http://www.harleystreet.sg/heart
Email **enquiries@harleystreet.sg**

HEART FAILURE AND DIABETES

A DANGEROUS INTERSECTION

The prevalence of Type 2 Diabetes (DM) has increased by 30% globally in the last 10 years, with 435 million diabetics worldwide in 2015. Heart failure (HF) affects at least 26 million people worldwide. The prevalence of both diseases is on the increase, largely due to the aging population. Both diseases commonly occur together, and each increases the risk of developing the other. DM is associated with a 2-3 X increased risk of HF and around 10-30% of patients with DM will have known HF. A recent study showed that 57% of Asian patients with HF also had DM in comparison to 24% of Caucasian patients. DM is therefore 3 times more common in Asian HF patients than in Non-Asian populations.

When the two conditions are present together, patients will have more symptoms and signs, worse quality of life and increased mortality and hospitalization. In addition, patients with both conditions frequently have greater numbers of comorbidities, making their management more complex.

The aetiology of HF in DM
In patients with DM, HF is largely caused by hypertension and coronary artery disease, however, there is increasing evidence that DM has a direct detrimental effect on myocardial function. Insulin resistance leads to left ventricular hypertrophy. Hyperglycemia leads to advanced glycation end products that result in myocardial fibrosis, and there are also detrimental changes in myocardial metabolism. Left ventricular diastolic dysfunction can be detected in 75% of patients with DM and dysfunction develops early in the course of the disease. The severity and duration of hyperglycemia are also related to worsening myocardial function.

Screening for diabetes in patients with heart failure
Given that HF is highly diabetogenic, it is important to ensure screening for glucose abnormalities is performed regularly in these patients. In the recent PARADIGM HF that examined entresto in HF, 13% of patients had undiagnosed diabetes and 25% had undiagnosed pre-diabetes.

Screening for heart failure in patients with diabetes
Observational studies have suggested that up to 28% of diabetics have unrecognized HF. Few studies have examined HF screening strategies in patients with DM. One approach to screening for HF in DM that has been tested, is to screen only those with certain clinical characteristics that increase the risk of HF: older age, history of coronary artery disease, high body mass index, exercise-related shortness of breath, physical signs of enlarged heart (ie laterally displaced apex beat). Patients at risk can be screened with a simple transthoracic echocardiogram +/- assessment of serum NTproBNP. Such a strategy may be used to prevent complications and possibly improve outcomes by establishing appropriate treatment at an earlier stage in the disease.

Treatment of heart failure in patients with diabetes
All current heart failure therapies, including devices such as CRT and ICD exert the same beneficial effects in diabetics and non-diabetics. Theoretically, beta-blockers can alter the awareness of hypoglycemia by decreasing tremor and palpitations, however, the benefits of beta blockers in HF far outweigh the theoretical risks. Interestingly, in the PARADIGM Trial, use of Entresto was associated with a greater reduction in HBa1c and reduced rate of initiation of insulin or other diabetic drugs in comparison to enalapril.

Treatment of Type 2 diabetes in patients with HF
Glycemic targets should be individualized to reflect patient comorbidities, severity of HF and life expectancy. For example, there is little point in targeting an HbA1c of 6-7% if the patient has a limited life expectancy.

We are entering an exciting time in the treatment of diabetes, with new treatments that have been shown to improve cardiovascular outcomes. In HF it is important to select the treatments that offer the most benefit in this group and avoid those that have been shown to do harm. Although there have been no randomized trials of metformin in DM and HF, large observational studies have suggested beneficial effect of metformin on outcomes in patients with HF. Therefore, metformin should be first line treatment of DM in patients with HF and an egfr >30ml/min.

Second line drug treatment in patients with HF and DM should be SGLT2 inhibitors. This class of drug has been shown to reduce the risk of HF hospitalization in clinical trials, in addition to its benefits on mortality. In the EMPA –REG trial this effect was apparent in patients with and without heart failure at baseline.

Certain diabetic drugs have been shown to increase the risk of developing HF in patients with DM, and should therefore be avoided. Thiazolidenediones were shown to increase HF events in the RECORD and PROACTIVE studies, and the DPP4 inhibitor saxagliptin also increased the risk of HF hospitalization in the SAVOR-TIMI 53 trial. However, not all DPP4s increase HF risk alogliptin, sitagliptin and linagliptin do not appear to increase the risk of HF.

Conclusions
DM and HF both increase the risk of developing the other condition. When DM and HF are present together, patients feel worse and have poorer outcomes. Physicians should consider screening for both DM and HF in at risk patients. Careful consideration should be given to the choice of DM treatment used.

Diabetes and Heart Failure: Key Considerations

Heart Failure Patients
- Screen regularly for diabetes

Diabetes Patients
- Screen for heart failure with echocardiography/ NTproBNP if:
  - *older age, history of coronary artery disease, high body mass index, exercise-related shortness of breath, physical signs of enlarged heart.*

Treatment of Diabetes in Patients with Diabetes and Heart Failure
- **First Line** Metformin (egfr>30ml/min)
- **Second Line** SGLT2i

- **DRUGS TO AVOID** - Thiazolidinediones and Saxagliptin

By **Dr. Michael MacDonald**
Senior Consultant Cardiologist
The Harley Street Heart & Vascular Centre

HEART FAILURE

DIAGNOSIS AND ASSESSMENT OF HEART FAILURE IN PRIMARY CARE

In patients with non-acute onset, presenting with symptoms and signs of heart failure in the primary care setting, the probability of heart failure should be determined by the history and symptoms supported by findings on clinical examination. Blood tests, an ECG and a chest X-ray should be requested on all patients. During these steps, at least one element should be positive to consider the diagnosis and then plasma natriuretic peptides and an echocardiogram should be requested. The diagnostic assessment, in accordance with the ESC 2016 guidelines is summarized in the Table.

Brain Natriuretic Peptide (BNP)
Guidelines from the American College of Cardiology/American Heart Association (ACC/AHA) and European Society of Cardiology (ESC) recommend the use of natriuretic peptides for assessment of patients with symptoms of heart failure.

BNP and N-terminal pro-BNP (the cleaved inactive N-terminal fragment of the BNP precursor) levels can be used to evaluate patients with shortness of breath suspected of having heart failure. BNP is secreted by the atria and ventricles in response to stretching or increased wall tension. The hormone then causes fluid and sodium loss in the urine and mild vasodilation. BNP levels increase with age, is higher in women and patients of African origin, and can be elevated in patients with renal failure. BNP appears to have better reliability than N-terminal pro-BNP, especially in older populations.

Most dyspnoeic patients with HF have plasma BNP values > 400 pg/mL, while values <100 pg/mL have a very high negative predictive value for HF as a cause of dyspnea. In the range between 100-400 pg/mL, plasma BNP concentrations are not very sensitive or specific for detecting or excluding HF. As BNP levels increase, the specificity increases and thus the likelihood of a heart failure diagnosis. BNP levels also increase in level according to New York Heart Association classification. Elevations in plasma BNP can establish the presence of HF due to diastolic dysfunction with similar accuracy to systolic dysfunction. However, the values do not differentiate between systolic and diastolic dysfunction.

BNP levels are strong predictors of mortality at two to three months and cardiovascular events in acute heart failure, specifically when BNP level is > 200 pg/mL or N-terminal pro-BNP level is > 5,180 pg/mL. Limited evidence supports monitoring reduction of BNP levels in the acute and outpatient settings. A 30 to 50 percent reduction in BNP level at hospital discharge showed improved survival and reduced re-hospitalization rates. Optimizing management for outpatient targets of a BNP level < 100 pg/mL and an N-terminal pro-BNP level < 1,700 pg/mL showed improvement in decompensations, hospitalizations, and mortality events.

Echocardiography
Echocardiography should be performed in all patients with new onset HF and can provide important information about ventricular size and function. The sensitivity and specificity of two-dimensional echocardiography for the diagnosis of systolic dysfunction are as high as 80 and 100 percent, respectively. Valvular structure and function in valve disease can be characterized. A number of other important findings can be detected. Regional wall motion abnormalities are compatible with coronary artery disease. Pericardial thickening may be indicative of constrictive pericarditis. Infiltrative cardiomyopathies are associated with an abnormal myocardial texture. Left ventricular diastolic function can be assessed by estimation of the pulmonary capillary wedge pressure via the ratio of tissue Doppler of early mitral inflow velocity (E) to early diastolic velocity of the mitral annulus (e'). An E/e' ratio >15 suggests a Pulmonary capillary wedge pressure (PCWP) >15 mmHg when e' is the mean of medial and lateral mitral annulus early diastolic velocities. There are limitations to use of the E/e' ratio, which are beyond the scope of this article. Right atrial and pulmonary artery pressures, determined by the peak velocity of tricuspid regurgitation on Doppler echocardiography. These findings correlate with the pulmonary artery wedge pressure, regardless of the etiology of HF or severity of tricuspid regurgitation; they can be used to assess changes in left ventricular filling pressures resulting from therapy.

Treadmill Exercise Testing
Exercise testing should also be part of the initial evaluation of virtually all patients with HF. In addition to detection of ischemic heart disease, assessment of exercise capacity can be used for risk stratification and determining prognosis; serial measurements also can assess the efficacy of therapy and clinical stability of patients over time.

With severe HF, more formal cardiopulmonary exercise testing (CPET) and measurement of the maximal oxygen uptake (VO2max) provides an objective estimate of the functional severity of the myocardial dysfunction. VO2max is one of the best indices of prognosis in patients with symptomatic HF. However, peak VO2 and exercise capacity can be affected by factors other than cardiac status, including deconditioning, pulmonary disease, and anemia. One advantage of measuring VO2max directly is that cardiac and non-cardiac causes of impaired exercise can be distinguished by assessing the anaerobic threshold and related indices

Diagnostic assessment in patient with acute heart failure adapted from the ESC guidelines (2016)[1]

1. **Measurement of plasma natriuretic peptide level (BNP, NT-proBNP or MR-proANP)***
2.
a) 12-lead ECG
b) Chest X-ray†
c) Laboratory assessments: cardiac troponins, BUN (or urea), creatine, electrolytes (sodium, potassium), glucose, complete blood count, liver function tests and TSH
3. **Echocardiography**◇

* to help in the differentiation of acute heart failure form non-cardiac causes of acute dyspnea

† to assess signs of pulmonary congestion and detect other cardiac or non-cardiac diseases that may cause or continue to the patient's symptoms

◇Recommended immediately in haemodynamically unstable patients with acute heart failure and within 48 hours when cardiac structure and function are either not known or may have since previous studies

By **Dr. Rohit Khurana**
Senior Consultant Cardiologist
The Harley Street Heart & Vascular Centre

TO SQUEEZE OR NOT TO SQUEEZE?

LOWER LIMB COMPRESSION THERAPY IN HEART FAILURE

Congestive heart failure (CHF) is characterized by a shortness of breath, peripheral edema, palpitations and reduced cardiac output. Underlying primary diseases include ischemic heart disease, hypertension, valvular heart disease and post-inflammatory changes in the myocardium. Decompensated heart failure is classified under NYHA stages III and IV in which the symptoms mentioned above appear even at low levels of physical activity or when the body is at rest. Decompensated heart failure is considered a contraindication for compression therapy and lymphatic drainage.

The contraindication for using compression therapy with decompensated heart failure is based on the idea that blood volumes in the extremities shift towards the heart causing a volume overload in the pulmonary circulation and resulting in varying degrees of pulmonary edema. Scintigraphy and air-plethysmography reveal the displacement of regional blood volumes when medical compression stockings (MCS) are used. This is compensated for in patients with healthy hearts. However, intrinsic structural cardiac disease has been shown to change the behavior of myocardial regulatory processes. An increased volume in the right atrium produces a local rise in pressure and an increased expression of natriuretic peptides – a key biochemical marker for heart failure.

Compression therapy for the treatment of venous disease, lymphedema and limb swelling is usually prescribed by vascular specialists, phlebologists, dermatologists and family physicians, and less often by the cardiologists managing heart failure. It is important therefore to understand the correlation between compression and heart failure, and to establish the significance of the latter in patients considered for compression therapy of the limbs.

Deep vein thrombosis and CHF
It has been reported that the occurrence of DVT in the lower extremities is related to the severity of CHF (Howell MD et al. Congestive heart failure and outpatient risk of venous thromboembolism: a retrospective, case–control study. J Clin Epidemiol 2001;54:810–6). The risk of venous thromboembolism (VTE) increases as LVEF decreases. The odds ratio for VTE increases from 2.8 to 38.3 with a decrease in LVEF from 45 to 20%, and the risk of DVT and VTE increases according to NYHA functional class (Beemath A et al. Risk of venous thromboembolism in patients hospitalized with heart failure. Am J Cardiol 2006;98:793–5). Furthermore, another study demonstrated that abnormal platelet function (levels of soluble P-selectin and von Willebrand factor) as well as high plasma viscosity contributed to a hypercoagulable state in CHF patients with more severe NYHA class (Gibbs CR et al. Abnormalities of hemorheological, endothelial, and platelet function in-patients with chronic heart failure in sinus rhythm: effects of angiotensin-converting enzyme inhibitor and beta-blocker therapy. Circulation 2001;103:1746–51).

However, studies have also shown that the increase in right atrial volume is temporary and is not accompanied by clinically relevant hemodynamic changes, suggesting that medical compression stockings pose no threat at NYHA stages I and II. Invasive measurements of patients suffering from heart failure NYHA stages III and IV have also identified that hemodynamic changes caused by pneumatic compression are compensated for after a few minutes and usually only have minor clinical impact (Ringley CD et al. Evaluation of pulmonary arterial catheter parameters utilizing intermittent pneumatic compression boots in congestive heart failure. Am Surg 2002;68:286-90). Nevertheless, compression therapy on patients with decompensated heart failure should be strictly monitored due to its prognostic implications.

Pneumatic compression therapy and CHF
The arrival of intermittent pneumatic compression as a form of compression therapy has revolutionized management of DVT risk in the acute care hospital setting. These devices are part now part of standard care in the peri-operative and intensive care settings of a modern hospital as part of their VTE prophylaxis protocols. Studies have shown them to be safe and effective in patients with CHF with an increase in cardiac output and a decrease in systemic vascular resistance. (Amitai Bickel et al. Hemodynamic Effect and Safety of Intermittent Sequential Pneumatic Compression Leg Sleeves in Patients With Congestive Heart Failure Journal of Cardiac Failure, Volume 20, Issue 10, 2014, pp. 739-74). On the limb, these devices increase popliteal and soleal vein flow velocities, decrease venous stasis, reduce DVT and VTE episodes significantly and heal ulcers. However these have, until now, been available only within hospitals as the pneumatic pumps that are required are bulky and expensive and the sleeves that generate compression not really suitable for mobilizing patients.

Technology moves quickly and controlled pneumatic compression at home and with sleeves that allow the patient to wear shoes, walk about as well regulate the degree of compression themselves have now arrived. This allows for the beneficial hemodynamic effect of compression to the limb and heart to continue (reduced swelling, healing of ulcers, decrease DVT and VTE risk and increased cardiac output via enhanced pre-load) whilst ensuring that if decompensation occurs the patient or their carer can release the compression instantly or reduce the degree at home without the need for wound nurses and emergency physicians to be in attendance. It also removes the need for medical compression stockings often poorly tolerated by the patients, cumbersome as they are to put on and take off. A simple pressure gauge that allows pressure adjustment to either 20-30 mmHg (low level Class 1 compression stocking equivalent) to 30-40 mmHg (medium grade Class 2-3 compression stocking equivalent) and 40-50 mmHg ((high grade Class 4 compression stocking equivalent) is connected to the sleeve and pressure raised like in a traditional BP cuff (Fig 1.) Pressure can be raised slowly as tolerated by the patient and the sleeve can be deflated easily if there is persistent breathlessness or chest discomfort. No rushing ambulances are necessary.

The benefits of compression therapy for patients with venous insufficiency, ulcers and lymphedema are well known. However the presence of co-existing CHF in the patients has made the choice of compression therapy difficult. Home pneumatic compression therapy now allows treatment on both the limb and the heart to proceed without the need for hospital admissions. One no longer has to choose between a swollen limb and a congested heart.

Fig 1. The Aero-Wrap ambulatory pneumatic compression system
(now available at the Harley Street Heart and Vascular Centre)

By **Dr. Sriram Narayanan**
Senior Consultant Vascular Surgeon
The Harley Street Heart & Vascular Centre

HEART FAILURE

HEART FAILURE WITH PRESERVED EJECTION FRACTION: A GROWING EPIDEMIC

Heart failure (HF) with preserved ejection fraction (HFpEF), previously termed "diastolic HF", is a clinical syndrome in which patients have symptoms and signs of HF with relatively normal LV volumes and ejection fractions (LVEF ≥ 50%). Patients with HFpEF may present with dyspnoea, fluid retention, lethargy and dizziness, making it difficult to differentiate clinically from HF with reduced EF (HFrEF). Although LV function may be relatively preserved in HFpEF, the echocardiograms in these patients usually show some degree of diastolic dysfunction (eg, abnormal LV filling patterns and elevated filling pressures). In contrast, patients with HFrEF usually have increased LV volumes as well as reduced EF.

HFpEF is a growing public health problem which has only begun to receive more attention in recent years. HFpEF as has a prognosis similar to that of HFrEF and accounts for approximately half of all patients with HF. Unlike the multiple treatment options available for patients with HFrEF, the options available for patients with HFpEF are more limited. This may be due in part due to the wide range of factors that may contribute to the development of HFpEF (see figure) and a subsequent lack of understanding about the pathophysiology of the condition. However, it is important that clinicians are able to detect and manage patients with HFpEF to reduce morbidity and improve quality of life.

Treatment overview:
Clinical trials in HFpEF have produced neutral results to date and the mainstay of current treatment is to treat associated conditions (such as hypertension) and symptoms (such as fluid overload and breathlessness). Key objectives in treating patients with HFpEF are:

1) Controlling systolic and diastolic blood pressures according to clinical practice guidelines - target blood systolic and diastolic pressures should in general be below 130mmHg and 80mmHg, respectively

2) Relieve symptoms due to volume overload using diuretics - diuretics can provide symptomatic improvement but should be used with caution to avoid excessive preload reduction and symptomatic hypotension

3) Specific medications - Medications such as ARBs, ACE inhibitors, and beta blockers which are often used to treat patients with HFrEF lack proven efficacy to alter clinical outcomes in HFpEF. Other medications, such as nitrates, should be avoided in the treatment of HFpEF. There is some evidence that mineralocorticoid receptor antagonists (MRA), such as spironolactone and eplerenone, may have some benefit in patients with definite HFpEF (and increased plasma B-type natriuretic peptide levels). In the Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist (TOPCAT) trial [Pitt et al, NEJM 2014], 3445 patients with symptomatic HF and LVEF ≥45% were randomly assigned to receive either spironolactone or placebo. The investigators found no difference in composite primary outcome (death from cardiovascular causes, aborted cardiac arrest, or hospitalization for HF) between the two groups over a mean follow-up was 3.3 years. However, hospitalization for HF was less frequent in the spironolactone group (12.0 percent) compared with the placebo group (14.2 percent; HR 0.83, 95% CI 0.69-0.99). Total deaths and total hospitalizations were similar in the two groups. MRAs should be carefully administered and uptitrated and the patient's serum potassium and renal function closely monitored. To initiate MRA, the patient's serum potassium must be <5.0 mEq/L and eGFR should be ≥30 mL/min. For spironolactone, the initial dose is 12.5 mg once daily, which can be slowly increased to the maximum tolerated dose (aiming for 25 to 50mg), depending on the serum potassium and renal function. For eplerenone, the initial dose is 25 mg once daily, which is titrated as tolerated with monitoring in four weeks to 50 mg.

4) Manage co-morbidities - An important component of managing patients with HFpEF is to treat the contributing factors and comorbidities that are frequently present and significantly impact the clinical course. These include hypertension, lung disease, coronary artery disease, obesity, anemia, diabetes mellitus, kidney disease, and sleep disordered breathing. Patients with ischaemic heart disease in which the myocardial ischaemia may be contributing to their symptoms should be actively managed. Also, rhythm disorders such as atrial fibrillation, should be actively treated as these may worse the symptoms in patients with HFpEF. Ideally, sinus rhythm should be restored in patients with AF; if this is not possible, then good rate control is important. Sodium restriction and moderation of alcohol consumption should be recommended. Obese patients with HFpEF should be advised to lose weight. As compared with non-obese HFpEF patients, those with obesity tend to have more severe HF symptoms, more abnormal hemodynamics, and lower exercise capacity.

5) Exercise training - This is the only intervention shown to improve exercise capacity and quality of life in HFpEF patients. Several small randomized trials suggest that exercise training improves functional capacity and quality of life without any clear effects on cardiac function. Patients may benefit from a formal cardiac rehabilitation program if available.

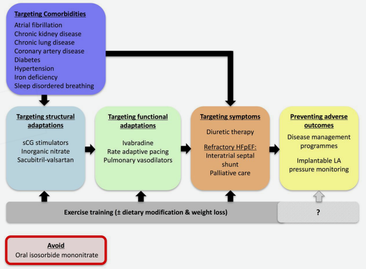

Figure- Heart failure with preserved ejection fraction- contributory factors

Conclusions:
The diagnosis and optimal treatment of patients with HFpEF can be challenging, in part due to the heterogenous nature of the condition and lack of treatments available with clear proven benefit. The risk factors for HFpEF include increasing age, obesity, hypertension, diabetes, chronic kidney disease and obstructive sleep apnoea. The diagnosis requires good clinical acumen combined with echocardiography and elevated plasma B-type natriuretic peptide levels. Management of HFpEF includes treating hypertension, fluid overload, considering starting a MRA (providing the serum potassium and renal function permit) and managing the co-morbidities and associated conditions. Prevention remains the best strategy and early recognition and diagnosis are also very important to tackle this growing epidemic.

By **Dr. Reginald Liew**

Senior Consultant Cardiologist
The Harley Street Heart & Vascular Centre

VASCULAR QUIZ

A 36 year old male presented with bilateral leg swelling and pigmentation with ulceration for 10 years (Fig 1 a.) He also had a large tortuous vein on the left side of his abdomen from the left groin towards the left axilla (Fig 1b.) He had had an episode of acute painful swelling of his left leg 10 years ago which had been treated by a procedure in the radiology department at that time. He has been on warfarin ever since.

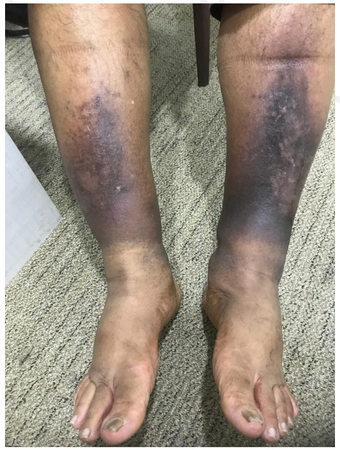

Fig 1a.

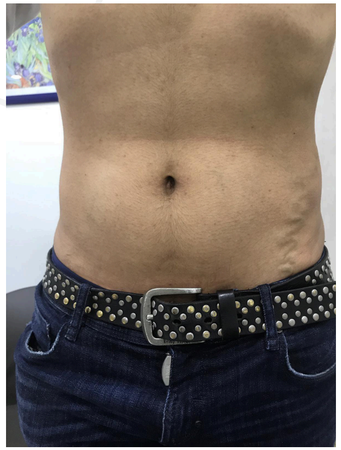

Fig 1b.

1. What is the diagnosis?
2. What may have been the procedure performed 10 years ago on him?
3. How can he be managed by his family physician?

Answer is available on our website:
http://www.harleystreet.sg/quiz-answers/medbulletin-sep-2019/

By **Dr. Sriram Narayanan**

Senior Consultant Vascular Surgeon
The Harley Street Heart & Vascular Centre

Mount Elizabeth Novena Specialist Centre
#05-30, 38 Irrawaddy Road
Singapore 329563

Gleneagles Hospital
#02-38/41 (Annex Block)
6A Napier Road
Singapore 258500

Mount Elizabeth Medical Centre
#11-07, 3 Mount Elizabeth
Singapore 228510

THE HARLEY STREET HEART & VASCULAR CENTRE

MEDBULLETIN SEPTEMBER2019

THE HARLEY STREET HEART & VASCULAR CENTRE

From left to right:
Dr. Sriram Narayanan, Dr. Reginald Liew
Dr. Michael MacDonald, Dr. Rohit Khurana

INTRODUCTION

Greetings from the Harley Street Heart and Vascular Centre! The aim of our second newsletter of 2019 is to provide the busy clinician with practical and succinct updates on key aspects of heart failure, a common but often under diagnosed condition, in which early diagnosis and treatment can have an important impact on patients' quality of life and prognosis. We would like to take this opportunity to welcome Dr. Michael MacDonald to our team. Dr. MacDonald's area of expertise is in heart failure and cardiac imaging, so the theme of the current newsletter is particularly relevant to his field. We are also pleased to announce the opening of our new heart and vascular centre at Mount Elizabeth Medical centre (#11-07) in September 2019, where we will be offering the same array of tests and expertise as we currently do in our other two centres at Gleneagles Hospital and Mount Elizabeth Novena Medical Centre.

In this edition, Dr. Khurana gives a summary of the common tests that can be ordered in primary which can help clinicians make an initial diagnosis of heart failure. These include NT-proBNP blood tests, echocardiography, exercise treadmill tests and cardiopulmonary exercise testing for a more detailed functional assessment of the breathless patient. Dr. MacDonald highlights the important link between heart failure and diabetes, both of which often occur in the same patient, and discusses the optimal treatment options. Dr. Liew has written a short article on the increasingly recognised syndrome of heart failure with preserved ejection fraction (HFpEF) and the measures that can be taken to reduce the impact of this condition, even though there are currently no specific medications available. Finally, Dr. Narayanan discusses the interesting but less commonly used method of lower limb compression therapy for heart failure patients, especially those with peripheral oedema and lower limb swelling.

As usual, we have included an interesting and challenging quiz at the end of the newsletter- the answer to the quiz will be posted on our website (www.harleystreet.sg) within a week of the newsletter being sent out.

We hope these articles stimulate and challenge your views on the latest ideas in the management of heart failure. Please feel free to contact us (at enquiries@harleystreet.sg) if you would like to provide any feedback or request a specific topic in future editions.

From The Harley Street Heart and Vascular Centre

http://www.harleystreet.sg/heart
Email **enquiries@harleystreet.sg**

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