Summer 2024

GP Connect

Supporting best practice in cardio-metabolic health

Sydney Cardiology

From the editor – Dr Gunjan Aggarwal

Specialising in general adult cardiology and non-invasive cardiac imaging, particularly echocardiography and cardiac computed tomography (CT).

Welcome to the summer edition of GP Connect 2024. This issue provides the latest clinical guidance on management of hypertension and myocardial ischemia with no obstructive coronary artery disease (INOCA) for General Practitioners.

Hypertension is an enormous clinical problem and undertreatment is a concern that can lead to hypertension mediated organ damage (HMOD). Dr James Wong provides an excellent succinct summary of the latest 2023 European Society of hypertension guidelines with an emphasis on tools available to general practitioners for the diagnosis of hypertension as well as the assessment of HMOD to aid effective treatment.

Dr Rudee Ting outlines the management of a common clinical condition called INOCA which can encompass a number of conditions such as vasospasm and microvascular angina. Diagnosis is often challenging as it requires additional specialised testing beyond routine cardiac catheterisation. However prompt identification and treatment is important to address the heightened risk of cardiovascular events and improve quality of life.

I hope you enjoy this edition of GP Connect. We remain available as always to provide continued care to you and your patients in any way possible.

Thank you for your continued support,

Dr Gunjan Aggarwal.

In this issue

Access past issues

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All Sydney Cardiology clinics have emergency appointments available for same-day referrals.

Contact your local clinic directly for assistance.

Update on hypertension

Dr James Wong

Specialising in general cardiology, prevention of coronary artery disease and hypertension.

The 2023 European Society of Hypertension Guidelines for the management of arterial hypertension are the most comprehensive and useful hypertension guidelines that I have seen and I would recommend that you download this resource to use on your desktop for everyday practice.

Click here to access.

The ESH guidelines aim to serve the clinician by summarizing the best available evidence for all aspects of hypertension management and provide recommendations for treatment based on level of evidence, with priority given to patient-important cardiovascular outcomes including stroke, myocardial infarction, heart failure, end stage kidney disease and cardiovascular and total mortality.

While it is long (152 pages), it is well set out in 22 sections which make it easier to read. It is also easy to find relevant information about hypertension in the various (and Immense) clinical settings and what is considered effective treatment in these specific situations. The best place to start is to navigate the Table of Contents which will direct you to your place of interest or concern.

Diagnosis

Blood pressure readings may be very variable and there is often dissent about the diagnosis of hypertension between patient and doctor which results in delay in treatment or nontreatment.

It is now clear that there is difficulty in blood pressure measuring methodology with the office BP being often inaccurate and patients being unconvinced about elevated blood pressure being significant. Ambulatory and Home blood pressure measurements (out of office) are recommended if diagnosis is not clear to establish or refute the diagnosis of hypertension. A further benefit of home blood pressure monitoring (HBPM) is the involvement of the patient in self-care with advantage of improved adherence to treatment and better hypertensive control. BP measurement and monitoring is covered at length.

The guidelines also discuss hypertension phenotypes including Masked Hypertension (MH), White Coat Hypertension (WCH), their respective uncontrolled phenotypes (MUCH and WUCH) and also current recommendations for treatment. No randomised controlled trials have been performed on MH but given the adverse prognostic significance of out-of-office BP elevations, it appears appropriate to consider BP lowering therapy in those at high cardiovascular risk and those with hypertension mediated organ damage (HMOD). Whether or not patients with WCH should receive BP lowering drugs is still unresolved but treatment may be considered in patients with HMOD and a high cardiovascular risk.

There is information on nocturnal hypertension and the associated increased risk of HMOD and adverse outcomes with this hypertensive phenotype. Possible mechanisms of night-time hypertension are described. Treatment is not evidence-based presently.

Undertreatment

The problem with hypertension management is undertreatment, not overtreatment. The consequence of this is the ongoing large burden of cardiovascular morbidity and mortality that we encounter and risks of other end organ damage including dementia that is untreatable.

Prevention of end organ damage is the key.

As hypertension is silent until complications occur, patients are often not aware of the dangers and do not seek treatment to reduce lifetime risk.

The assessment of hypertension mediated organ damage (HMOD) is recommended:

Update on hypertension (continued)

Table 1. Assessment of hypertension-mediated organ damage (HMOD)*

BASIC SCREENING TESTS FOR HMOO RECOMMENDED FOR ALL HYPERTENSIVE PATIENTS	AIM
12 lead ECG	Measure HR and AV conducbon, detect cardiac arrhythmias, myocardial ischemia and infarction, screen for LVH
Urine albumin: creatinine rabo (UACR)	Detect and classify CKD
Serum creatmine and eGFR	Detect and classify CKD
Extended screening for HMOD	
Echocardiography	Evaluate structure and function of the ventricles and left atrium, detect valvular disease. aortic root diameter and ascending aortic aneurysm
cfPWV or baPWV	Evaluate aortic/large artery stiffness
Carotid artery ultrasound	Determine carotid intima·media thickness, plaque and stenosis
Coronary artery calcium scan	Determine the presence and extent of coronary calcium to predict CAD events
Abdominal aorta ultrasound	Screen for aortic aneurysm
Kidney ultrasound	Evaluate size and structure of kidney, detect renovascular disease. determine RRI (by spectral doppler ultrasonography)
Spectral doppler uhrasonography	Diagnosis of renovascular disease and determination of RRI
ABI	Screen for LEAD
Retina microvasculature	Detect microvascular changes
Cognitrve function testing (MMSE, MoCA)	Screen for early stages of dementia
Brain imaging (CT, MRI)	Detect structural brain damage

*can be adapted according to the clinical cifcumstance.

Update on hypertension (continued)

2023 ESH Guidelines for the management of arterial hypertension

- HMOD refers to structural or functional changes in arteries or end organs (brain, heart, kidney and eyes) caused by elevated BP and is a marker of preclinical or asymptomatic disease.
- Unless treated, target organ disease gradually progresses from asymptomatic to symptomatic leading to overt cardiovascular events.
- Cardiovascular risk increases in the presence of HMOD and more so when damage increases progressively and affects multiple organs and functions.
- Detected asymptomatic HMOD may also reveal undiagnosed hypertension – missed or masked hypertension.
- Some types of HMOD can be reversed by antihypertensive treatment, especially when treatment starts early, but with longstanding hypertension, HMOD may become irreversible despite BP control. Nevertheless BP lowering treatment is important as it may delay further progression of HMOD and oppose the trend toward a progressive increase in cardiovascular risk.
- It is recommended that basic screening for HMOD be performed in all hypertensive patients and that a more detailed assessment be implemented when the presence of HMOD is important for treatment decisions.

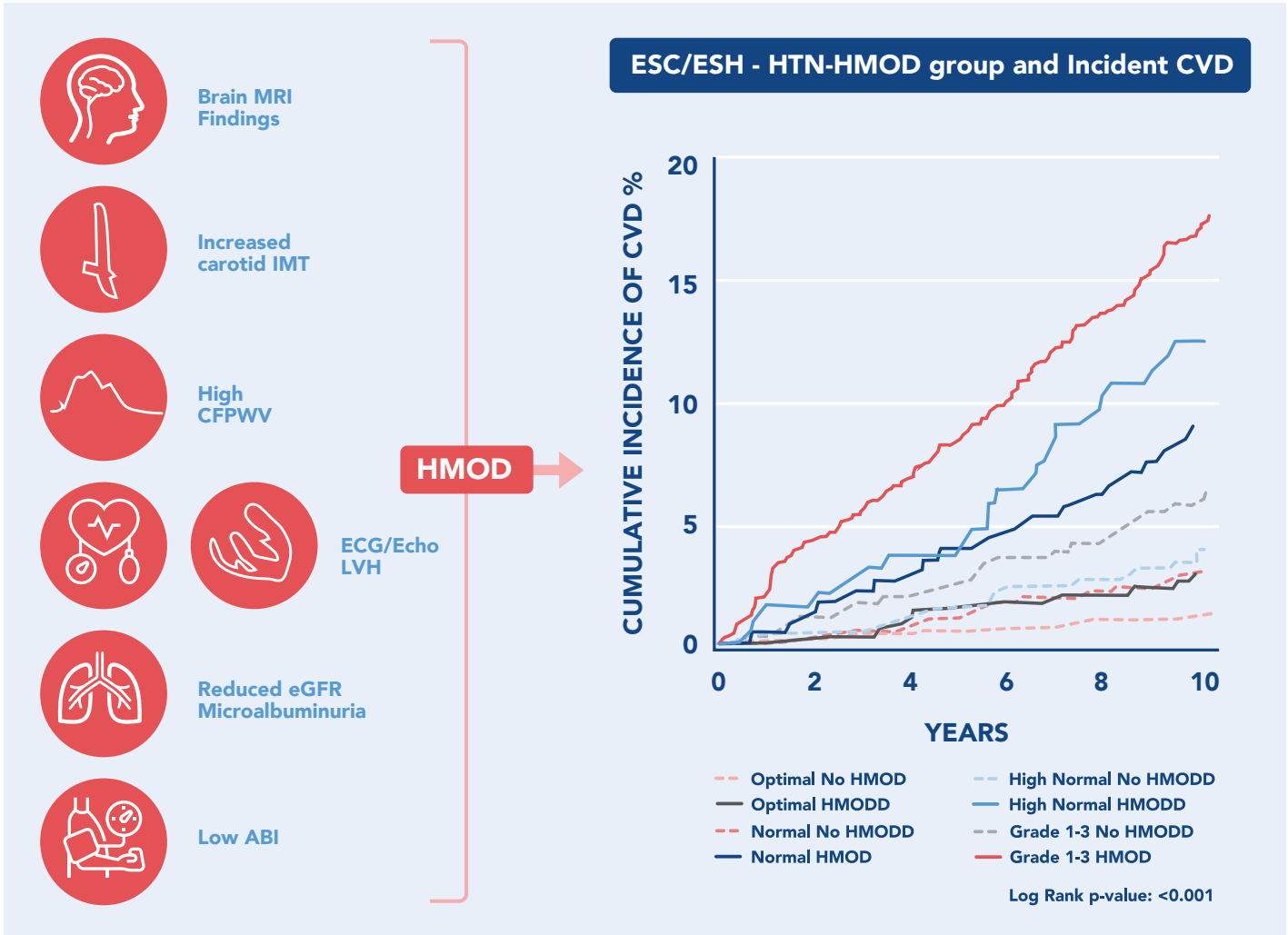


Figure 1. Presence of HMOD increased CVD risk at each BP level

Update on hypertension (continued)

It is recommended that basic screening for HMOD be performed in all hypertensive patients and that a more detailed assessment be implemented when the presence of HMOD is important for treatment decisions. HMOD in the heart may be picked up with transthoracic echocardiography which is readily available and a cost effective technique to assess left ventricular hypertrophy (LVH). LVH is recognized as an important predictor of adverse events and significantly increases cardiovascular risks. Effective treatment can be accompanied by a regression of LVH which seems to predict a better prognosis. The echo also provides useful evaluation of LV geometry, LA and aortic dimensions and LV diastolic and systolic function. Regression of LVH with treatment Is associated with a reduced risk of cardiovascular outcomes.

Coronary artery calcium scoring (CACS) is also available to detect coronary artery disease and can be performed at low cost with CT using low radiation. It is being used increasingly to stratify cardiovascular risk and to better gauge who may benefit from early initiation of BP medications as well as statin therapy.

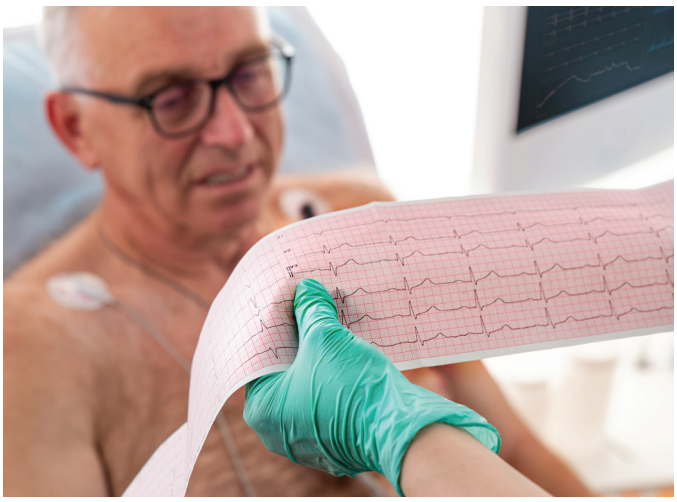
Hypertension is well known as a cause of acute stroke, ischaemic and haemorrhagic, and also TIA and there is also chronic and asymptomatic damage leading to cognitive decline and dementia. HMOD in the brain is well covered in section 5.5.4 and is essential reading.

The importance of HMOD assessment in cardiovascular and kidney risk stratification is emphasized to aid effective treatment. It is also relevant during follow up to evaluate the efficacy of therapy. A reduction in previous HMOD may indicate the success of the therapy while the persistence or increment of HMOD may indicate need to review treatment, with lack of adherence being a predominant reason.

In its 2023 global report on hypertension, the World Health Organization estimated that 1.3 billion adults worldwide have hypertension with 46% of adults with hypertension being unaware of the condition. Less than half of adults with hypertension are diagnosed and treated. Approximately 1 in 5 adults (21%) with hypertension have it under control.

The Australian rates of BP control are 38% in women and 28% in men which is concerning. There is evidence that we are falling behind other developed countries – USA (54% women, 49% men) Germany (58% women 48% men) Canada (50% women 68% men) and UK (37% women 37% men).

There is much commentary and lamentation about suboptimal BP control and the need to implement the knowledge and strategies for improved control. Nonadherence to treatment is high.



My suggested approach

1. Encourage awareness and understanding of BP management in your patient at risk
2. involving your patient in BP measurement (HBP) and giving the responsibility (empowerment) to the patient in managing his/her condition.
3. Assessment of HMOD to stratify risk, to inform the patient, and assist management.
4. A shared and agreed-upon treatment strategy which would include lifestyle intervention and drug treatment.
5. Follow up is crucial to ascertain BP control and to ensure satisfactory outcomes.

References: 1. 2023 ESH Guidelines for the management of arterial hypertension. Journal of Hypertension 2023, 41:000 **2.** Hypertension News Sep2023 **3.** YouTube Hypertension Guidelines 2023 **4.** Editors' Commentary on the 2023 ESH Management of Arterial Hypertension Guidelines Hypertension 2023;80:1795 **5.** WHO Hypertension 16 March 2023 **6.** Hypertension 2022:79:505-515 **7.** Heart Foundation Heart, Lung and Circulation (2023) 32,881 1443

Our team

We have experienced cardiologists in all major sub-specialties to provide the highest quality of patient care. We also have specialists in related fields including endocrinology and respiratory medicine. Our Sydney Cardiology team includes:

Cardiology

Dr James Wong

Specialising in general cardiology, prevention of coronary artery disease and hypertension.

Dr Abhinav Luhach

Specialising in general adult cardiology, cardiac CT, and preventive cardiology.

Dr Gunjan Aggarwal

Specialising in general adult cardiology and non-invasive cardiac imaging, particularly echocardiography and cardiac CT.

Dr Andrew Terluk

Specialising in general cardiology with an interest in cardiomyopathy in the setting of cancer.

Dr Ru-Dee Ting

Specialising in general and interventional cardiology, including cardiac haemodynamic studies and complex coronary intervention.

Dr Fiona Foo

Specialising in general and interventional cardiology with an interest in heart disease affecting women and sports cardiology.

Dr Bill Petrellis

Specialising in general adult cardiology and electrophysiology, including atrial fibrillation and device implantation.

A/Prof Martin Brown

Specialising in advanced heart failure, pulmonary hypertension, and transplant cardiology.

Endocrinology

Dr Suja Padmanabhan

Specialising in diabetes and general endocrinology with a special interest in diabetes in pregnancy and women's health.

Respiratory Medicine

Dr Tracy Smith

Respiratory and sleep physician specialising in respiratory disease with a special interest in respiratory failure due to lung or heart disease.

INOCA

Dr Ru-Dee Ting

Specialising in general and interventional cardiology, including cardiac haemodynamic studies and complex coronary intervention.

Ischaemic heart disease is a major cause of morbidity and mortality. Coronary atherosclerosis is a well recognised cause but patients with angina symptoms and no obstructive coronary lesions may have a condition known as Ischaemia with No Obstructive Coronary Artery disease (INOCA). The 2021 ACC/AHA chest pain guidelines redefined coronary disease to include any degree of coronary stenosis, including INOCA.[1]

INOCA encompasses coronary vasospasm, microvascular angina and microvascular spasm. These conditions can be underdiagnosed because traditional coronary angiography is not able to adequately detect these disorders.

Coronary vasomotion disorders

The epicardial coronary arteries run along the surface of the heart and can be seen macroscopically on angiography. Epicardial coronary spasm results in vasospastic angina (also known as variant angina or Prinzmetal angina). Microvascular angina can be caused by microvascular spasm but can also involve impaired coronary vasodilatation or microvascular obstruction (resulting in increased microvascular resistance). These disorders result in myocardial ischaemia due to a reduction in myocardial perfusion.

Coronary vasomotion disorders can occur in conjunction with traditional epicardial stenoses, and current diagnostic testing is limited in these situations. Invasive testing for vasomotion disorders is most useful in patients with no epicardial obstruction.[2]

Diagnosis

Traditional coronary angiography involves obtaining arterial access (usually via radial or femoral artery) and passing a catheter into the aortic root and engaging the coronary arteries. Contrast dye is then injected directly into the coronary arteries and fluoroscopic images obtained. A coronary angiogram does not assess coronary vascular function and does not visualise the coronary arterioles or capillaries (Fig 1). Coronary angiography has a spatial resolution of about 0.3mm which is inadequate to image the microvasculature. There are clear guideline recommendations regarding coronary angiography in patients with suspected epicardial disease. However, many guidelines do not emphasise the additional step of testing of coronary vascular function resulting in clinicians not assessing for these conditions and resulting in diagnostic uncertainty.

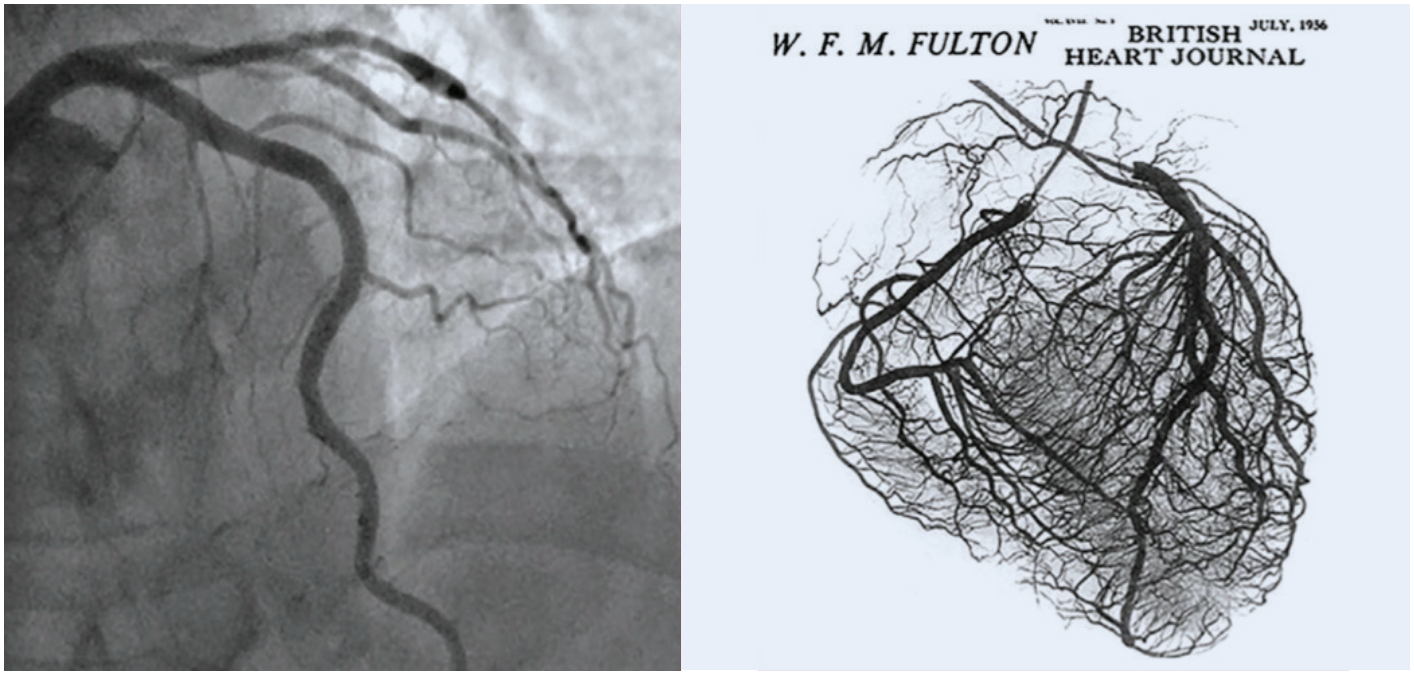


Figure 1. Left panel: Coronary angiogram visualising epicardial arteries, resolution to 0.3mm; Right panel: Ex vivo arteriogram demonstrating coronary microvasculature, resolution 0.03mm.[2]

INOCA (continued)

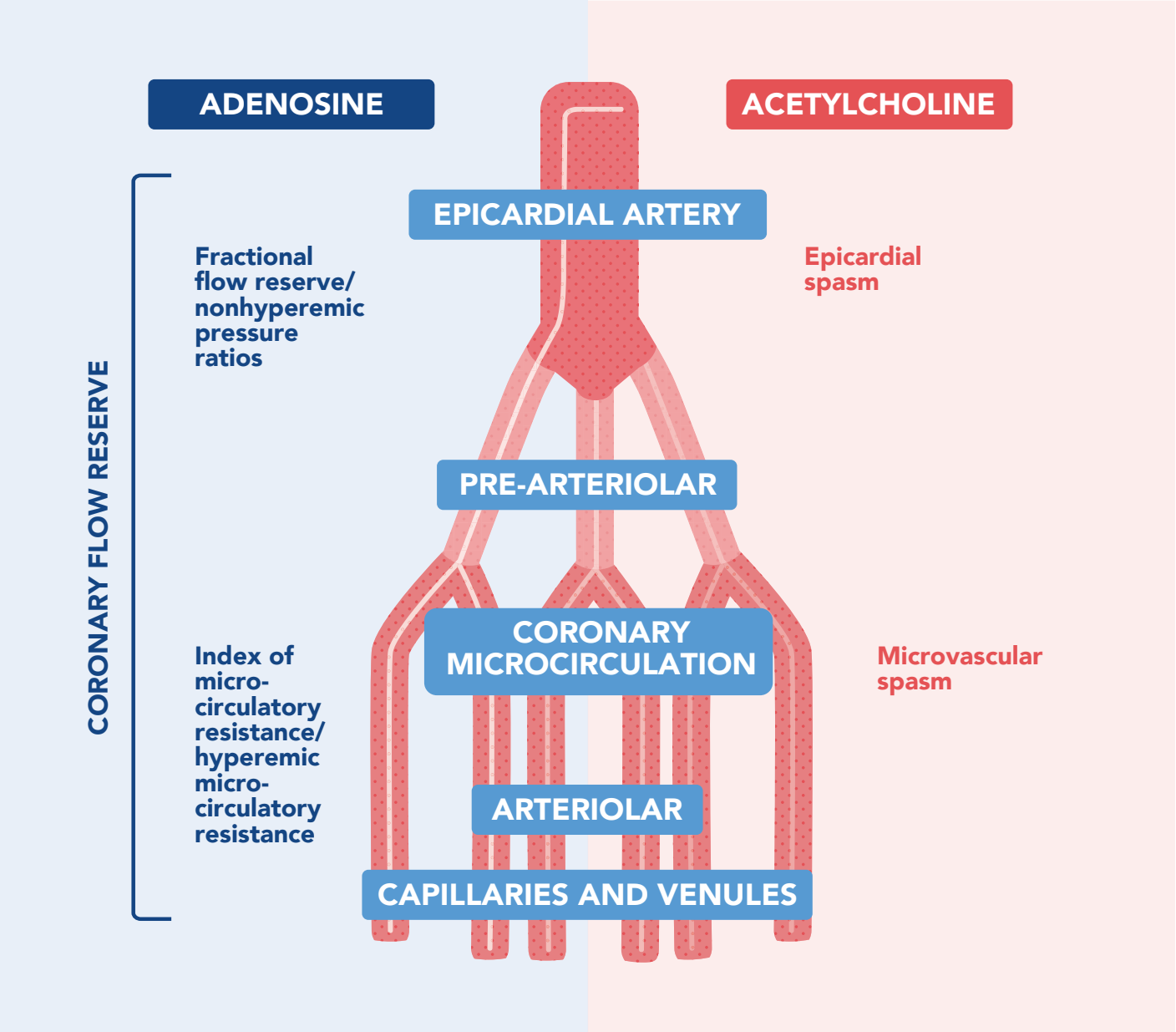


Figure 2. Functional testing of coronary arteries at different levels of the vascular tree.[2]

In patients in whom INOCA is suspected, adjunctive tests performed at the time of coronary angiography should be considered. Spasm can be diagnosed with an acetylcholine (ACh) challenge and microvascular obstruction can be diagnosed with measurement of coronary flow reserve (CFR) and the index of microvascular resistance (IMR).

Intracoronary ACh can be delivery via the coronary catheters and coronary angiography performed to look for spasming of the arteries in real time. CFR and IMR are measured using specialised coronary wires that are passed down into the coronary arteries and measure intracoronary pressures which can then be imputed to flow. Calculation of fractional flow reserve (FFR), CFR and IMR inform the state of flow at different levels in the coronary arterial tree (Fig 2).

Prognosis

Patients with undiagnosed chest pain are at increased risk of cardiovascular (CV) events for at least 5 years. Women have a greater incidence of INOCA and are more likely to be dismissed from medical care. INOCA is associated with an increased risk of CV events and reduced quality of life. Women with INOCA have a 10-fold increase in heart failure with preserved ejection fraction and stroke.[3] Older age, diabetes, hypertension, and smoking predict worse outcomes with patients with INOCA.[4]

INOCA (continued)

Treatment

Hypertension and dyslipidaemia are associated with coronary microvascular disease and vasospasm. Hypertensive microvasculature impairs perfusion and dyslipidaemia contributes to coronary endothelial dysfunction. Insulin resistance and metabolic syndrome are also associated with microvascular dysfunction. Therefore, lifestyle modification and pharmacological treatment with statins and ACE inhibitors can target coronary endothelial dysfunction.

Statins improve flow mediated dilation in INOCA and have been shown to improve coronary flow reserve and coronary microcirculation. Betablockers are beneficial in microvascular angina, in particular, nebivolol has vasodilatory effects and has demonstrated antianginal effects in patients with INOCA. Calcium channel blockers like diltiazem are useful in coronary spasm and microvascular spasm. They have less effect on improving coronary flow reserve.[5]

Short acting nitrates are beneficial in patients with coronary spasm, but its efficacy is inconclusive in patients with microvascular dysfunction, possibly due to different signalling pathways in the epicardial arteries and micro vessels.[6] Long-acting nitrates have not been shown to be of benefit in microvascular dysfunction but have a role in epicardial coronary disease.[7]

Second line therapies include nicorandil, ivabradine, and trimetazidine.

Summary

INOCA is a difficult entity to diagnose due to its heterogeneity. Patients with chest pain without obstructive coronary disease should be considered for further testing for INOCA. It is important to confirm a diagnosis of INOCA given its association with increased CV events. Excluding INOCA avoids unnecessary medication prescription. Practice guidelines provide defined treatment protocols, and this translates to improve quality of life for patients with these conditions.[8]

References: **1.** M. Gulati et al, Circulation 2021, 144 (22), pp e368-454 **2.** T.J. Ford et al, JACC: Cardiovasc Interv 2020, 13 (16), pp1847-64 **3.** C.L. Schuman et al, JACC Cardiovasc Imaging 2021, 14 (7), pp 1369-79 **4.** M. Gulati et al, Arch Intern Med 2009, 169 (9), pp 843-50 **5.** B. Hansen et al, Current Problems in Cardiology 2023, 48 (1), 101420 **6.** T. Matsumoto et al, J Cardiovasc Pharmacol 1997, 29, pp 772-9 **7.** G. Russo et al, Cardiovasc Drugs Ther 2013, 27, pp 229-334 **8.** J. Knuuti et al, Eur Heart J 2020, 41, pp 407-77

The Top 10 of Obesity

Dr Andrew Terluk

Dr Andrew Terluk specialises in general adult cardiology, with research interests in cardiac echocardiography.

Recent literature has suggested that visceral adipose tissue is the most strongly associated increase in cardiovascular events.

It's a well known fact that almost all westernised countries are suffering under the increasing burden of obesity and, in Australia, this has now overtaken tobacco as the leading preventable health burden.

The following are Dr Andrew Terluk's quick facts on obesity and cardiovascular health.

10. Body Mass Index (BMI) has not been entered into risk factor calculators because it does not offer sufficient value above other traditional risk factors (e.g., those in Framingham).

9. BMI is not a useful tool to determine the extent of visceral fat – waist circumference is better.

8. Visceral fat expansion in post-menopausal women has been postulated to be a mechanism for increased cardiovascular risk during this phase of a woman's life.

7. Retroperitoneal fat has been associated with hypertension. This is thought to be due to a local paracrine effect on renal sodium absorption

6. Increased pericardial fat has been associated with increased ischaemic heart disease, although this remains an area of controversy.

5. Lower body subcutaneous adipose tissue being gluteal, femoral and peripheral leg areas is associated with a lower cardiovascular risk and decreased cancer risk.

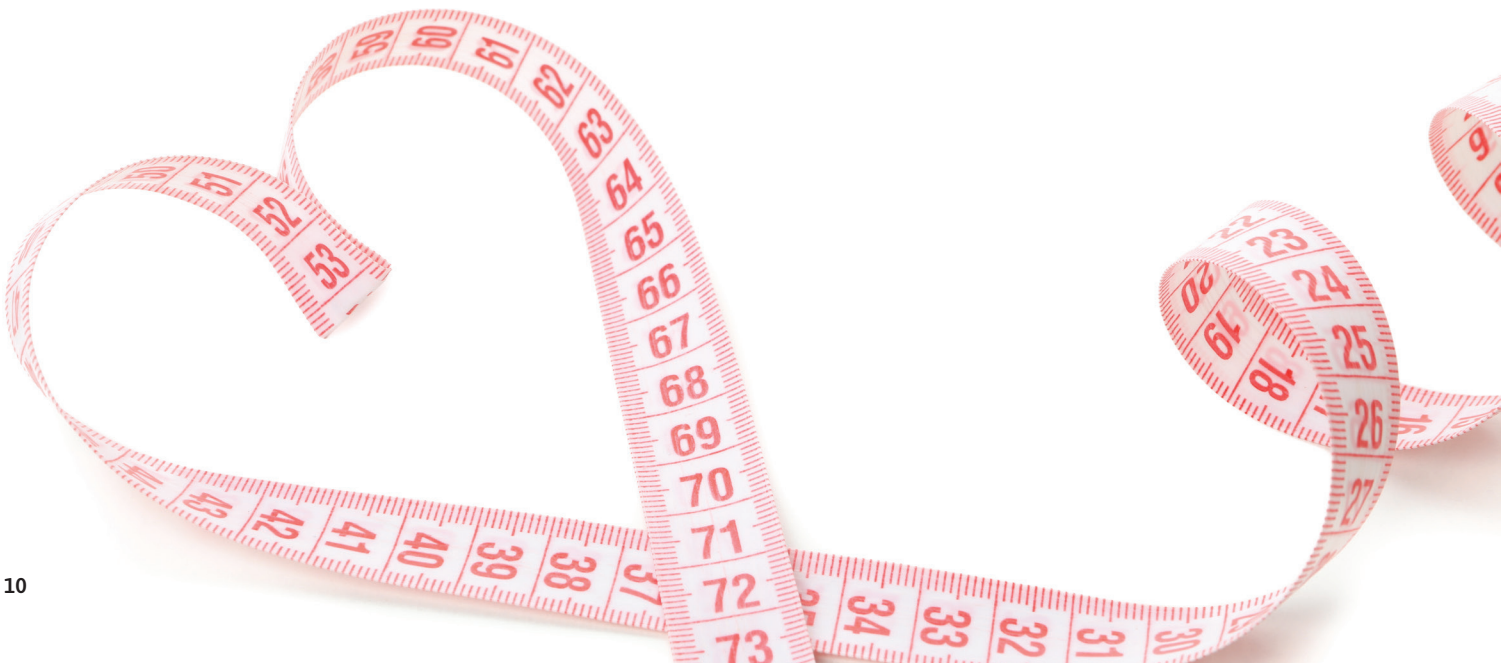
4. The concept of Metabolically healthy obese is controversial and the definition lacks standardization. One large prospective Norweigian study (N = >61000) followed individuals over a 12-year span with no increase in myocardial infarction but with an increase in heart failure among healthy obese.

3. The PREDIMED trial or Mediterranean diet demonstrated a reduction in cardiac events over 4.5 years independent of weight loss.

2. A very low calorie diet in conjunction with aerobic exercise promotes preferential loss of visceral fat.

1. Patients who underwent gastric bypass and gastric sleeve surgery have demonstrated a reduction in visceral fat by 40-50%.

Dr Terluk has previously presented scientific posters nationally and internationally at the Cardiac Society of Australia and New Zealand (Melbourne and Adelaide, AUS) and Transcatheter Cardiovascular Therapeutics (San Francisco, USA). Dr Terluk is currently involved in scientific research, including the echocardiographic assessment of right heart function and cardiac failure in the context of cancer.



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Sydney Cardiology is a world class comprehensive cardiology service, delivered with expertise and experience. Using state of the art diagnostic equipment in all five clinic locations, Sydney Cardiology strives to provide exemplary outcomes for long term patient care.

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Including stress-echocardiography, echocardiography, holter monitor studies, ambulatory blood pressure studies, coronary calcium score, dobutamine stress echo, electrocardiogram and event monitor recording.

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We provide echo-only, ABP-only, and holter monitor-only referral services, with a summary report on any adverse findings.

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Including diagnostic electrophysiology studies, ablation of cardiac arrhythmias, cardiac device implantation, pacemakers and defibrillators, and follow up of implanted cardiac devices.

Cardiac procedures

Including coronary angiography, cardiac biopsies, right heart catheterisation, transesophageal echocardiogram and coronary angioplasty.

Including renal and lower limb angioplasty, ankle brachial index and SphygmoCorR central blood pressure testing.

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Parramatta - Fax: 02 9635 1247

Sydney City - Fax: 02 9422 6081

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In-hospital care

All patients with appropriate private health coverage undergoing hospital procedures, do not incur any out-of-pocket costs. Sydney Cardiology has access to leading private hospitals, including:

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Wahroonga

Norwest Private Hospital
Bella Vista

Macquarie University Hospital
North Ryde

Northern Beach Hospital
Frenchs Forest

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15-17 Kildare Road,
Blacktown NSW 2148

Tel: 02 9422 6050 | Fax: 02 9676 8900

Chatswood

Suite 901, Level 9, Tower B,
799 Pacific Highway,
Chatswood NSW 2067

Tel: 02 9422 6040 | Fax: 02 9411 1904

Parramatta

Level 5 Suite 501, B1 Tower,
118 Church Street,
Parramatta NSW 2150

Tel: 02 9422 6060 | Fax: 02 9635 1247

Sydney City

Suite 1303, Level 13
68 Pitt Street
Sydney NSW 2000

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