine Output <30 mL/h	**Lactate ≥2 mmol/L**	Creatinine increase to 1.5 x baseline (or 0.3 mg/dL) or > 50% drop in GFR Increased LFTs Elevated BNP	If invasive hemodynamics assessed (**strongly recommended**) • Cardiac index <2.2 L/min/m^2 • PCWP >15 mmHg	
D Deteriorating	A patient who is similar to category C but is getting worse. **Failure of initial support strategy to restore perfusion** as evidenced by worsening hemodynamics or rising lactate.	**Any of stage C and worsening (or not improving) signs/ symptoms of hypoperfusion despite the initial therapy.**		**Any of stage C and lactate rising and persistently >2 mmol/L**	Deteriorating renal function Worsening LFTs Rising BNP	**Any of stage C and requiring escalating doses or increasing numbers of pressors or addition of a mechanical circulatory support device to maintain perfusion**	
E Extremis	**Actual or impending circulatory collapse**	**Typically unconscious**	Near pulselessness Cardiac collapse Multiple defibrillations	**Lactate ≥8 mmol/L**[a]	CPR (A-modifier) Severe acidosis • pH < 7.2 • Base deficit >10 mEq/L	**Profound hypotension despite maximal hemodynamic support**	Need for bolus doses of vasopressors

BNP, B-type natriuretic peptide; CPR, cardiopulmonary resuscitation; CVP, central venous pressure; GFR, glomerular filtration rate; JVP, jugular venous pressure; LFT, liver function tests; MAP, mean arterial pressure; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; SVP, systolic ventricular pressure.

[a] Stage E prospectively is a patient with cardiovascular collapse or ongoing CPR.

shortly after presentation can be in stage C, but an element of time must pass after initiating therapy to define stage D. Stage D may be the most challenging to define for clinical practice and research because of uncertainty about what constitutes an adequate therapeutic trial in terms of vasoactive drug dosing, device selection, and time.

The CA modifier has been refined to include only those individuals after CA who fail to respond to verbal commands and/or who have a Glasgow Coma Scale of <9 and no longer includes brief CA with normalization of neurologic status.

SCAI SHOCK stage in the context of acuity of presentation, etiology, phenotype, and other risk modifiers—the 3-axis model

Acute versus acute-on-chronic presentation and shock etiology

The SCAI SHOCK classification was designed for patients presenting acutely, but acute and acute-on-chronic processes can differ in important ways. Patients with decompensation in the context of chronic HF may present with different symptomatology and may also have different hemodynamic profiles in that they may have developed adaptations to allow them to tolerate lower cardiac output and blood pressure.[30] Indeed, because of compensatory mechanisms and adaptations, patients with chronic HF may display a lower SCAI SHOCK stage than those without such adaptive mechanisms or may provide a falsely reassuring clinical picture despite high-risk hemodynamics.[26] Accordingly, it is critical to interpret physical findings and hemodynamics in this clinical context. That said, these differences are most evident in patients in SCAI SHOCK stages A and B and converge in later stages. SCAI SHOCK stages C, D, and E tend to appear similar regardless of underlying chronicity. Another crucial distinction relates to the etiology of shock which may influence the clinical presentation and outcomes, such as patients with AMICS versus decompensated HF progressing to CS. Although the SCAI SHOCK classification applies equally to both groups of patients, their clinical and hemodynamic findings, prognosis, and optimal treatment strategies may differ markedly.

Shock phenotype

Although hemodynamic measurements are commonly used to make the diagnosis of CS, formal definitions of hemodynamic shock phenotypes may help guide therapy and improve outcomes. The hemodynamic parameters shown in Table 3 generally define the diagnosis of shock with low cardiac output or cardiac power output, high filling pressures, and increased oxygen extraction (ie, reduced venous oxygen saturation) indicative of systemic perfusion failure overall despite adequate volume. Other hemodynamic parameters, such as the ratio of right atrial to pulmonary capillary wedge pressure (right atrial pressure/pulmonary capillary wedge pressure),[28] and pulmonary artery pulsatility index[31,32] among others are now advised to identify patients with RV failure who may potentially require dedicated RV or biventricular support.[28,29] These hemodynamic and echocardiographic measurements reflecting ventricular function may facilitate risk stratification within the SCAI SHOCK stage classification. Recent application of machine learning algorithms supports that distinct phenotypes of CS can be identified and further stratifies mortality risk within each SCAI stage.[20] It is essential to differentiate between diagnostic variables that enable assignment of the SCAI SHOCK stage from prognostic variables that help predict mortality risk or assign the structural problem leading to shock, recognizing that many variables serve both purposes.

The 3-axis model of predictors of mortality

The outcome of shock is based on a number of factors, including the severity of the shock, risk modifiers such as age, comorbidities, and prior CA with evidence of anoxic encephalopathy, and certain features of the hemodynamic phenotype and clinical presentation. We propose a 3-axis model of CS evaluation and prognostication that integrates shock severity, clinical phenotype, and risk modifiers as distinct constructs that must be considered during clinical decision-making (Fig. 3). Established and emerging biomarkers may further refine risk stratification, and future research will be needed to define how to integrate these into shock severity assessment. Many of these factors are captured in the SCAI SHOCK classification, but others are not, underscoring the importance of evaluating individual parameters in the context of the entire clinical picture. It is essential to differentiate a patient who is "high risk" due to severe shock with poor hemodynamics from a patient who is "high risk" due to nonmodifiable risk factors for mortality.

Revised SCAI SHOCK pyramid

A revision of the SCAI SHOCK pyramid is shown in Figure 4. The underlying structure is the same to prioritize simplicity and widespread applicability. Each of the stages now has gradients of color to denote

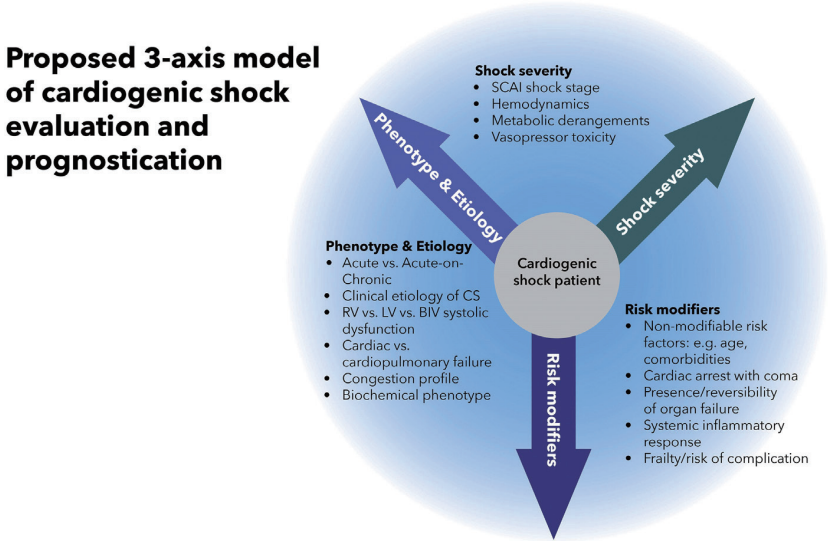

Fig. 3. The proposed 3-axis conceptual model of cardiogenic shock evaluation and prognostication.

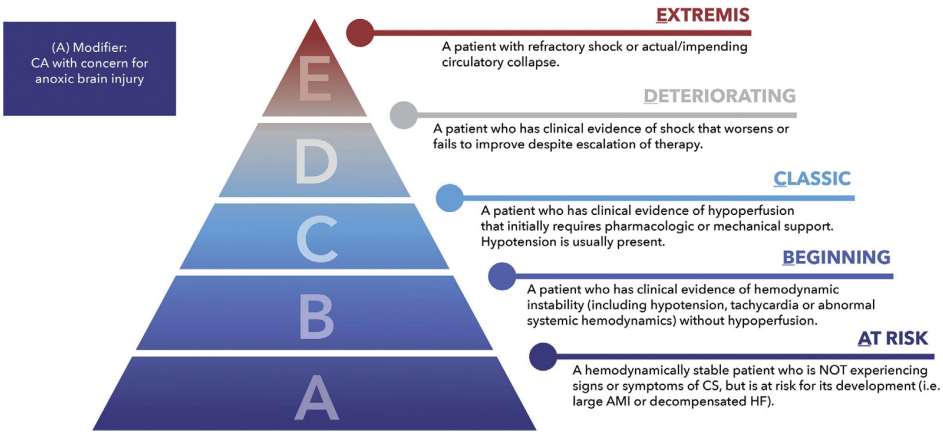

Fig. 4. Updated SCAI SHOCK classification pyramid. AMI, acute myocardial infarction; CS, cardiogenic shock; HF, heart failure; SCAI, Society for Cardiovascular Angiography and Interventions.

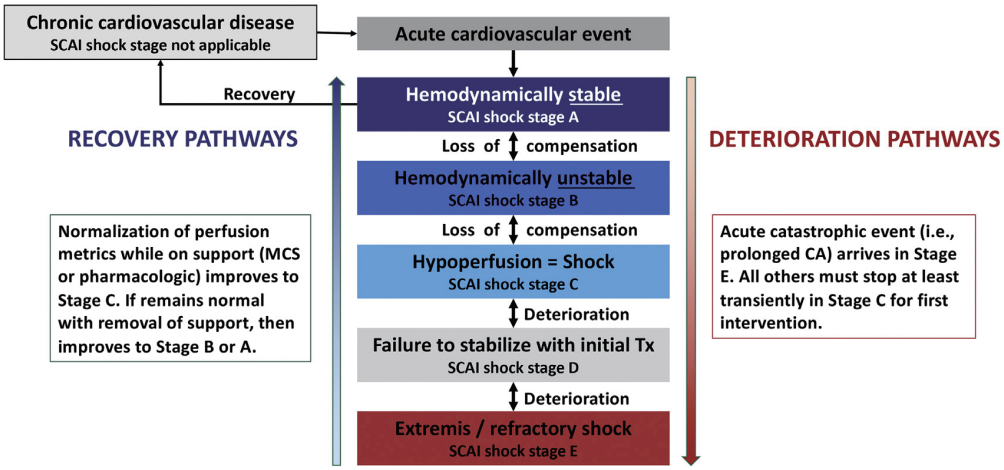

Fig. 5. Cardiogenic shock is a dynamic process. CA, cardiac arrest; MCS, mechanical circulatory support; SCAI, Society for Cardiovascular Angiography and Interventions.

gradations of shock severity and risk within each stage, as a reminder of the need to individualize patient care based on phenotype, risk modifiers, and comorbidities. We explicitly did not add subcategories within each SCAI SHOCK stage to preserve the simplicity of the classification. The updated CA modifier is incorporated into the pyramid.

Classification and patient trajectories

Figure 5 shows the initial and reclassification process in response to patient response and trajectory. It should be noted that the SCAI SHOCK classification only applies to acute presentations and is not used to stage chronic cardiovascular disease. Patients may respond to therapy, stabilize, and recover, in which case they would move to a progressively lower SCAI SHOCK stage. Alternatively, they may fail to respond to therapy, deteriorate, or experience an acute catastrophic event such as CA or myocardial rupture, in which case they would move to a higher stage. In addition, response failure includes not only patients who are getting worse but also those who are failing to improve with appropriate therapy. Note that decompensation into SCAI SHOCK stage D requires spending some time in SCAI SHOCK stage C because an intervention and an element of time are required, whereas a catastrophic event or decompensation may result in SCAI SHOCK stage E from any of the lower stages.

Summary of the new classification

The revision of the SCAI SHOCK stage classification moves toward eliminating variables that are either redundant or that have been shown not to add additional prognostic value in the interest of making the classification simpler to use and more data-driven. This process is ongoing and will be refined as high-quality data accumulate. Some of the elements are defined with greater precision, including lactate levels and also the CA modifier, which now excludes very brief episodes with rapid response to defibrillation and comprises only those patients who have impaired mental status with unknown neurologic recovery status after CPR.[28,29,33]

The classification tends to err on the side of being practical and simple over being comprehensive and is most applicable to acute presentations with CS. However, we have now outlined a 3-axis model for evaluation and prognostication that takes into account shock severity, risk modifiers, etiology, and phenotypes that should be applied to individualize patient management. The revised pyramid has gradations of color to represent gradations of risk within each stage.

Finally, additional emphasis has been placed on patient trajectories, to help recognize patients who are responding to therapy but more importantly to identify those who are failing to respond or deteriorating and who should be considered for more intensive therapy (or

interhospital transfer) or conversely considered for palliation based on patient and family wishes or futility.

Our hope is that the revised criteria will allow for more uniform classification to help clinicians choose patients for advanced therapies, but also define criteria for entry into clinical trials to better understand the value of potential therapies. A crucial next step in this field will be to compare the outcomes associated with drug and device therapies, systems of care, and treatment protocols for patients at different stages or trajectories, phenotypes, and modifiers of shock.

Future considerations and research

The clinical uptake and scientific confirmation of the SCAI SHOCK stage classification framework, as outlined in section 1 of this document, have been rapid; however, ongoing validation, refinement, knowledge translation, and implementation are required. The staging system has yet to be evaluated in all clinical environments throughout the CS spectrum of care including in the prehospital setting, in the emergency department, or among patients treated with durable MCS or postcardiotomy shock. Moreover, the SCAI SHOCK model was designed to be applied dynamically throughout all phases of care, and more work is required to understand the optimal reassessment intervals and the association between mortality risk and temporal changes in SCAI SHOCK stages from presentation through deterioration and recovery, destination therapy, or palliation.[1]

A major limitation of the current system is that multiple elements within the staging remain subject to variable interpretation including differential threshold for MCS deployment between institutions, necessitating unified definitions of each SCAI SHOCK stage that are less dependent on local practice patterns. The CA modifier continues to include a heterogeneous population with variable risk of neurologic injury; thus, improved collection of intra-arrest information such as arrest duration, rhythm, and treatment could facilitate hypoxic-ischemic neurologic injury discrimination and could refine or improve this "A" modifier.[34] Similarly, the development of uniform definitions of hypoperfusion, hypotension, and LV, RV, or biventricular failure has the potential to improve interuser reliability of shock staging. It remains unclear how best to utilize invasive hemodynamic parameters, laboratory measures of hypoperfusion, biomarkers, or a combination thereof to discriminate the risk of morbidity and mortality. The framework for defining the SCAI SHOCK stages described in this document may be inadequate to directly use without modification for clinical trial enrollment, and precise individualized research definitions of the SCAI SHOCK stages will be required if stratification by stage is desired based on the target population.

Strategies to improve clinical dissemination of this model and uptake among frontline health care workers potentially include incorporation into international societal clinical practice guidelines, embedding the score within institutional electronic health records, and increasing education though traditional scientific (eg, congress presentation, journal clubs) and emerging educational streams (eg, social media awareness, podcasts, and SCAI SHOCK stage calculators). We believe that CS registries and clinical trials could be improved by including the SCAI SHOCK stage classification system as a risk marker of acuity, as a study inclusion/exclusion criterion, and/or by stratifying therapeutic interventions across SCAI SHOCK stages. This could potentially allow for a better understanding of the baseline risk of each population, facilitate interstudy comparisons of CS populations which traditionally pooled this group of patients, and allow for the evaluation of the efficacy and safety of treatments across the severity spectrum. We acknowledge, however, that these prospective strategies require uniform definitions of all variables to allow for accurate SCAI SHOCK staging and good interuser reliability.

Summary and conclusion

In summary, since 2019, the SCAI SHOCK stage classification has been widely adopted and subsequently validated by multiple groups across the spectrum of CS. The SCAI SHOCK consensus workgroup reviewed the validation studies in detail to identify potential areas of refinement for the classification scheme. In particular, we clarified the precise role of the SCAI SHOCK classification within a more comprehensive 3-axis model incorporating predictors of mortality, provided more granularity to the CA modifier and the constituent domains of the classification, including physical examination, biochemical, and hemodynamic criteria, and allowed for gradations of risk within each SCAI SHOCK stage. More emphasis is placed on the trajectory of the patient with CS through hospitalization, including as patients are transferred to higher levels of care (hubs and spokes), as well as potential future directions. It is our desire and belief that the revised SCAI SHOCK stage classification will enhance both clinical care and CS research trial design.

Declaration of competing interest

Disclosure information for all authors is available in Supplemental Table S1.

Supplementary material

To access the supplementary material accompanying this article, visit the online version of the *Journal of the Society for Cardiovascular Angiography & Interventions* at https://doi.org/10.1016/j.jscai.2021.100008.

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簡介 SCAI 2019 年心因性休克分級系統（含 2022 年專家共識更新）

編譯：台中榮總嘉義分院 心臟內科 林俊呈醫師

時空背景

臨床心臟病有不少的分級、分期、或分類系統，如心衰竭有美國紐約心臟學會 NYHA 的 Functional Class 功能分級系統、及美國心臟學會 ACC/AHA Stage 分期系統、狹心症胸悶症狀等級有加拿大心血管學會的分級系統 Canadian Cardiovascular Society Angina Classification，這些系統有助於臨床醫師用統一的方式來描述心臟病人的病情及作為各種研究論文的共同語言，那麼在心臟疾病光譜最嚴重一端的心因性休克 Cardiogenic Shock（CS），有沒有類似的分級系統呢？CS 病患族群異質性很大，預後可能因疾病嚴重度、病因、是否有合併症等而有顯著不同，涵蓋範圍從單純心肌收縮功能障礙導致 CS 的高風險患者、到合併多重器官衰竭及血流動力學障礙的重症患者、或猝死 Cardiac Arrest（CA），根據不同族群及病因，治療也可能不同。尤其在過去 30 年間，急性心肌梗塞（AMI）合併 CS 病人，即使有各種侵入性機械式循環支持系統（Percutaneous Mechanical Circulatory Devices, MCS）輔助，預後還是極差的背景下，一個細緻的心因性休克嚴重度分級系統更是需要。

歷經了幾年的專家共識討論，在 2019 年 5 月，美國心血管造影和介入學會（Society of Cardiovascular Angiography and Interventions, SCAI）發佈了心因性休克分級分類系統（Stage Classification）專家共識聲明[1]（Expert Consensus），這分級分類系統由臨床觀點出發，簡單實用，因此得到美國心臟病學會 ACC、美國心臟學會 AHA、美國重症醫學會 SCCM、以及美國胸外科醫學會 STS 的廣泛支持。在共識發表的兩年期間，有不少驗證研究（Validation）發表，證實該共識的可行性，也包括可以改善的論述。基於這些驗證研究，SCAI 於 2022 年初發表專家共識更新[2]，也加入了歐洲心臟學會 ESC、ACVC、ISHLT、ACEP 等學會支持。

這個分級分類系統分五級，從 Stage A 到 E，考慮到患者的多樣性，希望能包含的角度從介入性心臟科起，還包括嚴重（Advanced）心衰竭、非侵入性心臟學、急診、重症醫學、護理等角度均能一體適用，亦即不管病人是在院外急救場域、急診室、導管室、心臟加護病房或等均能快速應用上手，因此希望可以達到以下要求：要夠簡單（Simple）不需要計算、彈性大不需要收集到所有參數方能分析、快速、在病人邊（Bedside）即可評估、可以隨時重新評估分級、可以回溯分析、可以跨團隊、跨科別、跨醫院、跨系統以適應不同環境、有評估預後的潛力、容易記憶等。這樣從到院前、急診室、導管室、CCU、轉院等均能連續性、即時性評估，且具有一致性。也因為 SCAI SHOCK Stage 具有即時及連續性特性，隨時可以加入新資訊動態更新，對於下一階臨床工作及決策有其重要性。

SCAI CS Stage 介紹

SCAI CS 分級為金字塔設置，奠基於理學檢查、實驗室生化數值及生物標記、及血流動力學等相關指標（Figure 1 and Table 1），這五級包括每個階段的嚴重度、患者進展或康復的途徑，2019 年金字塔版本沒有明顯顏色變化，但 2022 年版本加入顏色變化以標示嚴重程度和風險等級，提醒需要根據病人表現型（Phenotype）、風險修飾因子（Risk Modifiers）、和共病（Co-morbidities）對患者進行個人化醫療。

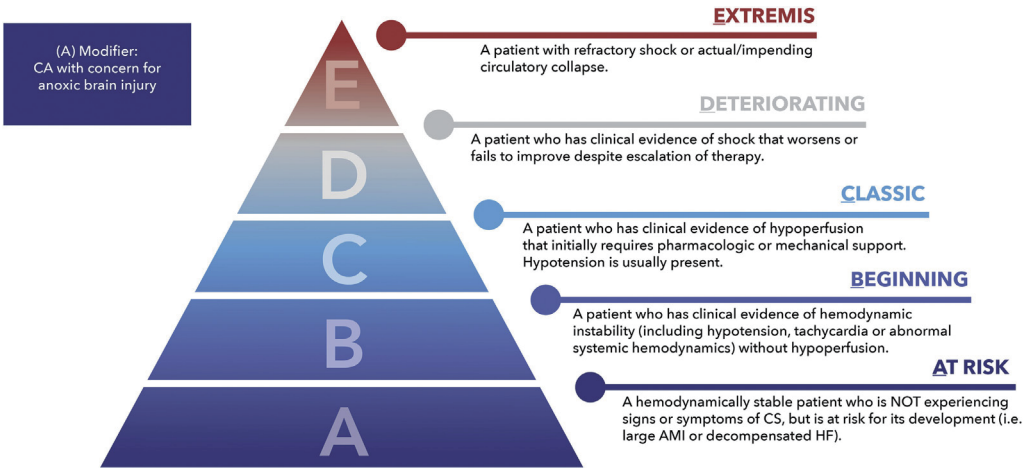

Figure 1

Stage A 風險期 At Risk

病人尚未有 CS 的症狀或徵象，但已有 CS 的疾病進展風險因子如 AMI，病人此時通常生命徵象穩定。

理學檢查：正常，頸靜脈壓正常。

實驗室檢查：大致正常（乳酸、腎功能等）。

血流動力學（若有）：血壓正常、Cardiac Index (CI) ≥ 2.5 L/min/m^2, CVP < 10 mmHg, PCWP ≤ 15 mmHg, PA (Pulmonary Artery) Saturation ≥ 65%。

發病率：46%

死亡率：3%

Stage B 起始期 Beginning（Pre-shock/Compensated Shock）

病人有血流動力學不穩定現象如低血壓或心搏過速但尚未有組織灌流不足（hypoperfusion）如肢體冰冷、小便量減少、意識改變等的臨床證據。

理學檢查：頸靜脈壓上升、肺囉音、肢體溫暖、遠端肢體脈動仍強。

實驗室檢查：乳酸正常、腎功能輕度異常、BNP 上升。

血流動力學：SBP < 90 mmHg or MAP < 60 mmHg or 30mmHg Drop from Baseline, Pulse ≥ 100 bpm, Cardiac Index (CI) > 2.2 L/min/m^2, PA Saturation ≥ 65%。

發病率：30%
死亡率：7.1%

Stage C 典型期 Classic

病人開始有組織灌流不足現象，病人此時通常表現有低血壓（但不一定）並需要開始使用一種藥物如強心劑、升壓劑、機械式循環支持 MCS 等初步穩定生命徵象（不包含輸液支持）。
理學檢查：有體液過多（Volume Overload）現象、病人極不舒服、恐慌、蒼白、皮膚溼冷或紫斑、微血管回充時間延遲（Delayed Capillary Refill）、肺部廣泛囉音、意識障礙、尿量每小時 <30cc、可能需要機械式通氣，Killip III or IV。
實驗室檢查：Creatinine 比基礎值上升 1.5 倍（2019 版本上升 2 倍）或 GFR 下降 50% 以上、乳酸 Lactate ≥ 2 mmol/L、BNP 上升、肝指數上升等。
血流動力學：MAP ≤ 60 mmHg or SBP ≤ 90 mmHg 需要藥物或機械式循環支持 MCS, Cardiac Index (CI) < 2.2 L/min/m^2, PCWP > 15mmHg, PAPi < 1.85, RAP/PCWP ≥ 0.8, Cardiac Power Output ≤ 0.6。
發病率：15.7%
死亡率：12.4%

Stage D 惡化期 Deteriorating（Doom）

對起始的支持性治療（如強心劑、升壓劑、機械式循環支持）無效，需要更近一步升級（Escalate）支持性治療。
理學檢查：同 Stage C。
實驗室檢查：同 Stage C 但更嚴重、Lactate 持續上升且持續 > 2 mmol/L。
血流動力學：同任何 Stage C 但需要多重升壓劑、或需要額外機械式循環 MCS 支持。
發病率：7.3%
死亡率：40.4%

Stage E 極端期 Extremis

病人循環衰竭、可能隨時會需要急救、心因性猝死 CA，可能需要 E-CPR（ECMO-facilitated CPR）及多種方法維持生命徵象。
理學檢查：幾乎摸不到脈搏、時常需要去顫／電擊。
實驗室檢查：“Trying to Die”，嚴重酸中毒 pH ≤ 7.2, Lactate ≥ 8 mmol/L（2019 版本 ≥ 5 mmol/L）, Base Deficit > 10 mEq/L。
血流動力學：若無復甦支持則無血壓、無脈搏的電氣活動（PEA）或頑固性心室頻脈 / 心室顫動（Refractory VT/VF）, 在極度支持之下仍低血壓、需要不間斷給予劑型（Bolus）注射升壓劑。
發病率：1%
死亡率：67%

Table 1

TABLE 3 Descriptors of Shock Stages: Physical Examination, Biochemical Markers, and Hemodynamics

		Physical examination/ bedside findings		Biochemical markers		Hemodynamics	
Stage	**Description**	**Typically includes**	**May include**	**Typically includes**	**May include**	**Typically includes**	**May include**
A At risk	A patient who is **not currently experiencing signs or symptoms of CS, but is at risk for its development**. These patients may include those with large acute myocardial infarction or prior infarction and/or acute or acute-on-chronic heart failure symptoms.	**Normal JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Clear lung sounds	**Normal lactate**	Normal labs • Normal (or at baseline) renal function	**Normotensive** (SBP ≥100 mmHg or at baseline)	If invasive hemodynamics are assessed: • Cardiac Index ≥2.5 L/min/m^2 (if acute) • CVP ≤10 mmHg • PCWP ≤15 mmHg • PA saturation ≥65%
B Beginning CS	A patient who has **clinical evidence of hemodynamic instability** (including relative hypotension or tachycardia) **without hypoperfusion**.	**Elevated JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Rales in lung fields	**Normal lactate**	Minimal acute renal function impairment Elevated BNP	**Hypotension** • SBP <90 mmHg • MAP <60 mmHg • >30 mmHg drop from baseline **Tachycardia** • Heart rate ≥100 bpm	
C Classic CS	A patient who manifests with **hypoperfusion and who requires one intervention (pharmacological or mechanical) beyond volume resuscitation**. These patients typically present with relative hypotension (but hypotension is not required).	**Volume overload**	Looks unwell Acute alteration in mental status Feeling of impending doom Cold and clammy Extensive rales Ashen, mottled, dusky, or cool extremities Delayed capillary refill Urine Output <30 mL/h	**Lactate ≥2 mmol/L**	Creatinine increase to 1.5 x baseline (or 0.3 mg/dL) or >50% drop in GFR Increased LFTs Elevated BNP	If invasive hemodynamics assessed (**strongly recommended**) • Cardiac index <2.2 L/min/m^2 • PCWP >15 mmHg	
D Deteriorating	A patient who is similar to category C but is getting worse. **Failure of initial support strategy to restore perfusion** as evidenced by worsening hemodynamics or rising lactate.	**Any of stage C and worsening (or not improving) signs/ symptoms of hypoperfusion despite the initial therapy.**		**Any of stage C and lactate rising and persistently >2 mmol/L**	Deteriorating renal function Worsening LFTs Rising BNP	**Any of stage C and requiring escalating doses or increasing numbers of pressors or addition of a mechanical circulatory support device to maintain perfusion**	
E Extremis	**Actual or impending circulatory collapse**	**Typically unconscious**	Near pulselessness Cardiac collapse Multiple defibrillations	**Lactate ≥8 mmol/L**[a]	CPR (A-modifier) Severe acidosis • pH <7.2 • Base deficit >10 mEq/L	**Profound hypotension despite maximal hemodynamic support**	Need for bolus doses of vasopressors

BNP = B-type natriuretic peptide; CPR = cardiopulmonary resuscitation; CVP = central venous pressure; GFR = glomerular filtration rate; JVP = jugular venous pressure; LFT = liver function tests; MAP = mean arterial pressure; PA = pulmonary artery; PCWP = pulmonary capillary wedge pressure; SVP = systolic ventricular pressure.
[a]Stage E prospectively is a patient with cardiovascular collapse or ongoing CPR.

Stage B & Stage C 間的差異

需要仔細的鑑別。有「組織灌流不足」但「沒有低血壓」的病人，其預後比「低血壓」但「沒有組織灌流不足」的病人，來得較差。Lactate 在此情況下可以幫助鑑別，但是若病人有慢性心衰竭病史則可能干擾診斷，因為即使病人 Cardiac Index 已經下降，Lactate 數值仍可能是正常的。另外有些情況如腸系膜動脈缺血（Mesenteric Ischemia）或腔室症候群（Compartment Syndrome）也可能造成 Lactate 上升，需特別注意。

Stage C & stage D 間的差異

若病人須要起始給予血管活性（Vasoactive）藥物或機械式循環支持 MCS 來反轉（Reverse）組織灌流不足狀態或維持血流動力學穩定，分類為 Stage C。如果起始的治療無效，需要更多血管活性藥物或增加機械式循環支持 MCS 來升級治療，則分類為 Stage D。因此在 Stage C 到 Stage D 當中，有包含一小段時間因素。若需要多種極大量的血管活性藥物或 MCS 支持仍無法維持有效循環、血流動力學穩定，則進展到 Stage E。2022 更新版則強調“Hub-and Spoke model”（輻軸網路模式），若病人進展到 Stage D，則應順「輻軸網路」將病人快速轉到更高層級的休克中心進行救治。

心因性猝死 Cardiac Arrest（CA）修飾因子（Modifier）-A

因為 CA 是重大事件，會顯著影響預後，故加上修飾因子（CA modifier）。例如 Stage B_A 的病人，為 Stage B 病人加上有 CA 的事件。CS 及 CA 常常同時發生，同時具有兩事件（CS with CA）的病人族群預後，當然比任何單一事件的病人族群來得差。2022 年的共識則淡化 CA Modifier 的角色，新版共識認為若是病人心室性心律不整 CA 若第一時間就接受去顫治療，在沒有任何神經缺陷的後遺症下，預後不會有顯著差異。因此 2022 版本只針對可能有腦損傷（Anoxic Brain Injury）的病人加上 CA Modifier：如 Glasgow Coma Scale 小於 9 分、或對於聲音刺激沒有運動反應的病人（Not Following Voice Command）。

年齡修飾因子 Age Modifier

2019 年版本沒有提到，2022 年版有提到年齡修飾因子對於預後影響很重要，但要如何妥善加入 SCAI Shock Stage 則尚未形成共識。2022 的專家共識強調，雖然 SCAI CS 分級系統有死亡風險分層，特別是整合風險修正因子時，但維持一個簡單容易應用、標準化但保持彈性的 CS 嚴重度分級系統用來溝通及治療決策擬定才是其主要的價值所在。

其他要點

肌鈣蛋白 T（Troponin T, TnT）：是預後不佳的獨立的風險預測因子，可作為病患風險分層的工具，CS 晚期的患者其 TnT 上升可作識別。

乳酸 Lactate：通常以 2 mmol/L 作為分界點（Cut-off），且動脈乳酸數值比靜脈乳酸數值來得好。

重碳酸鹽 Bicarbonate：降低在 30 日短期死亡率的預測能力比最高 lactate 指標來得好。
BNP：心衰竭指標，也可做為 CS 獨立的存活預後因子。在低血壓情境下，低 BNP 水平不支持 CS 的診斷，但是 BNP 上升也不能確定診斷，因為有眾多心房心室疾病會干擾鑑別。
其他生物標記：諸如 FGF-23, GDF-15, hsCRP, sTNFR1, Angiopoietin-2, sFas, sFasL, Endothelin-1, PIINP 等尚未適合臨床階段使用。
PAC 肺動脈導管：建議 CS 的診斷和治療中使用肺動脈導管（Pulmonary Artery Catheter, PAC）並建議病人需迅速轉移到經驗豐富的休克中心（Shock Center）。
混合性休克：因為病人可能有混合性休克（Mixed Shock）如合併有敗血性休克，故侵入性血流動力監測很重要可作為鑑別

範例：

SL 先生是 67 歲男性有三高及抽菸病史，10 年前因嚴重三條冠狀動脈疾病接受心臟繞道手術（CABG）。某日病人有胸悶現象，讓他從睡夢中醒來，到急診室時疼痛加劇，TnT 上升，血壓 94/70 mmHg，心搏每分鐘 100 次（基礎血壓 140/70 mmHg），因此他的 CS Stage 為 B。第二天在導管室時，他的心搏每分鐘 110 次，尿量減少，PAC 置放後得到 Cardiac Index 1.8 L/min/m^2，PCWP 為 29 mmHg，此時他的 CS Stage 為 C。心導管攝影發現 Saphenous Vein Graft 接到 RCA 有血栓產生，導管團隊考慮置放 IABP 並進行 Thrombectomy，但此時病人產生 Vf 並立即接受去顫電擊治療，若以 2019 年共識為 CS Stage C_A，但若依照 2022 年修正版本仍為 Stage C。術中有使用低劑量強心劑，在完成 Thrombectomy 後置入 IABP，病人晚上在 CCU 繼續治療，但病人小便量持續減少，即使加大強心劑劑量及 IABP 1:1 支持下其 Cardiac Index 仍舊 < 2 L/min/m^2，此時病人 CS Stage 為 D，應考慮加上 MCS 支持。

回溯性驗證研究 Retrospective Validation Study

2019 年 SCAI CS Stage 專家共識發布後，兩年間有不少回溯性驗證研究發表（Table 2），強調 CS Stage 和次群組（Subgroup）死亡率的相關性，包括有無急性冠狀動脈症候群（ACS）、CICU（CCU）病人族群、到院前心跳停止（OHCA）的患者。幾個觀察性驗證研究證明每個 CS Stage 的罹病率因研究族群、定義不同而有很大差異（Figure 2），短期（住院中死亡、或 30 日）死亡率也因不同族群有所差別（Figure 3），但較高的 CS Stage 均有較高的短期死亡率。這些研究還顯示，評估 CS 嚴重度是 CS 患者整體死亡風險分層的核心組成部分，但是其他臨床變數也會影響預期的死亡風險，例如某些風險修正因子（CA Modifier, Age Modifier 等），未來生物標記也可能被整合到風險評估當中，CS Stage 是一個動態過程會不斷變動（Figure 4 顯示患者反應和軌跡初始及重新分級過程），Stage 之間的轉換也具有重要的預後價值，但請注意 CS Stage 僅適用於急性期，不適用於慢性心臟疾病。

Table 2

TABLE 1 Characteristics of Studies Validating the Association Between the SCAI SHOCK Stage and Mortality

Study	Years Included	Population	Design	Patients, n	Primary Outcome
Schrage et al 2020[a]	2009-2017	CS or large MI	Retrospective single-center	1007	30-day survival
Baran et al 2020	2019-2020	CS	Prospective single-center	166	30-day survival
Thayer et al 2020	2016-2019	CS	Prospective multicenter[b]	1414	In-hospital mortality
Hanson et al 2020	2016-2019	AMICS	Prospective multicenter[b]	300	Survival to discharge
Jentzer et al 2021[a]	2007-2015	CS	Retrospective single-center	934	30-day survival
Jentzer et al 2019	2007-2015	CICU	Retrospective single-center	10,004	In-hospital mortality
Lawler et al 2021	2017-2019	CICU or CS	Retrospective multicenter	1991	In-hospital mortality
Jentzer et al 2020	2007-2015	CICU survivors	Retrospective single-center	9096	Postdischarge survival
Pareek et al 2020	2012-2017	OHCA	Retrospective single-center	393	30-day mortality

Duplicate data from the same cohort are not shown. AMICS = CS from acute myocardial infarction; CICU = cardiac intensive care unit; CS = cardiogenic shock; MI = myocardial infarction; OHCA = out-of-hospital cardiac arrest; SCAI = Society for Cardiovascular Angiography and Interventions.
[a]Patients with CS from the Schrage 2020 study were included in the Jentzer 2021 study, so only the nonduplicated patients are reported for the Jentzer 2021 study.
[b]Patient enrollment in these studies was prospective, but the SCAI SHOCK stage was assigned retrospectively.

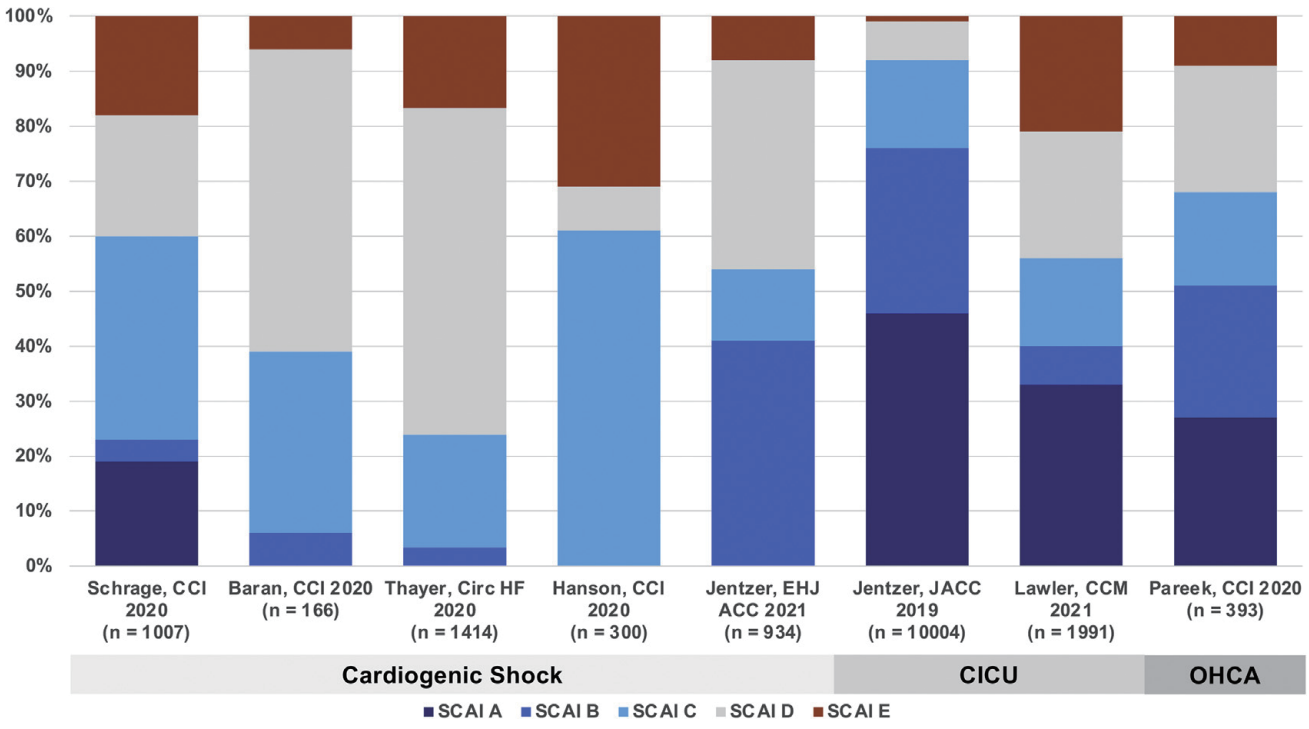

Figure 2

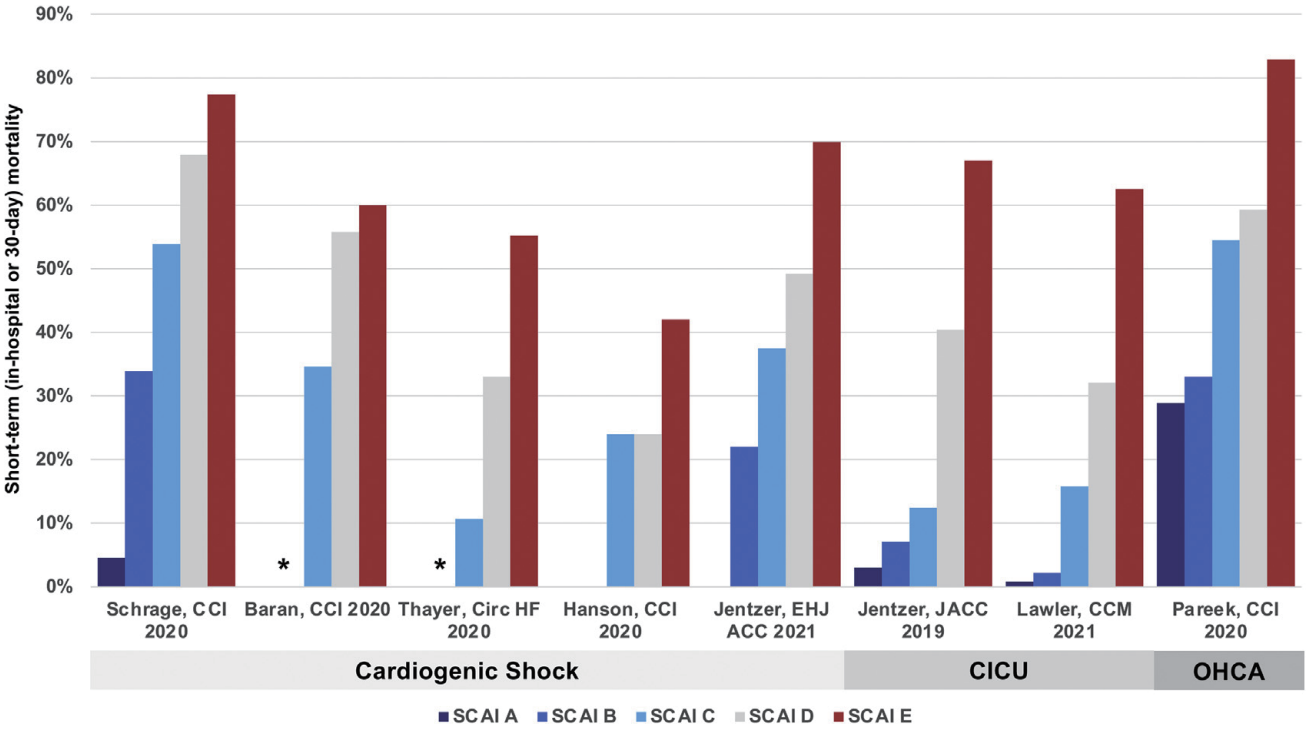

Figure 3

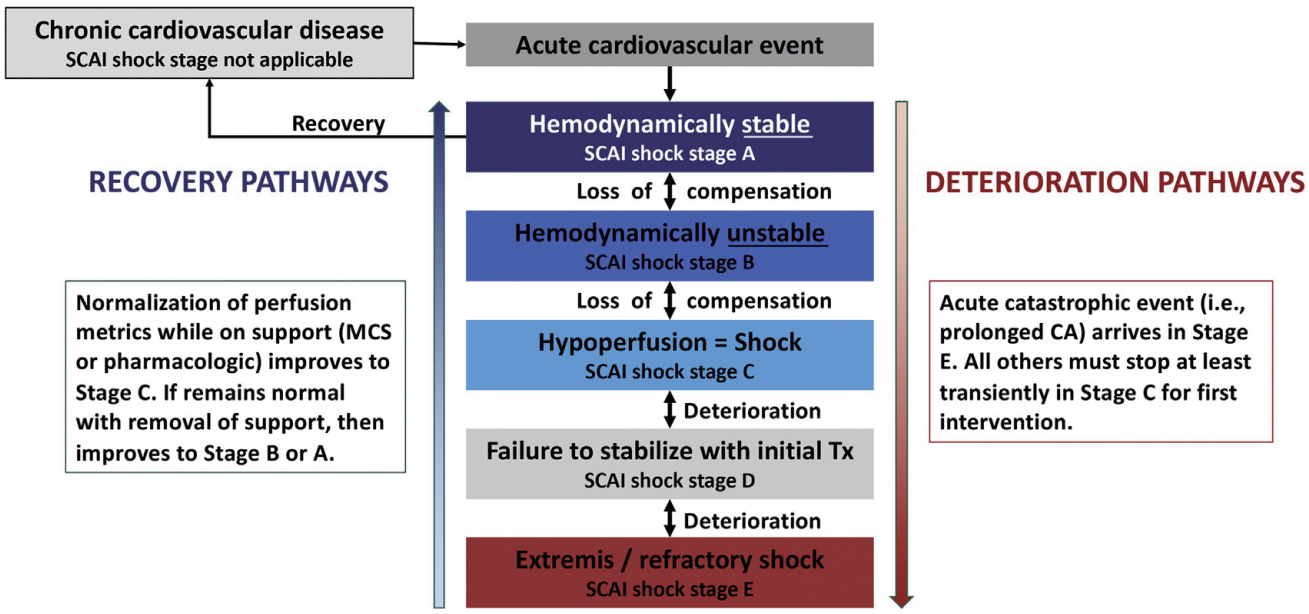

Figure 4

三軸模型 3-axis Model

2022 修訂之專家共識提出了 CS 評估和預測的三軸模型 (Figure 5)，將休克嚴重度、臨床表現型（Phenotype）及病因（Etiology）、風險修飾因子（Risk Modifiers）進行整合，用於病患的個人化管理。現有及新興的生物標記可能會進一步完善風險分層，但這需要未來的研究。

休克嚴重度：包括 SCAI CS Stage、血流動力學、代謝錯亂（Metabolic Derangement）、血管收縮劑毒性（Vasopressor Toxicity）。

表現型及病因：包括急性、慢性疾病急性發作（Acute-on-chronic）、CS 的臨床病因（Clinical Etiology）、右心室 vs 左心室 vs 雙心室（BIV）收縮障礙、心衰竭（Cardiac）vs 心肺衰竭（Cardiopulmonary）、鬱血狀態（Congestion Profile）、生化表型（Biochemical Phenotype）。

風險修飾因子：包括無法校正（Non-modifiable）的風險因子如年齡、共病（Co-morbidities），其他尚有 CA 合併昏迷（Coma）、有無可復原的器官衰竭（Reversible Organ Failure）、系統性炎症反應（Systemic Inflammatory Response）、衰弱（Frailty）及併發症（Complication）。

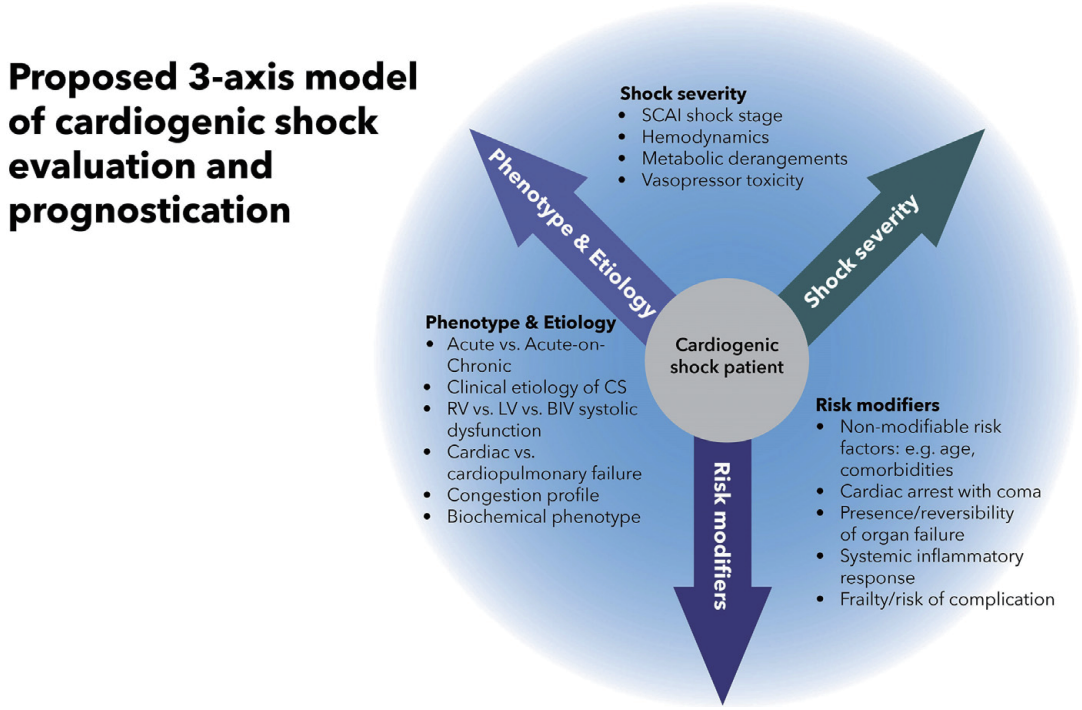

Figure 5

結語

在沒有標準化分級系統的情況下，無法確定哪些群體可能受益，SCAI CS Stage Classification 是廣泛多學科專家通力合作的產物，建立在簡單應用、臨床實踐的基礎上，透過標準化的分類系統，有助于提高不同機構、不同 CS 治療團隊的溝通效率，並為臨床研究提供互相溝

通的共同語言（Lingua Franca），例如廣泛引用的 Impress Trial，這個研究是比較 Impella 和 IABP 的預後，若是用 SCAI Stage 來分級，其病人族群是 SCAI CS Stage D，但是另外一個廣泛引用的 IABP Shock II Trial，其研究族群是 SCAI Stage C 患者，因此要比較這兩個研究時要特別注意。2022 年版本最後強調 CS 患者住院期間的軌跡，除了希望識別出對治療有反應的患者，更要識別出對於治療沒有反應或病情惡化的患者，來調整升階介入治療策略，或考慮緩和治療。

Reference:

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Late Balloon Valvuloplasty for Transcatheter Heart Valve Dysfunction

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ABSTRACT

BACKGROUND
Transcatheter heart valve (THV) dysfunction with an elevated gradient or paravalvular leak (PVL) may be documented late after THV implantation. Medical management, paravalvular plugs, redo THV replacement, or surgical valve replacement may be considered. However, late balloon dilatation is rarely utilized because of concerns about safety or lack of efficacy.

OBJECTIVES
We aimed to evaluate the safety and efficacy of late dilatation in the management of THV dysfunction.

METHODS
All patients who underwent late dilatation for symptomatic THV dysfunction at 2 institutions between 2016 and 2021 were identified. Baseline, procedural characteristics, and clinical and echocardiographic outcomes were documented. THV frame expansion was assessed by multislice computed tomography before and after late dilatation.

RESULTS
Late dilatation was performed in 30 patients a median of 4.6 months (IQR: 2.3-11.0 months) after THV implantation in the aortic (n = 25; 83.3%), mitral (n = 2; 6.7%), tricuspid (n = 2; 6.7%) and pulmonary (n = 1; 3.3%) position. THV underexpansion was documented at baseline, and frame expansion substantially improved after late dilatation. The mean transvalvular gradient fell in all patients. For aortic THVs specifically, mean transaortic gradient fell from 25.4 ± 13.9 mm Hg to 10.8 ± 4.1 mm Hg; $P < 0.001$. PVL was reduced to ≤mild in all 11 patients with a previous >mild PVL. Embolic events, stroke, annular injury, and bioprosthetic leaflet injury were not observed. Symptomatic benefit was durable at 19.6 months (IQR: 14.8-36.1 months) follow-up.

CONCLUSIONS

Balloon dilatation late after THV implantation appears feasible and safe in appropriately selected patients and may result in THV frame expansion resulting in improvements in hemodynamic performance and PVL.

延遲性氣球擴張術治療經導管置換心臟瓣膜之功能失常

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背景

經導管置換心臟瓣膜功能失常 (Transcatheter Heart Valve [THV] Dysfunction)，包括經瓣膜的壓力差增加或是瓣膜旁側滲漏 (Paravalvular Leak [PVL]) 都可以在瓣膜置換術後發生。治療的方式包括藥物治療、使用封堵裝置、經導管重置瓣膜、或者是外科手術重新置換等等。然而在術後延遲利用氣球擴張術 (Late Balloon Dilatation) 治療的方式一直因為安全性的考量以及缺乏效益的實證而很少被使用。

研究目的

本研究之目的即在評估 THV dysfunction 之患者術後延遲性使用氣球擴張術治療之安全性以及有效性。

研究方法

本研究在加拿大 St Paul's and Vancouver General Hospitals 所屬之兩個中心在 2016 至 2021 年針對所有因為有症狀的 THV Dysfunction 而接受延遲性氣球擴張術治療之患者進行收案。研究針對病患的術前及術後之症狀及心臟超音波進行分析，THV 的支架擴張在術前及術後均經由電腦斷層影像評估量測。

結果

總計有 30 位病患在 THV 中位數 4.6 個月後進行延遲性氣球擴張術 (IQR: 2.3-11.0 個月)。經導管置換主動脈瓣有 25 位 (83.3%)、二尖瓣 2 位 (6.7%)、三尖瓣 2 位 (6.7%) 以及肺動脈瓣 1 位 (3.3%) (圖一)。THV Dysfunction 在氣球擴張術後都可以明顯改善瓣膜骨架擴張不足的現象，尤其是主動脈瓣 (圖二及圖三)。同時經氣球擴張術後，經瓣膜壓力差 (Mean Transvalvular Gradient) 都有顯著下降，尤其主動脈 THV 的壓力差由 25.4 ± 13.9 mm Hg 顯著下降至 10.8 ± 4.1 mm Hg；P < 0.001. PVL 在所有術前評估為超過輕度之病患都在氣球擴張術後減輕至 Mild 以下 (圖四)。 嚴重的副作用如栓塞，瓣膜環損傷或是瓣膜受損的狀況都沒有觀察到。術後症狀的改善在追蹤至 19.6 個月 (IQR: 14.8-36.1 個月) 都能持續地維持 (圖五)。

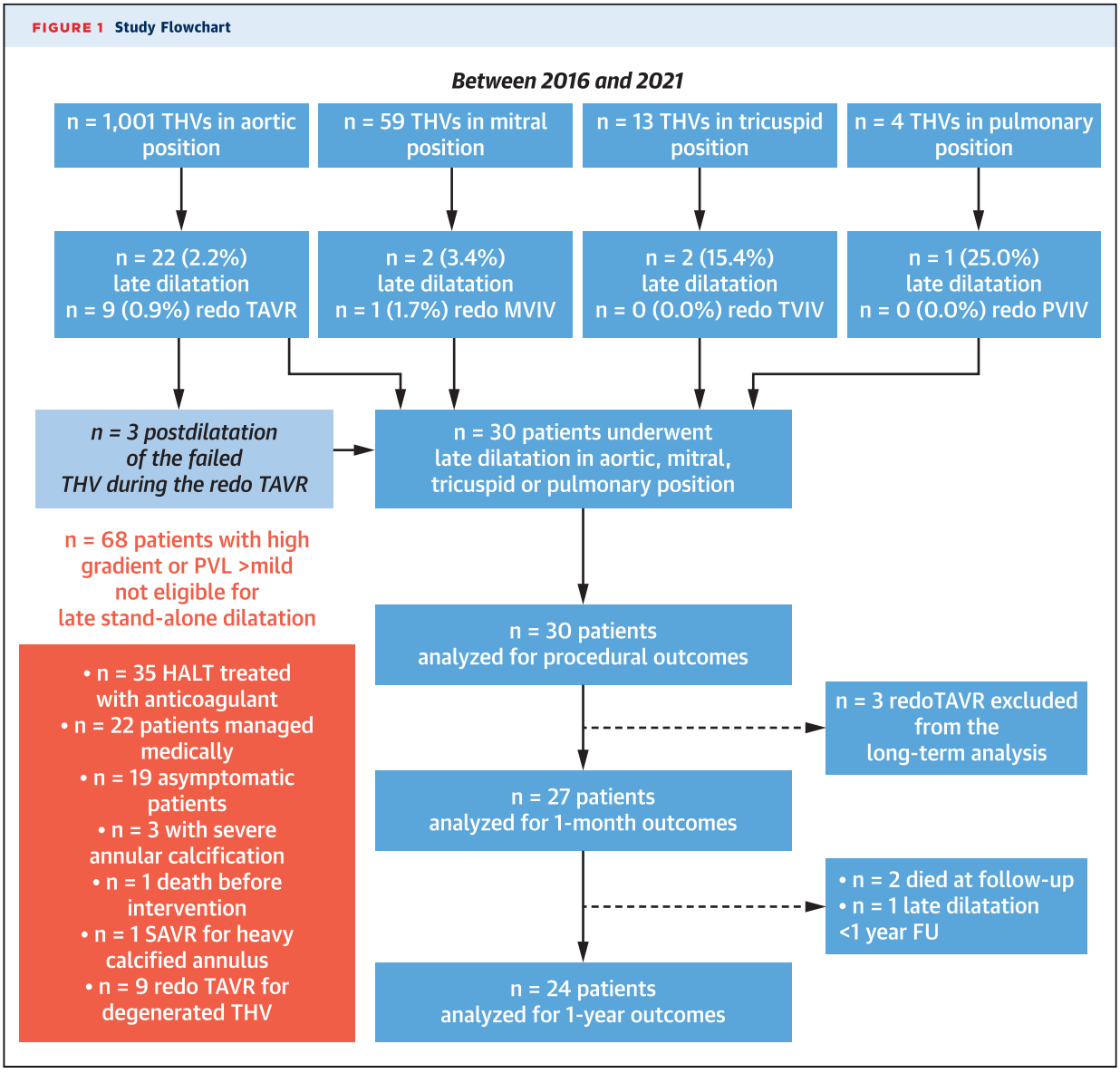
討論

本研究的分析發現 THV dysfunction 多是由於瓣膜骨架擴張不全造成，因此在執行瓣膜置換時，確保瓣膜充分擴張是相當重要的。術後延遲施做氣球擴張術在本研究中顯示可以改善

瓣膜擴張、血流動力學以及症狀的改善。惟本研究之觀察期較短，樣本數也較小，針對主動脈瓣之外的 THV 分析尚無法達到統計學意義，尚待未來有更大樣本數及長時間的研究來釐清。

結論

在 THV 術後進行延遲性的氣球擴張術在本研究中顯示在適當選擇的病患上施作是安全的。氣球擴張術後在本研究也顯示可以改善人工瓣膜支架的擴張，並且有血流動力學及症狀上的持久改善。



圖一

TABLE 6 Pre and Post Late Stand-Alone Balloon Dilatation MSCT Analysis for Paired Aortic and Nonaortic THVs

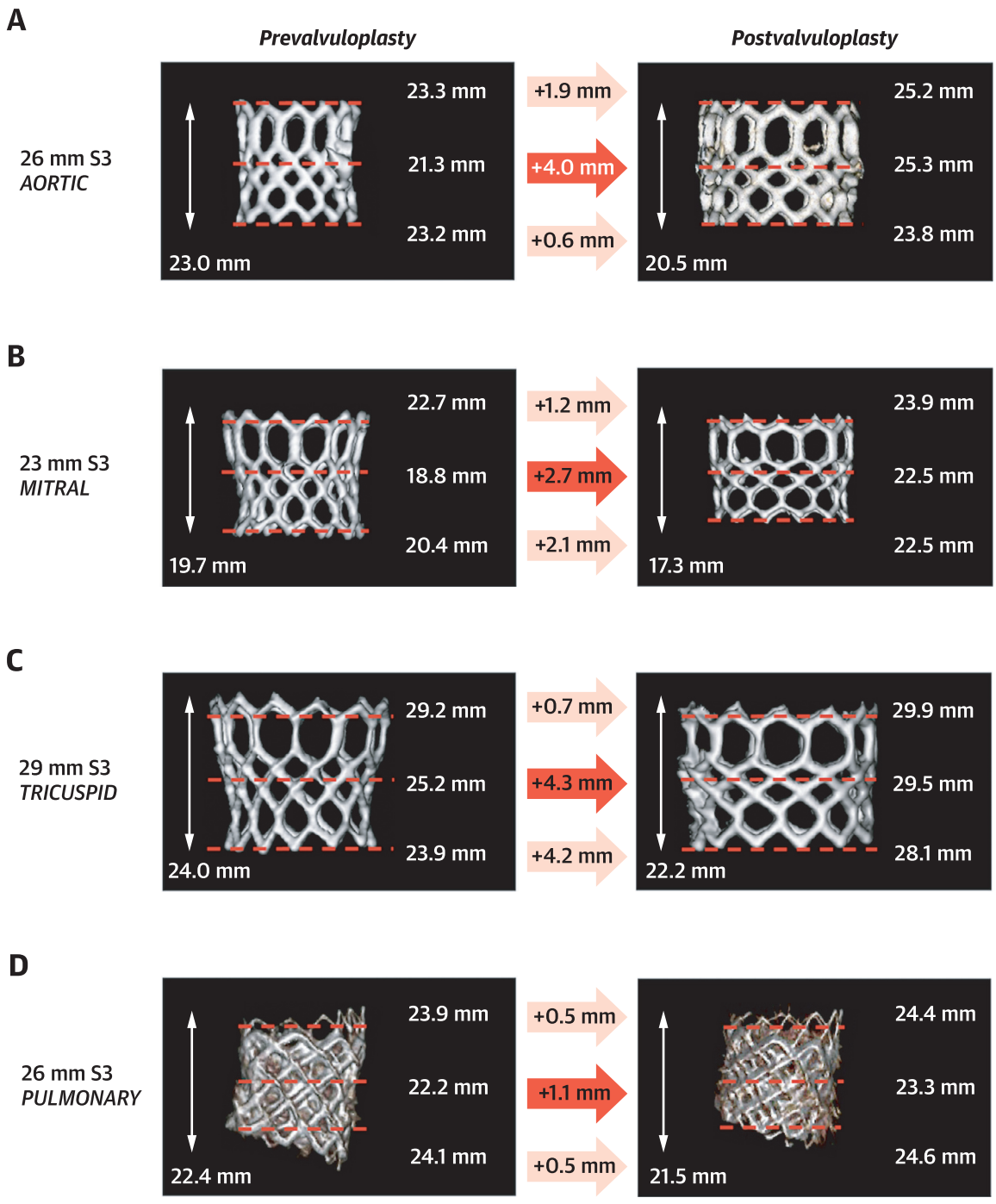
	Pre Late Stand-Alone Dilatation	Post Late Stand-Alone Dilatation	Difference Pre/Post Late Dilatation	*P* Value
All paired THVs in aortic position (n = 10)				
THV expansion, %				
Inflow	80.7 (75.8 to 84.7)	89.2 (82.5 to 99.1)	+7.3 (6.0 to 11.4)	0.05
Mid	65.7 (63.3 to 68.4)	96.3 (86.5 to 98.3)	+25.8 (19.2 to 33.1)	<0.001
Mean diameter, mm				
Inflow	21.3 (20.4 to 22.5)	23.1 (22.1 to 23.5)	+1.0 (0.8 to 2.1)	0.09
Mid	18.6 (17.9 to 20.8)	23.0 (21.1 to 25.3)	+3.9 (3.0 to 4.7)	0.003
Sphericity index				
Inflow	0.9 (0.9 to 1.0)	0.9 (0.9 to 1.0)	0.0 (0.0 to 0.0)	0.60
Mid	0.9 (0.9 to 1.0)	0.9 (0.9 to 1.0)	0.0 (0.0 to 0.0)	0.80
Paired balloon expandable THVs in aortic position (n = 8)				
THV expansion, %				
Inflow	81.8 (79.3 to 86.6)	94.8 (85.1 to 100.5)	+8.5 (6.8 to 14.3)	0.01
Mid	65.7 (62.3 to 68.1)	97.1 (94.9 to 99.4)	+29.9 (23.5 to 34.3)	<0.001
Outflow	93.8 (91.7 to 97.7)	103.4 (100.9 to 105.4)	+9.0 (6.7 to 10.3)	0.001
Mean diameter, mm				
Inflow	21.6 (20.9 to 22.9)	23.3 (23.0 to 23.7)	+1.0 (0.8 to 1.9)	0.04
Mid	19.5 (18.4 to 21.0)	24.3 (22.4 to 25.3)	+4.1 (3.0 to 5.0)	0.003
Outflow	23.0 (22.5 to 24.7)	24.4 (23.4 to 25.9)	+1.2 (0.8 to 1.3)	0.07
Sphericity index				
Inflow	0.9 (0.9 to 1.0)	0.9 (0.9 to 1.0)	0.0 (0.0 to 0.0)	0.90
Mid	0.9 (0.9 to 1.0)	0.9 (0.9 to 1.0)	0.0 (0.0 to 0.0)	1.00
Outflow	0.9 (0.9 to 1.0)	0.9 (0.9 to 1.0)	0.0 (0.0 to 0.0)	0.60
Mean THV height, mm	20.8 (20.0 to 22.0)	19.8 (19.8 to 20.5)	−1.5 (−1.9 to −1.1)	0.10
Paired THV in surgical bioprosthetic valve (n = 7)				
THV expansion, %				
Inflow	78.4 (72.5 to 80.6)	83.9 (79.8 to 97.5)	7.8 (6.4 to 16.6)	0.09
Mid	65.4 (72.5 to 80.6)	96.0 (83.1 to 100.1)	31.9 (17.8 to 35.1)	<0.01
Mean diameter, mm				
Inflow	20.6 (18.8 to 21.3)	23.0 (20.4 to 23.3)	+1.7 (0.9 to 2.4)	0.09
Mid	18.1 (17.8 to 19.5)	22.7 (20.6 to 24.3)	+4.1 (3.3 to 5.1)	0.01
Sphericity index				
Inflow	1.0 (0.9 to 1.0)	0.9 (0.9 to 1.0)	0.0 (0.0 to 0.0)	1.00
Mid	1.0 (0.9 to 1.0)	1.0 (0.9 to 1.0)	0.0 (0.0 to 0.0)	0.80
All paired THVs in nonaortic position (n = 4)				
THV expansion, %				
Inflow	80.4 (76.1 to 89.5)	95.6 (93.6 to 107.3)	22.1 (15.9 to 26.2)	0.20
Mid	72.2 (66.1 to 81.5)	100.5 (96.0 to 104.0)	28.5 (23.2 to 29.3)	0.06
Outflow	101.0 (97.9 to 104.9)	108.4 (107.5 to 110.3)	7.4 (5.4 to 9.6)	0.20
Mean diameter, mm				
Inflow	22.4 (20.7 to 23.9)	23.6 (22.2 to 25.5)	+1.2 (0.5 to 2.6)	0.30
Mid	20.5 (18.6 to 22.9)	22.9 (22.4 to 24.8)	+4.0 (3.7 to 4.3)	0.30
Outflow	23.3 (22.6 to 25.2)	24.1 (23.8 to 25.8)	+1.0 (0.7 to 1.2)	0.40
Sphericity index				
Inflow	1.0 (1.0 to 1.0)	1.0 (1.0 to 1.0)	0.0 (0.0 to 0.0)	1.00
Mid	0.9 (0.9 to 1.0)	1.0 (0.9 to 1.0)	0.0 (0.0 to 0.0)	1.00
Outflow	0.9 (0.9 to 1.0)	1.0 (0.9 to 1.0)	0.0 (0.0 to 0.0)	0.70
Mean THV height, mm	21.1 (19.6 to 22.8)	19.8 (18.0 to 21.7)	−1.4 (−1.9 to −1.0)	0.30

Values are median (IQR). THV expansion, mean THV diameter, THV sphericity index, and mean THV height assessed by MSCT pre and post late balloon dilatation in paired aortic THVs, in aortic balloon expandable THVs, in aortic valve-in-valve configuration, and in non-aortic THVs. Outflow and THV height were not analyzed for self-expandable THVs.

THV = transcatheter heart valve.

圖二

FIGURE 3 Volume Rendered MSCTs Showing Underexpanded THVs With Improved Expansion Postvalvuloplasty



(A) A 26-mm S3 aortic THV expanded using a 25-mm True balloon; **(B)** 23-mm S3 THV in a 25-mm Epic mitral valve expanded using a 23-mm True balloon; **(C)** 29-mm S3 THV in a 29-mm Epic tricuspid valve expanded using a 29-mm True balloon; **(D)** 23-mm S3 THV in a 22-mm Melody pulmonary implant using a 23-mm True balloon. **Red lines** = mean THV diameter at the outflow, mid, and inflow; **white arrow** = mean THV height; **orange arrows** = mean THV diameter increase after dilatation at the outflow and inflow; **red arrow** = mean THV diameter increase after dilatation at the mid portion.
MSCT = multislice computed tomography; THV = transcatheter heart valve.

圖三

TABLE 5 Echocardiographic Parameters Immediately Post-THV Implantation, Pre and Post Late Stand-Alone Balloon Dilatation (Excluding Redo TAVR)

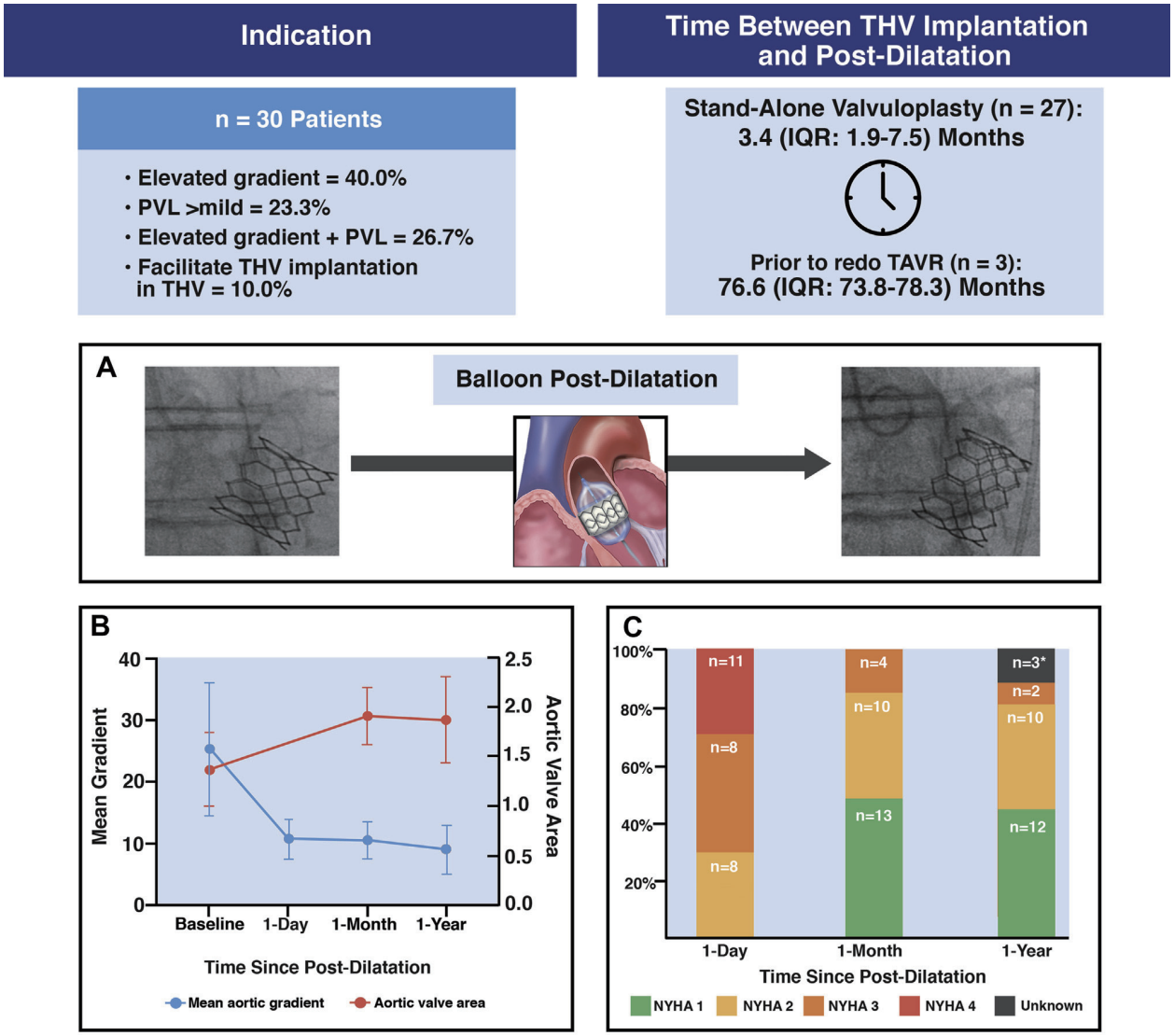
	Post-THV Implantation (n = 27)	Pre Stand-Alone Postdilatation (n = 27)	Post Stand-Alone Postdilatation (n = 27)	*P* Value
LVEF, %				0.8
>50	21 (77.8)	18 (66.7)	18 (66.7)	
35-50	4 (14.8)	8 (29.6)	8 (29.6)	
<35	2 (7.4)	1 (3.7)	1 (3.7)	
Aortic position (n = 22)				
Mean gradient, mm Hg	15.5 ± 9.8	25.4 ± 13.9	10.8 ± 4.1	<0.01
PVL >mild	6 (27.3)	11 (50.0)	0 (0.0)	<0.01
Mitral position (n = 2)				NA
Mean gradient, mm Hg	3.5 ± 0.7	14.0 ± 1.4	3.5 ± 0.7	
PVL >mild	0 (0.0)	0 (0.0)	0 (0.0)	
Tricuspid position (n = 2)				NA
Mean gradient, mm Hg	5.0 ± 2.8	7.5 ± 0.7	4.0 ± 0.0	
PVL >mild	0 (0.0)	0 (0.0)	0 (0.0)	
Pulmonary position (n = 1)				NA
Peak gradient, mm Hg	29	49	19	
PVL >mild	0 (0.0)	1 (100.0)	0 (0.0)	

Values are n (%) or mean ± SD, unless otherwise indicated. The *P* value is given for pre and post late stand-alone balloon dilatation.

LVEF = left ventricular ejection fraction; PVL = paravalvular leak; THV = transcatheter aortic valve.

圖四

CENTRAL ILLUSTRATION **Indications, Transcatheter Heart Valve Hemodynamic Performance, and Symptoms After Late Balloon Dilatation**



Akodad M, et al. J Am Coll Cardiol. 2022;79(14):1340–1351.

(A) Fluoroscopy showing an underexpanded Sapien 3 THV 2 months after implantation. Late balloon dilatation resulted in full expansion with resolution of moderate paravalvular regurgitation. **(B)** Mean aortic gradient and aortic valve area pre and post late balloon dilatation with error bars representing 1 SD. **(C)** NYHA functional class pre and post late balloon dilatation. Elevated mean gradient was defined as a mean gradient >20 mm Hg and/or an increase in mean gradient >10 mm Hg for aortic implants and mean gradient >10 mm Hg and/or an increase in mean gradient >5 mm Hg for mitral, tricuspid, and pulmonary implants. *At 1-year follow-up, 2 patients had died, and 1 clinical follow-up was not performed as follow-up is <1 year. NYHA = New York Heart Association; PVL = paravalvular leak; TAVR = transcatheter aortic valve replacement; THV = transcatheter heart valve.

圖五

Treatment of Coronary De Novo Lesions by a Sirolimus- or Paclitaxel-Coated Balloon

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J Am Coll Cardiol Intv 2022; : -

ABSTRACT

OBJECTIVES
The aim of this randomized controlled trial was to investigate a novel sirolimus-coated balloon (SCB) compared with the best investigated paclitaxel-coated balloon (PCB).

BACKGROUND
There is increasing clinical evidence for the treatment of coronary de novo disease using drug-coated balloons. However, it is unclear whether paclitaxel remains the drug of choice or if sirolimus is an alternative, in analogy to drug-eluting stents.

METHODS
Seventy patients with coronary de novo lesions were enrolled in a randomized, multicenter trial to compare a novel SCB (SeQuent SCB, B. Braun Melsungen; 4 μg/mm^2) with a PCB (SeQuent Please, B. Braun Melsungen; 3 μg/mm^2). The primary endpoint was angiographic late lumen loss (LLL) at 6 months. Secondary endpoints included major adverse cardiovascular events and individual clinical endpoints such as cardiac death, target lesion myocardial infarction, clinically driven target lesion revascularization, and binary restenosis.

RESULTS
Quantitative coronary angiography revealed no differences in baseline parameters. After 6 months, in segment LLL was 0.01 ± 0.33 mm in the PCB group versus 0.10 ± 0.32 mm in the SCB group. The mean difference between SCB and PCB was 0.08 (95% CI: -0.07 to 0.24). Noninferiority at a predefined margin of 0.35 was shown. However, negative LLL was more frequent in the PCB group (60% of lesions vs 32% in the SCB group; $P = 0.019$). Major adverse cardiovascular events up to 12 months also did not differ between the groups.

CONCLUSIONS

This first-in-human comparison of a novel SCB with a crystalline coating showed similar angiographic outcomes in the treatment of coronary de novo disease compared with a clinically proven PCB. However, late luminal enlargement was more frequently observed after PCB treatment.

比較 Sirolimus 或 Paclitaxel 塗藥氣球在冠狀動脈原發病灶的治療

編譯：台北馬偕紀念醫院 心臟內科 總醫師 丁勇翔
主治醫師 余法昌 / 蔡政廷

前言

塗藥支架 (DES) 在冠狀動脈介入治療已經具有重要的地位，在近年來，Sirolimus 和其類似物已經成為塗藥支架的藥物選擇之一，然而在塗藥氣球 (DCBs) 的隨機對照試驗 (RCT) 中，除了一個研究之外，其餘都是使用紫杉醇塗藥氣球 (Paclitaxel-coated Balloons, PCBs)。Sirolimus 應用在塗藥氣球相較紫杉醇更加著重於延長藥物釋放時間，部分研究發現，Sirolimus 塗藥氣球的藥物組織濃度會快速下降，反之，結晶狀 Sirolimus 使用 2,6- 二丁基對甲酚 (Butylated Hydroxytoluene) 作為賦形劑在目前的臨床實驗中發現可在一個月內維持 50% 的起始血管濃度。

在塗藥支架內再狹窄 (ISR) 首次於人體執行的 50 人隨機分派試驗研究中發現，新型 sirolimus 塗藥氣球 (SCB) (SeQuent SCB, B Braun Melsungen; 4 μg/mm^2) 和紫杉醇氣球 (PCB) (SeQuent Please, B Braun Melsungen; 3 μg/mm^2) 在 6 個月的追蹤下並無血管攝影的顯著差異。目前對於冠狀動脈原發性病灶迄今則沒有隨機分派試驗的發表。本研究為多中心隨機對照試驗，其的目為觀察 sirolimus 塗藥氣球 (SeQuent SCB) 與紫杉醇塗藥氣球 (SeQuent Please PCB) 對於冠狀動脈原發病灶的臨床療效。

研究方法

此研究隨機選取了 70 名來自多中心，18 歲以上，臨床上有心絞痛或功能性檢查陽性且有冠狀動脈原發病灶 (大於等於 70% 狹窄或是大於等於 50% 狹窄合併有陽性功能性檢查或是心肌缺血症狀) 的患者，比較新式 SCB 和 PCB。主要評估指標為 6 個月內血管攝影下的晚期血管丟失 (Late Lumen Loss, LLL) (用血管攝影比較術後和六個月後的最小血管直徑差異)。次要評估指標為主要心血管不良事件 (MACE) 和個別的臨床評估指標 (包含心因性死亡、目標病灶心肌梗塞、臨床目標病灶血管重建和血管再狹窄)。

結果

35 位病人分配至 SCB 組，35 位病人分配至 PCB 組，兩組的病人屬性很相似 (表一)，超過一半的病人有糖尿病 (63%) 和多血管冠狀動脈疾病 (77%)，在 PCB 組當中有 38 個病灶被治療，SCB 組中則有 37 個病灶被治療，平均每個病人使用 1.1 個氣球，平均擴張壓力為 8 atm，平均擴張時間為 58 秒，處置的資料可參考表二，研究的流程則可參考圖一。

表一

TABLE 1 Clinical Baseline Data

	PCB (n = 35)	SCB (n = 35)	*P* Value
Age, y	59 ± 12	60 ± 11	0.607
Male	30 (86)	26 (74)	0.371
Height, cm	163 ± 9	163 ± 9	0.905
Weight, kg	71 ± 17	69 ± 12	0.498
Angina pectoris status stable	17 (49)	15 (43)	0.811
CCS class			0.424
1	18 (51)	14 (40)	
2	9 (26)	12 (34)	
3	2 (6)	1 (3)	
4	0	2 (6)	
Missing	6	6	
Prior PTCA	11 (31)	13 (37)	0.802
Prior CABG	1 (3)	0 (0)	1.000
History of any myocardial infarction	16 (46)	12 (34)	0.332
Hypertension	23 (66)	24 (69)	1.000
Prior stroke	1 (3)	2 (6)	1.000
Diabetes	17 (49)	19 (54)	0.811
Insulin	3 (9)	3 (9)	1.000
Hyperlipidemia	20 (57)	18 (51)	0.451
Smoking	6 (17)	7 (20)	0.766

Values are mean ± SD or n (%).

CABG = coronary artery bypass graft; CCS = Canadian Cardiovascular Society; PCB = paclitaxel-coated balloon; PTCA = percutaneous transluminal coronary angioplasty; SCB = sirolimus-coated balloon.

表二

TABLE 2 Procedural Data

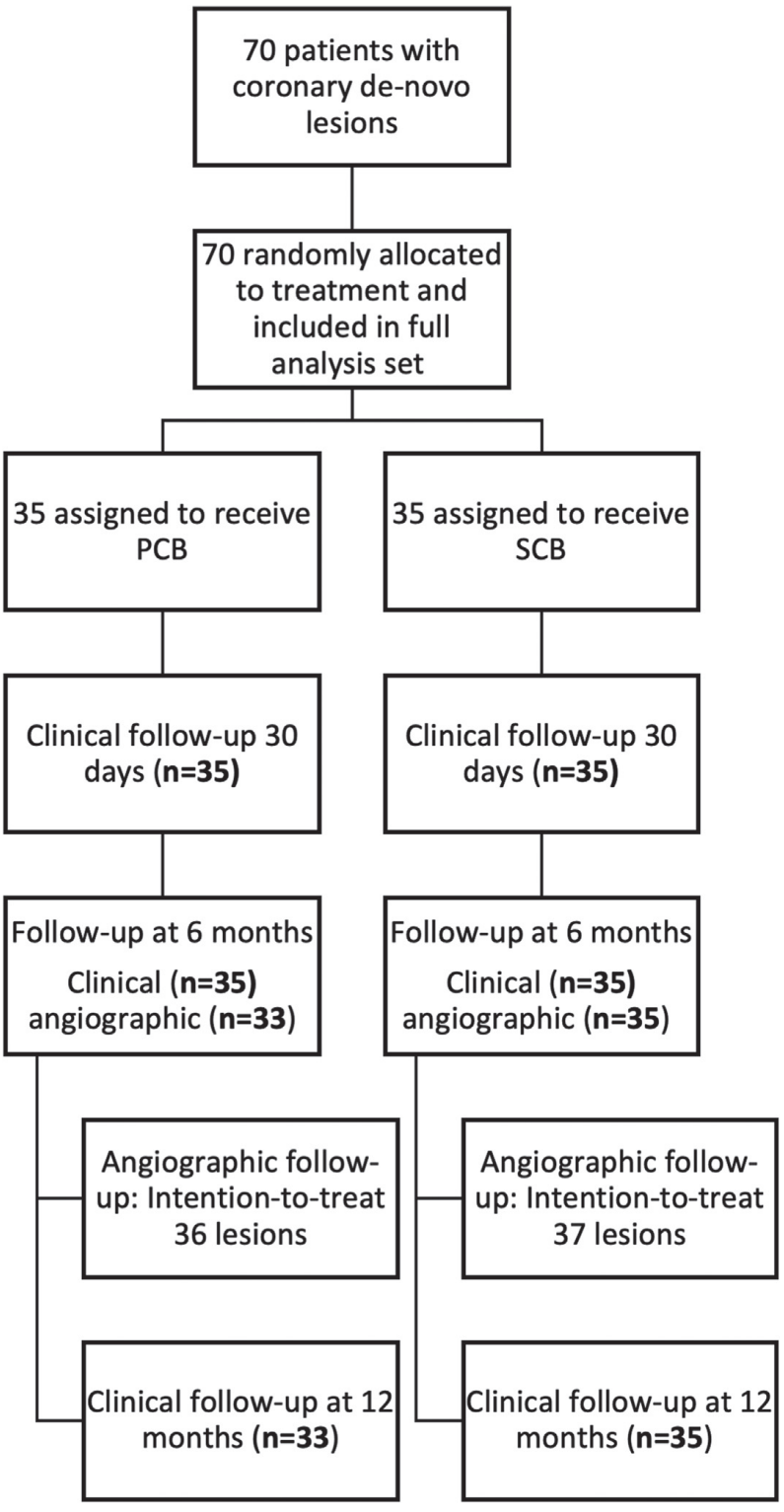
	PCB (n = 35)	SCB (n = 35)	*P* Value
Number of lesions	36	37	
Multivessel disease	28 (80)	23 (66)	0.392
Left ventricular ejection fraction, %	52 ± 11 (n = 16)	57 ± 16 (n = 17)	0.296
TIMI flow grade before procedure			0.415
1	0	2 (6)	
2	9 (26)	9 (26)	
3	24 (69)	19 (54)	
Missing	2 (6)	5 (14)	
Percentage stenosis (visual estimation by operator)	79 ± 9	77 ± 11	0.444
Predilatation	35 (100)	35 (100)	
Number of balloons used			0.541
Mean ± (range)	1.5 ± 0.6 (1-3)	1.6 ± 0.9 (1-5)	
Median (IQR)	1 (1-2)	1 (1-2)	
Balloon type used			0.116
Scoring	18	20	
POBA	27	34	
Other	8	2	
Number of inflations			0.611
Mean (range)	4.9 ± 3.6 (1-17)	4.5 ± 2.9 (1-13)	
Median (IQR)	4 (2-7)	4 (2-5)	
Highest pressure used, bar	13.8 ± 2.8 (9-20)	13.5 ± 3.1 (8-18)	0.852
Total duration of inflation, s	110.6 ± 127.4 (1-726)	104.2 ± 84.1 (1-387)	0.568
Number of study balloons	40	39	
Balloon pressure, bar	8 ± 2	7 ± 2	0.184
Balloon inflation time, s	58 ± 11	57 ± 8	0.603
Bail-out stenting	1 (3)	1 (3)	
TIMI flow grade at end of procedure (n,%)			1.000
2	1 (3)	0	
3	34 (97)	35 (100)	
Final diameter stenosis, %	10 ± 8	9 ± 8	0.906
Final dissection			1.000
Type A	5 (14)	3 (9)	
Type B	2 (6)	1 (3)	
Type D	1 (3)	0	

Values are n, n (%), mean ± SD, mean ± SD (range), or median (IQR).

CAD = coronary artery disease; POBA = plain old balloon angioplasty; TIMI = Thrombolysis In Myocardial Infarction; other abbreviations as in Table 1.

圖一

FIGURE 1 Study Flowchart



PCB = paclitaxel-coated balloon; SCB = sirolimus-coated balloon.

兩組病人在定量的冠狀動脈血管攝影中基本上沒有差異，在 6 個月後的追蹤中，使用 PCB 組的區段內 Late Lumen Loss (In-segment LLL) 為 0.01 ± 0.33 mm，在 SCB 組則為 0.10 ± 0.32 mm，平均差異為 0.08 (95% CI: -0.07-0.24)，驗證了預先定義的 0.35 邊際之非劣效性測試 (表四)。

負向的 LLL 在 PCB 組比較多見 (60% vs 32%; P=0.019) (表三)，在 12 個月的追蹤中，兩組病人的臨床事件發生率也沒有顯著差異 (表五)，圖二和圖三則為兩組病人的區段內最小管徑和 LLL 的累積頻率分布。

表三

TABLE 3 Quantitative Coronary Angiography: Intention-to-Treat Analysis

	PCB (n = 36 Lesions)	SCB (n = 37 Lesions)	*P* Value
Lesion length pre-PCI, mm	26.67 ± 8.04	23.82 ± 7.32	0.117
RFD pre-PCI, mm	2.81 ± 0.59	2.72 ± 0.40	0.494
In-lesion MLD pre-PCI, mm	0.89 ± 0.38	0.97 ± 0.44	0.453
In-segment MLD pre-PCI, mm	0.90 ± 0.38	0.96 ± 0.43	0.533
In-lesion diameter stenosis pre-PCI, %	63.6 ± 13.2	61.1 ± 15.0	0.454
In-lesion area stenosis pre-PCI, %	85.2 ± 11.3	81.6 ± 17.0	0.292
In-lesion MLD after predilatation, mm	1.74 ± 0.58	1.79 ± 0.44	0.722
In-lesion diameter stenosis after predilatation, %	30.5 ± 16.9	24.9 ± 13.6	0.124
In-lesion area stenosis after predilatation, %	47.1 ± 20.7	41.8 ± 18.1	0.256
In-lesion mean final MLD, mm	2.42 ± 0.52	2.40 ± 0.40	0.876
In-lesion final MLD, mm	2.11 ± 0.52	2.11 ± 0.40	0.976
In-segment final MLD, mm	2.02 ± 0.55	2.03 ± 0.39	0.909
In-lesion final diameter stenosis, %	18.3 ± 10.0	14.1 ± 9.3	0.075
In-lesion final area stenosis, %	30.8 ± 15.3	25.4 ± 15.5	0.150
In-lesion acute gain, mm	1.21 ± 0.44	1.14 ± 0.45	0.470
FU, d	189 (175-210)	189 (180-205)	0.685
FU RFD, mm	2.86 ± 0.64	2.64 ± 0.44	0.099
FU in-lesion MLD, mm	2.08 ± 0.56	1.99 ± 0.49	0.473
FU in-segment MLD, mm	2.01 ± 0.57	1.92 ± 0.47	0.463
FU in-lesion diameter stenosis, %	18.6 ± 14.2	19.1 ± 13.1	0.883
FU in-lesion area stenosis, %	30.3 ± 20.4	32.3 ± 19.0	0.676
In-lesion binary restenosis	1 (2.9)	3 (8.1)	0.647
In-lesion LLL negative	18 (51.4)	13 (35.1)	0.163
In-segment LLL negative	21 (60.0)	12 (32.4)	0.019

Values are mean ± SD, median (IQR), or n (%).

FU = follow-up; LLL = late lumen loss; MLD = minimal luminal diameter; PCI = percutaneous coronary intervention; RFD = reference diameter; other abbreviations as in Table 1.

表四

TABLE 4 Quantitative Coronary Angiography: Primary Endpoint LLL Noninferiority Testing Intention-to-Treat Analysis

	PCB (36 Lesions)		SCB (37 Lesions)		Mean Difference (PCB − SCB)		Threshold for Noninferiority
	LS Mean	95% CI	LS Mean	95% CI	Difference	95% CI	
In-lesion LLL, mm	0.05	−0.08 to 0.17	0.11	−0.01 to 0.23	0.06	−0.11 to 0.24	<0.35
In-segment LLL, mm	0.01	−0.10 to 0.13	0.10	−0.01 to 0.21	0.08	−0.07 to 0.24	<0.35

LLL = late lumen loss (primary efficacy endpoint); LS = least squares; other abbreviations as in Table 1.

表五

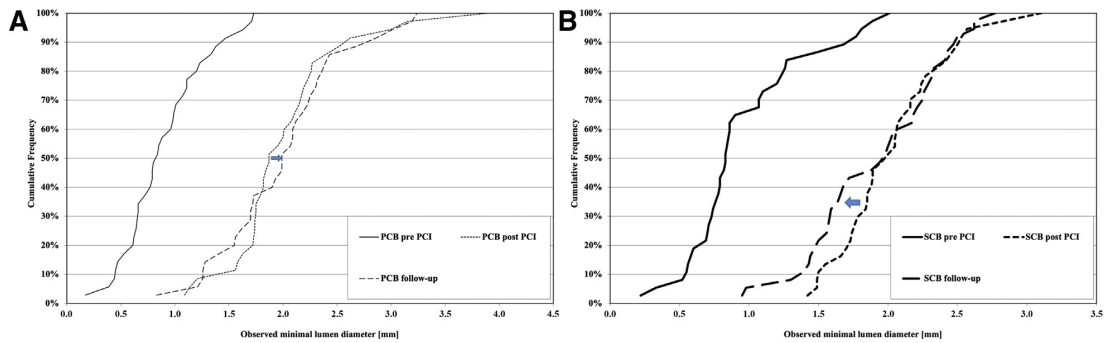
TABLE 5 Clinical Follow-Up at 1 Year: Intention-to-Treat Analysis

	PCB (n = 35)	SCB (n = 35)	*P* Value
TLR	0	0	1.000
Stent thrombosis	0	0	1.000
Death	2 (6)	0	0.493
TV MI	0	0	1.000
Unscheduled angiography	2 (6)	3 (9)	1.000
MACE	2 (6)	0	0.493

MACE = major adverse cardiovascular events (cardiac death, target vessel myocardial infarction, or clinically driven target lesion revascularization); MI = myocardial infarction; TLR = target lesion revascularization; TV = target vessel; other abbreviations as in Table 1.

圖二

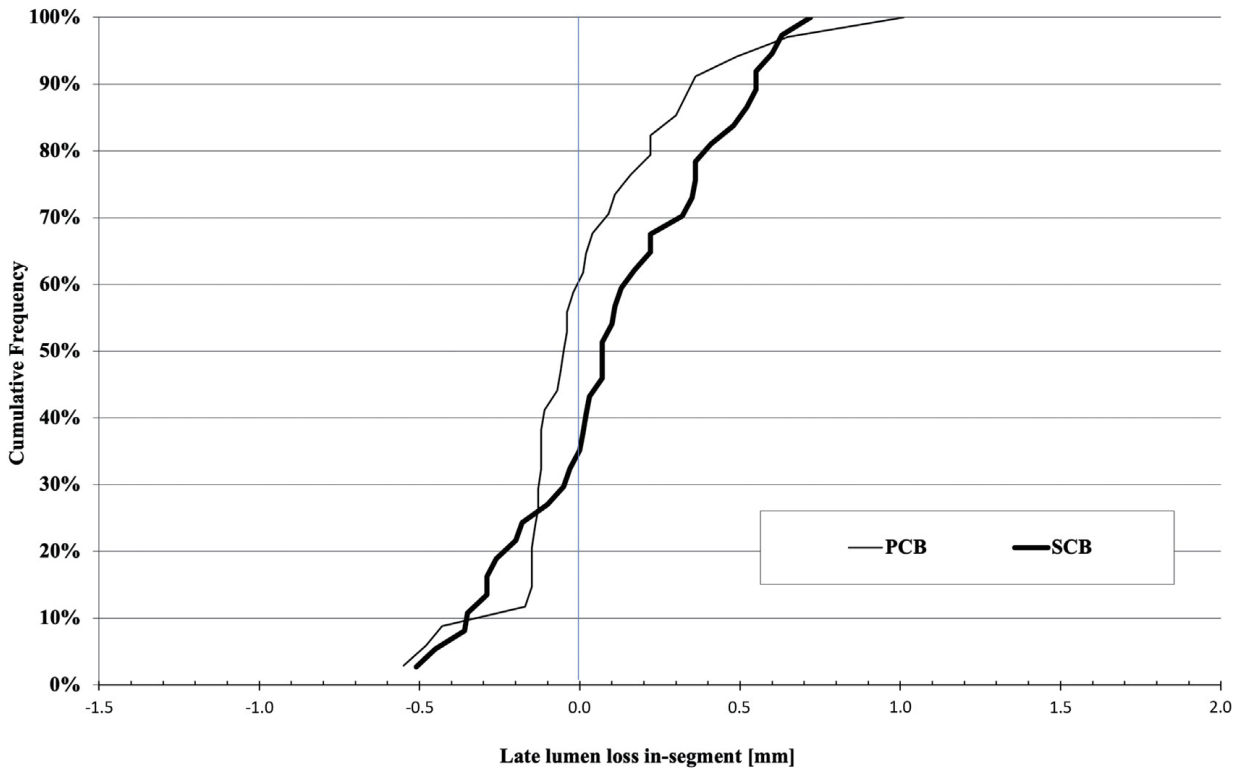
FIGURE 2 Angiographic Patency: Cumulative Frequency Distribution of In-Segment Minimal Luminal Diameter Determined Using Quantitative Coronary Angiography



(A) PCB before percutaneous coronary intervention (PCI), post-PCI, and at 6-month follow-up. **(B)** SCB pre-PCI, post-PCI, and at 6-month follow-up. Abbreviations as in Figure 1.

圖三

FIGURE 3 Angiographic Patency: Cumulative Frequency Distribution of In-Segment Late Lumen Loss Determined Using Quantitative Coronary Angiography



PCB versus SCB. Abbreviations as in Figure 1.

討論

目前歐洲指引中塗藥氣球對於支架內再狹窄有充足的證據，但不建議應用在原發性病灶的治療。因此，最近幾年有愈來愈多的隨機對照試驗支持單獨使用塗藥氣球治療的概念，其定性標準為病灶經事前處理之後，沒有影響血流的血管剝離 (Flow-limiting Dissection, Types A and B)，狹窄不超過 30% 的血管。此外，塗藥氣球在高出血風險的病人、分岔病灶的側支治療上能有益處。在急性冠心症中，單獨使用塗藥氣球似乎亦安全有效。

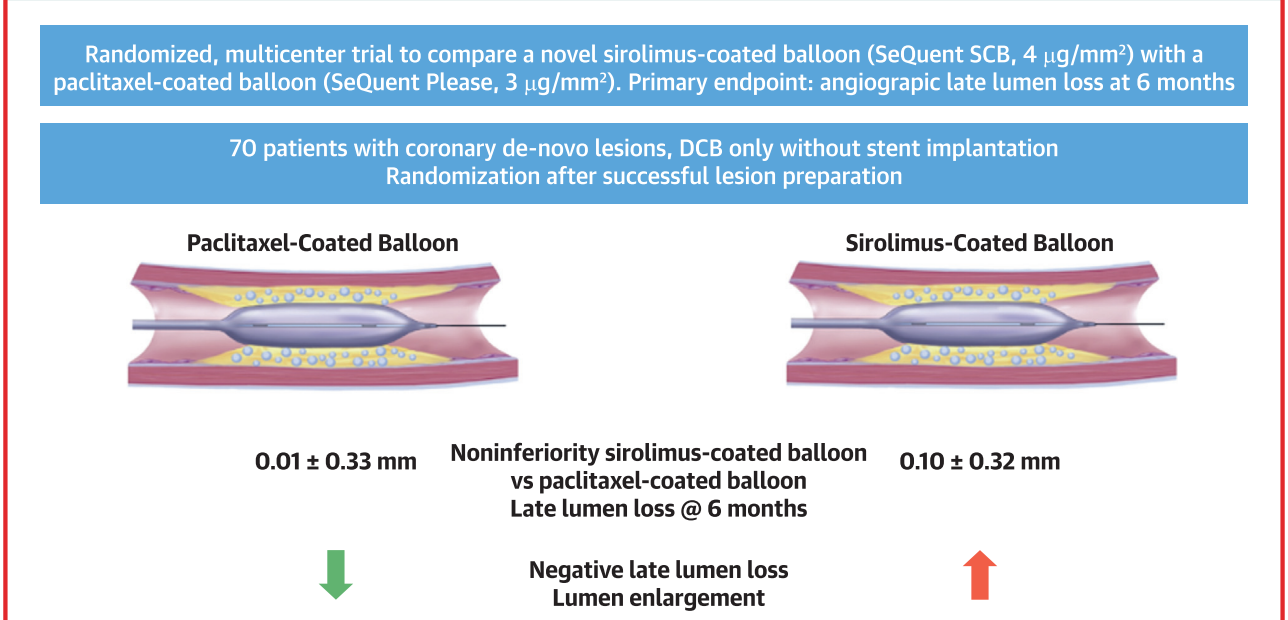
藥物分布在血管壁的方式不同，塗藥氣球和塗藥支架使用後對於組織的藥物濃度也不同。迄今仍不清楚 PCB 治療原發性病灶之後，源自於模仿早期動脈粥樣硬化的格拉戈夫效應 (Glagov Effect) 出現的管腔擴大現象，是否同樣可見於 Sirolimus，畢竟這兩種藥物在血管壁橫向分佈的情形非常不同。紫杉醇僅在 >100 ng/mg 的組織濃度下才能觀察到細胞毒性作用。單獨使用塗藥氣球的治療中，僅在應用後最初幾個小時內能看到這種濃度，長期觀察組織濃度則相對低很多。本試驗亦觀察到紫杉醇的細胞抑製作用而非細胞毒性作用，目前的研究中 PCB 組約有三分之二的病人在追蹤時發現有管腔擴大，SCB 組則有約三分之一，和傳

統的氣球擴張術差不多，未來仍需要更多研究來決定哪種藥物適合塗藥氣球應用在原發性病灶。

結論

這是首次研究治療人體冠狀動脈原發性病灶，比較一種具有結晶塗層的新型 SCB (Novel SCB) 與已經臨床驗證的 PCB (Clinically Proven PCB) 相比的臨床結果。比較結果顯示，兩者在冠狀動脈原發性病灶的治療中，血管造影結果相似。novel SCB 和 PCB 的研究，呈現出相似的血管攝影和臨床結果，但 PCB 治療的這組病人更常觀察到晚期管腔擴大 (Late Lumen Enlargement)。

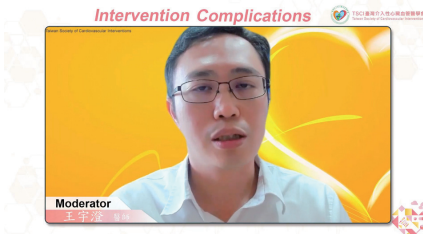
CENTRAL ILLUSTRATION **Treatment of Coronary De Novo Lesions With a Sirolimus- or Paclitaxel-Coated Balloon**

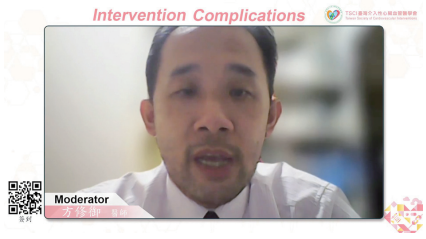


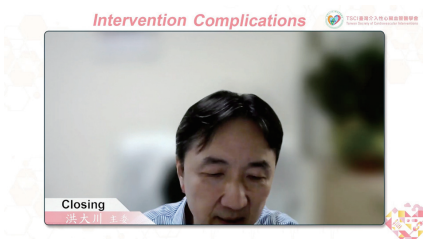
Ahmad, W.A.W. et al. J Am Coll Cardiol Intv. 2022;■(■):■-■.

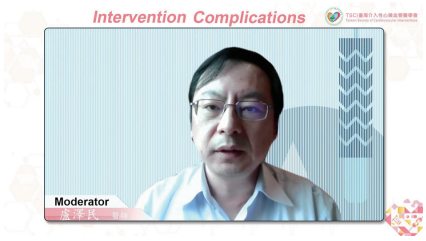
DCB = drug-coated balloon; SCB = sirolimus-coated balloon.

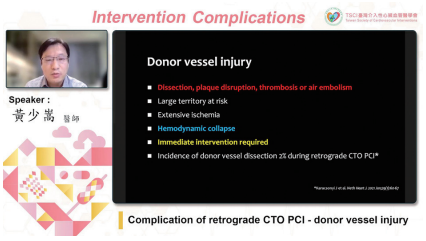
活動集錦－111.5.28_Intervention Complications

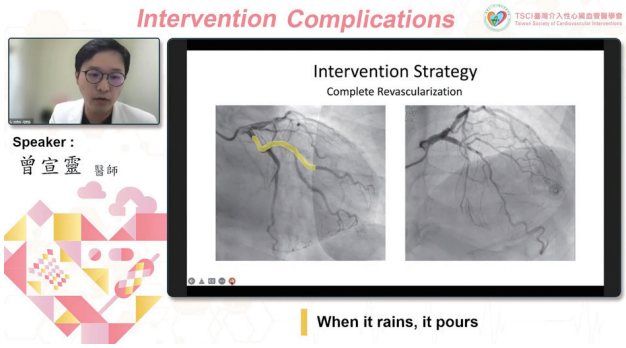


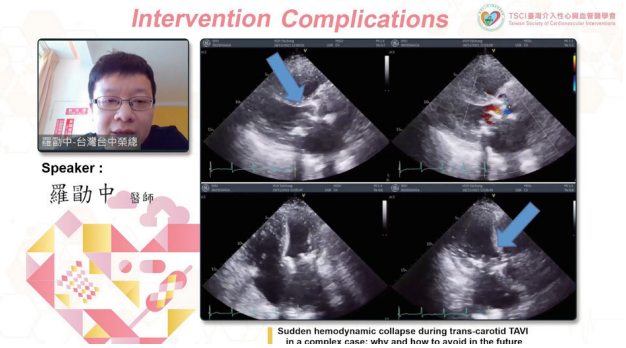


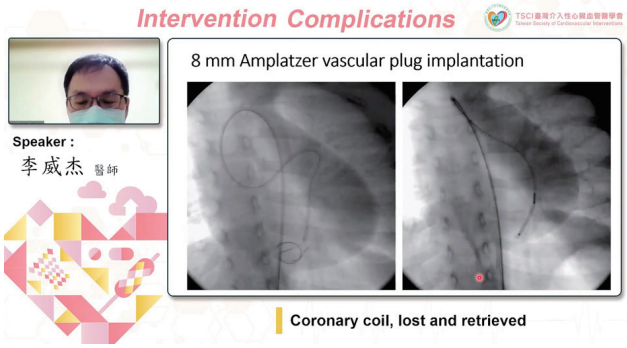


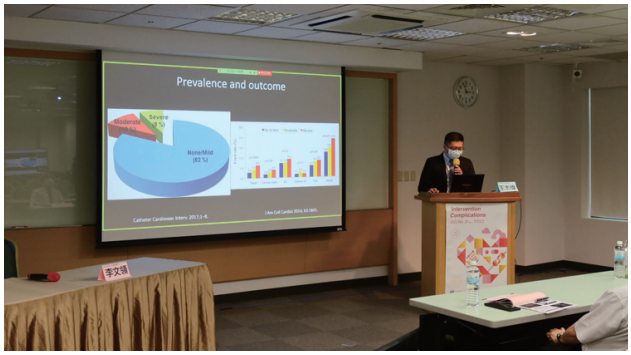






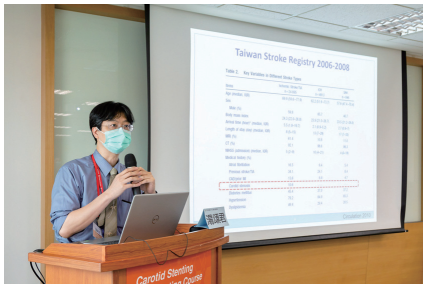






活動集錦-111.6.18_Carotid Stenting Cetificating Course頸動脈支架術認證課程











PAD Update
Opening

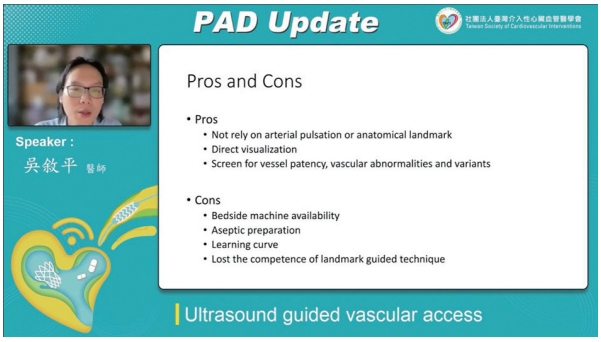

PAD Update
Moderator

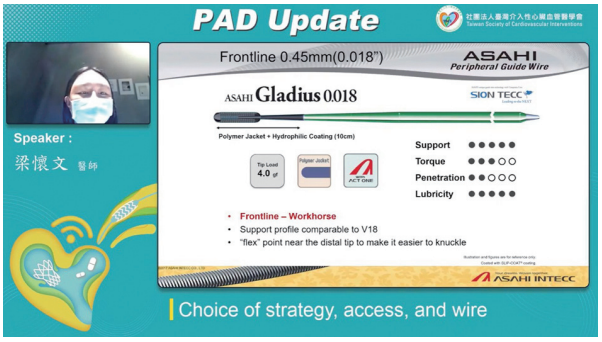

PAD Update
Moderator

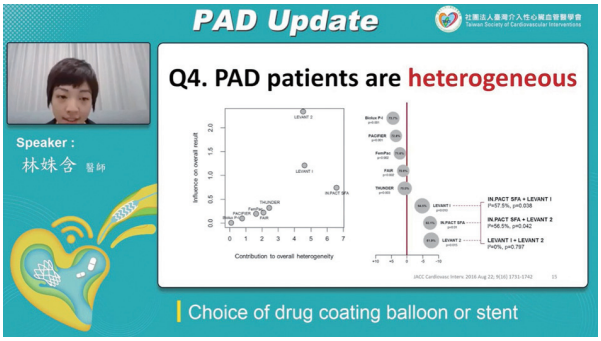

PAD Update
Moderator

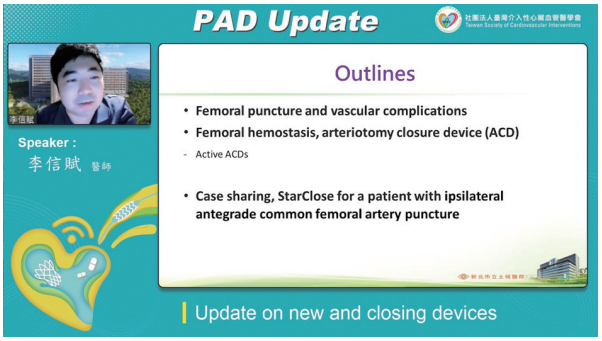

PAD Update
Moderator

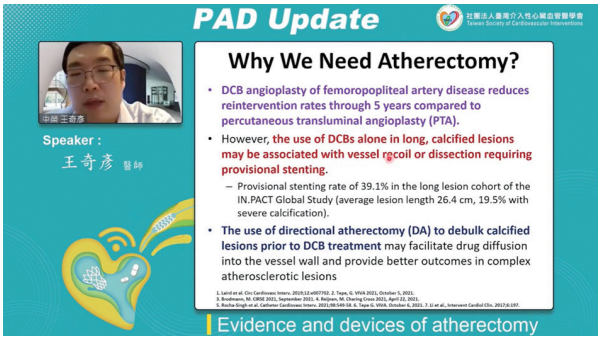

PAD Update
Closing

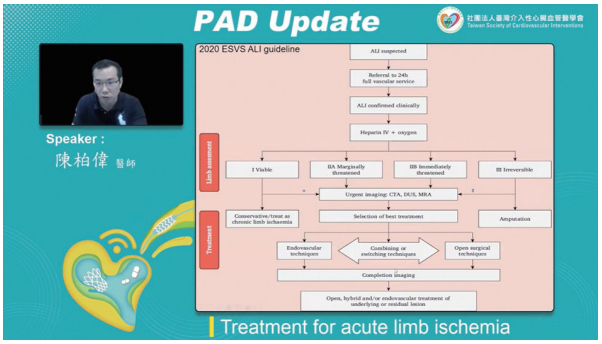

PAD Update
Speaker :
吳敘平 醫師
Pros and Cons
• Pros
• Not rely on arterial pulsation or anatomical landmark
• Direct visualization
• Screen for vessel patency, vascular abnormalities and variants
• Cons
• Bedside machine availability
• Aseptic preparation
• Learning curve
• Lost the competence of landmark guided technique
Ultrasound guided vascular access

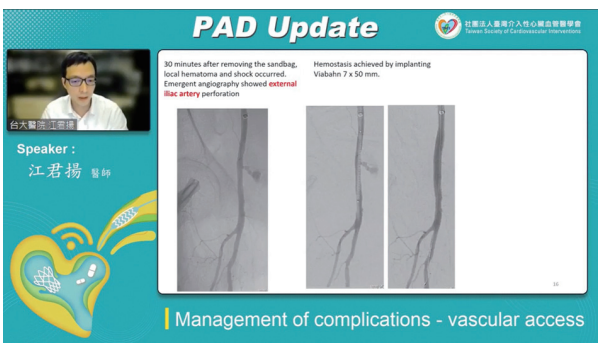

PAD Update
Speaker :
梁懷文 醫師
Frontline 0.45mm(0.018")
ASAHI
Peripheral Guide Wire
ASAHI Gladius 0018
SION TECC
Polymer Jacket + Hydrophilic Coating (10cm)
Support
Torque
Penetration
Lubricity
• Frontline – Workhorse
• Support profile comparable to V18
• "flex" point near the distal tip to make it easier to knuckle
ASAHI INTECC
Choice of strategy, access, and wire

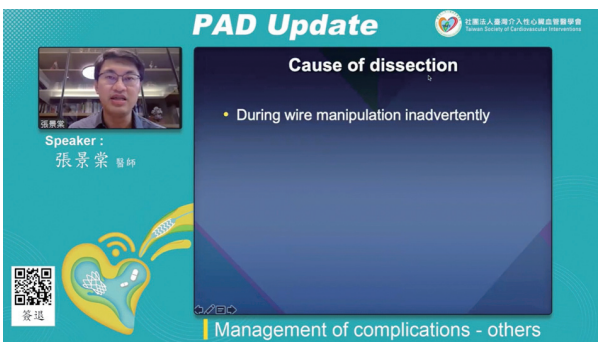

PAD Update
Speaker :
林姝含 醫師
Q4. PAD patients are heterogeneous
Influence on overall result
Contribution to overall heterogeneity
IN.PACT SFA + LEVANT I
P=57.9%, p=0.038
IN.PACT SFA + LEVANT 2
P=56.5%, p=0.042
LEVANT I + LEVANT 2
P=0%, p=0.797
Choice of drug coating balloon or stent


PAD Update
Speaker :
李信賦 醫師
Outlines
• Femoral puncture and vascular complications
• Femoral hemostasis, arteriotomy closure device (ACD)
- Active ACDs
• Case sharing, StarClose for a patient with ipsilateral antegrade common femoral artery puncture
Update on new and closing devices


PAD Update
Speaker :
王奇彥 醫師
Why We Need Atherectomy?
• DCB angioplasty of femoropopliteal artery disease reduces reintervention rates through 5 years compared to percutaneous transluminal angioplasty (PTA).
• However, the use of DCBs alone in long, calcified lesions may be associated with vessel recoil or dissection requiring provisional stenting.
– Provisional stenting rate of 39.1% in the long lesion cohort of the IN.PACT Global Study (average lesion length 26.4 cm, 19.5% with severe calcification).
• The use of directional atherectomy (DA) to debulk calcified lesions prior to DCB treatment may facilitate drug diffusion into the vessel wall and provide better outcomes in complex atherosclerotic lesions
Evidence and devices of atherectomy


PAD Update
Speaker :
陳柏偉 醫師
2020 ESVS ALI guideline
ALI suspected
Referral to 24h full vascular service
ALI confirmed clinically
Heparin IV + oxygen
Limb assessment
I Viable
IIA Marginally threatened
IIB Immediately threatened
III Irreversible
Urgent imaging: CTA, DUS, MRA
Treatment
Conservative/treat as chronic limb ischaemia
Selection of best treatment
Amputation
Endovascular techniques
Combining or switching techniques
Open surgical techniques
Completion imaging
Open, hybrid and/or endovascular treatment of underlying or residual lesion
Treatment for acute limb ischemia


PAD Update
Speaker :
江君揚 醫師
30 minutes after removing the sandbag, local hematoma and shock occurred. Emergent angiography showed external iliac artery perforation
Hemostasis achieved by implanting Viabahn 7 x 50 mm.
Management of complications - vascular access


PAD Update
Speaker :
張景棠 醫師
Cause of dissection
• During wire manipulation inadvertently
Management of complications - others

INFORMATION FOR AUTHORS

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Journals

1. Xu J, Cui G, Esmailian F, et al. Atrial extracellular matrix remodeling and the maintenance of atrial fibrillation. *Circulation* 2004;109:363-8.
2. Boos CJ, Lip GY. Targeting the renin-angiotensin-aldosterone system in atrial fibrillation: from pathophysiology to clinical trials. *J Hum Hypertens* 2005;19:855-9.

Books

1. Gotto AJ, Farmer JA. Risk factors for coronary artery disease. In: Braunwald E, Ed. *Heart Disease: A Textbook of Cardiovascular Medicine.* 3rd ed. Philadelphia: Saunders, 1988:1153-90.
2. Levinsky NG. Fluid and electrolytes. In: Thorn GW, Adams RD, Braunwald E, et al, Eds. *Harrison's Principles of Internal Medicine.* 8th ed. New York: McGraw-Hill, 1977:364-75.

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Number figures in the order in which they appear in the text. Figure legends should correspond to figure/illustration numbers and appear on a separate sheet of paper. Prepare your figures according to your mode of submission:
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Regular Mail: Photographs and drawings should be unmounted, glossy prints, 5"×7" in size. Three sets of each illustration must be submitted in a separate envelope. Label the back of each figure with the title of the article and an arrow indicating the top of the figure.

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