GP Handbook

Cardiovascular Medicine

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ATRIAL FIBRILLATION

Atrial fibrillation (AF) is the most common sustained arrhythmia, and is associated with increased cardiovascular morbidity and mortality, and preventable stroke. The incidence and prevalence of AF rise with age, with a prevalence of 8% in people > 80. Atrial fibrillation can be due to valvular and non-valvular causes.

The $CHADS_2$ Score

For AF in valvular heart disease such as rheumatic mitral valve disease, anti-coagulation is indicated. For non-valvular AF, a commonly used clinical risk stratification system used is the CHADS2 Score:

	Condition	**Points**
C	Congestive heart failure	1
H	Hypertension	1
A	Age >75	1
D	Diabetes mellitus	1
S	History of stroke/TIA	2

According to the score, the following strategies are recommended:

$CHADS_2$ Score	**Risk**	**Anticoagulation**
0	Low	Aspirin
1	Moderate	Aspirin or Warfarin
>=2	Moderate or high	Warfarin

Dabigatran (Pradaxa) is a new oral direct thrombin inhibitor approved for anticoagulation in patients with AF. Studies have shown that a dose of 110mg BD was associated with rates of stroke and systemic embolism that were similar to those associated with warfarin, as well as lower rates of major haemorrhage. At a dose of 150mg BD, as compared with warfarin, it was associated with lower rates of stroke and systemic embolism but similar rates of major haemorrhage.

Rate or Rhythm Strategy?

Most patients with AF require rate control for symptomatic relief, and prevention of cardiomyopathy. Digoxin is no longer the drug of first choice in this regard. β-blockers are the most effective agents for monotherapy, followed by verapamil and diltiazem, as these drugs control both exertional and resting heart rate.

The decision to attempt cardioversion and maintain sinus rhythm, rather than just control heart rate, depends on the long-term frequency and hazards of AF, and risks of cardioversion and

antiarrhythmic therapy. Rate control is more suitable for older patients with asymptomatic persistent or permanent AF, whereas young patients with symptomatic paroxysmal AF may require rhythm control.

Sinus rhythm probably does confer a benefit, particularly for patients with heart failure. However, because drugs are relatively ineffective at long-term maintenance of sinus rhythm (60% in sinus rhythm at 1 year on amiodarone and 40% with sotalol), and have significant cardiac and extra-cardiac toxicities, including ventricular tachycardia and pulmonary fibrosis, this benefit is negated.

Cardioversion

Electrical cardioversion remains the mainstay for conversion of persistent AF to sinus rhythm, but there are significant issues of anticoagulation. Cardioversion can also be achieved with amiodarone and flecainide, but up to 2/3 of paroxysmal episodes revert spontaneously within 24 hours.

Catheter ablation

This procedure involves application of radiofrequency ablation to electrically isolate the pulmonary veins, with or without other lesions to attempt cure of AF. Success rates are variable, but approximate 75%, although this may require multiple procedures. If AF recurs, episodes may be less symptomatic or asymptomatic. Catheter ablation is associated with a 3–6% risk of major complications, including pulmonary vein stenosis, thromboembolism, and the rare (0.6%) but often fatal atrio-oesophageal fistula. It is safest and most successful in patients < 70 years old with paroxysmal AF for whom anti-arrhythmic therapy has been ineffective and who have a left atrial diameter < 5 cm and left ventricular ejection fraction > 40%.

CORONARY HEART DISEASE

Coronary heart disease (CAD), or ischemic heart disease (IHD) is a common disease (2.4-6.8% of population) and the second most common cause of death. The pathology is progressive atherosclerotic narrowing of coronary arteries and plaque rupture.

The common risk factors are:

- smoking
- lack of exercise
- hypertension
- hyperlipidemia
- diabetes mellitus
- central obesity (metabolic syndrome)

In high-risk patients, **chest pain means coronary heart disease** until proven otherwise. Other diagnoses not to be missed include:

- pulmonary embolism
- aortic aneurysm
- spontaneous pneumothorax
- malignancy of lung
- pneumonia / pleurisy / pericarditis

Homocysteine

Although there is evidence of linking homocysteine levels to cardiovascular risk, causal association is still not clear.

Exercise ECG

Treadmill stress test (exercise ECG) used to be the initial test of choice for patients with suspected ischaemic chest pain who have interpretable ECGs and can exercise. With its low sensitivity (68%) and specificity (77%), it is no longer a preferred choice of investigation for patients suspected of coronary heart disease.

Cardiac MRI (Functional)

MRI, once useful for evaluating coronary heart disease, has largely been replaced by cardiac CT.

Cardiac CT (Structural)

Advanced helical CT is now commonly used to allow identification of both calcified and non-calcified plaques within the coronary artery walls or to exclude significant stenosis due to atherosclerosis. With contrast-enhanced 3-dimensional CT scanning, early stages of coronary atherosclerosis can be seen before development of arterial narrowing. It allows early detection of potentially unstable "soft plaques" and non-calcified plaques in a reversible stage, making it possible to non-invasively monitor plaque regression with various therapies.

A normal cardiac CT scan excludes coronary artherosclerotic blockage for 5 years. It is useful as baseline study for symptomatic patients and those with significant coronary risks.

A calcium score, representing the degree of calcium deposition in atherosclerotic plaques, is routinely generated in a cardiac CT study. More coronary calcium indicates more atherosclerosis, and a higher chance of arterial narrowing and future cardiovascular events. However, the predictive value of coronary calcium scoring is not fully defined, and the score by itself is considered to be of limited value.

Cardiac CT poses no risk to the average patient if appropriate safety guidelines are followed. Because cardiac CT involves use of iodine-based contrast and x-ray, special precautions should be taken if the patients are allergic to iodine or shellfish or any medications, being treated with metformin, undergoing radiation therapy, over 60 years old or have a history of renal diseases.

Coronary angiography

Coronary angiography is performed by inserting a specialized, pre-shaped catheter into an artery (most commonly the radial artery nowadays), and with minimal manipulation, the catheter will then engage into the coronary ostia. Contrast agent is then injected to opacify the coronary system for visual evaluation.

Coronary angiography requires a high level of technical competence and specific equipments, which makes it relatively expensive. It is associated with mild patient discomfort during the procedure. The risk of serious complications is < 0.1%.

Intravascular ultrasound (IVUS)
IVUS is performed during cardiac catheterization using miniature ultrasound probes mounted on the tip of a specialized coronary catheter. The probe is placed beyond the target lesion site and the ultrasound catheter is then slowly pulled back during continuous imaging. This results in a series of images displaying cross-sectional views of the coronary artery as well as the lumen.

Cardiac Enzymes

Creatine kinase - MB fraction (CK-MB)
The MB fraction of creatine kinase is more specific for cardiac muscles. It rises in serum within 2-8 hours of onset of acute myocardial infarction. Serial measurements every 2 hours for a period of 12 hours in the acute stage will provide a pattern to determine whether the CK-MB is rising, indicative of myocardial injury. CK-MB is useful for diagnosing re-infarction as it begins to fall 1 day after the incident and dissipate in 3 days, so subsequent elevations may indicate another event.

Troponins
Troponin I and T are structural components of cardiac muscles, being released into the bloodstream with ischaemic myocardial trauma. They are highly specific for myocardial injury (more so than CK-MB) and help to exclude elevations of CK with skeletal muscle trauma. Troponins begin to increase following MI within 3-12 hours.

Troponin I remain elevated up to 9 days and troponin T up to 2 weeks. This makes troponins a superior marker for diagnosing myocardial infarction in the recent past - better than lactate dehydrogenase (LDH). Elevated Troponin T can occur in skeletal myopathies and renal failure.

Emergency Management

Angina pectoris
Angina can be relieved with *TNG tab 1 SL,* every 5 minutes. Patients should be warned about the common side effects of headache and flushing. Refer immediately to hospital if MI is suspected.

Emergency treatment of suspected MI that can be given in our clinic: TNG tab 1 SL stat, Cartia 100mg tab 3 PO stat, and morphine 15mg IM stat.

Thrombolytic, or fibrinolytic therapy with agents such as streptokinase or tissue plasminogen

activator (t-PA) can be used with the intention to lyse a recently formed thrombus, and re-establish blood flow. This helps to prevent and reduce myocardial injury, if given early in the course of the cardiac event. The 1st hour of onset of symptoms is the "Golden Hour", and no mortality benefit will be achieved by fibrinolytics in patients treated after 12 hours of onset of symptoms.

Acute myocardial infarction

Percutaneous Transluminal Coronary Angioplasty and Stenting (PTCA/S), or its modern name Percutaneous Coronary Intervention (PCI) is the preferred management for ST Elevating Myocardial Infarction (STEMI) and high risk Non-ST Elevating Myocardial Infarction/Unstable Angina (NSTEMI/UA) over medical therapy alone.

Fibrinolytics vs PCI

The morbidity and mortality data in recent 10 years favours primary PCI over fibrinolytics. PCI aims to open the occluded artery in a rapid, total and sustained way without the risk of causing excessive bleeding in the brain and other sites. The key point is whether the technical expertise and catheterization laboratory can be readily available. In HK, we have seen a decline in the use fibrinolytics in the private sector over the past 10 years.

Treatment for Chronic IHD

Medical therapy is the mainstay of treatment. PCI is reserved for high-risk patients. Life-style management including smoking cessation, diet modification, exercise prescription and psychosocial management can achieve more than 35% reduction in morbidity and mortality.

Antiplatelet agents

Platelets play a key role in coronary thrombosis after atherosclerotic plaque rupture. They are stimulated via different pathways including the cyclo-oxygenase pathway, the P2Y12 receptor and the thromoxane receptor. Drugs to inhibit each of these pathways have been developed. In patients with high coronary risks, with history of myocardial infarction or actually having a myocardial infarction, aspirin can reduce 22% of the composite end point of death, non-fatal recurrent infarction and nonfatal stroke.

The recommended dose of aspirin (soluble or enteric-coated) for Chinese is 75-150 mg qd. The main concern with aspirin is the risks of allergy (asthma) and gastrointestinal bleeding.

Clopidogrel (Plavix) is a the recommended first line oral antiplatelet agent:

- It is more effective than aspirin against IHD, stroke and peripheral vascular disease.
- Taken with aspirin, it improves the outcome of ST-elevation MI, non-ST-elevation MI and unstable angina.
- It has significantly less side effects, especially gastrointestinal symptoms and bleeding.

Clopidogrel resistance has been well documented and in part relates to the metabolic pathways required for the activation of clopidogrel which can result in a large spread of platelet inhibition within a population.

Some proton-pump inhibitors (PPI), eg omeprazole (Losec), Nexium (esomeprazole) have been shown to reduce the anti-platelet activity of clopidogrel by inhibiting CYP2C19, a hepatic cytochrome P450 enzyme. Using PPI with less interaction with CYP2C19, eg Pariet (rabeprazole), or administering PPI and clopidogrel at different times may alleviate this potential interaction.

Dabigatran (Pradaxa) is a new oral direct thrombin inhibitor. In patients with atrial fibrillation (AF), a dose of 110mg BD was associated with rates of stroke and systemic embolism that were similar to those associated with warfarin, as well as lower rates of major haemorrhage. Its role in treatment of CHD remains to be confirmed.

Beta-blockers
Beta-blockers were proved beneficial to IHD by numerous trials. The once-daily dosing of long-acting preparations eg Betaloc ZOK can greatly improve drug compliance.

Calcium channel blockers
Amlodipine (Norvasc) 5-10mg qd is slow-released with no significant reflex tachycardia. It is safe in congestive heart failure, and is the preferred calcium channel blocker for chronic IHD.

Nifedipine can be used concurrently with a beta-blocker to avoid reflex tachycardia. The long-acting preparation Adalat GITS 30mg/60mg is preferred because of its lower tendency towards sudden BP drop and reflex tachyarrhythmia)

Verapamil (Isoptin) and diltiazem (Herbesser) were once widely used to reduce angina and increase exercise tolerance. They are contraindicated in sick sinus syndrome, congestive heart failure, AV nodal disease. When used with beta-blockers, there are risks of worsening heart failure and bradyarrhythmias.

Anti-anginal medications
Nitroglycerin (nitrates) sublingual tablets, patches and sprays can be used before exertion to improve exercise tolerance; they are seldom used nowadays. Their side-effects are common: headache, flushing and hypotension. Absolute contraindication: sildenafil (Viagra) because of serious, prolonged, life-threatening hypotension.

Trimetazidine (Vastarel) inhibits long-chain 3-ketoacyl CoA thiolase activity (3-KAT), thus reducing fatty acid oxidation and stimulating glucose oxidation, resulting in less intracellular acidosis. Compared to nitrates, Vastarel MR is much more user friendy (no headache and dizziness, not affecting the BP, no interaction with Viagra).

Lipid lowering agents
Please refer to the chapter "Hyperlipidaemia".

Antioxidant Vitamin Supplement
At the moment, the scientific data do not justify supplementation of antioxidant vitamins (such as vitamin E, vitamin C, and ß-carotene) for prevention of coronary heart disease.

DYSPNOEA

Most common causes: anxiety, poor exercise tolerance.
Commonest organic aetiology: asthma and COAD.
Do not forget: heart failure.

Inhalational bronchodilators are superior to their oral counterparts, in terms of effectiveness and side-effect profiles.

COAD

Principles on management

- Stop smoking.
- Stay away from polluted air.
- Avoid contact with people who have colds or flu.
- Chest physiotherapy.
- Use antibiotics when sputum turns purulent to prevent further lung damage.

Heart Failure

Points to note

- Control of precipitating factors: arrhythmias, further MI, excessive salt intake, anaemia.
- Reduction in physical activity, salt restriction, water restriction to 1.5L/day.
- Caution in using NSAIDs as many drugs in this group cause fluid retention.
- ARB, with or without a diuretic (eg Diovan, Co-Diovan) are effective.
- Digoxin is useful in treating heart failure associated with AF.

ECONOMY CLASS SYNDROME

This is deep vein thrombosis (DVT) allegedly caused by the cramped leg room in airlines' economy class seating sections, although people who are seated for a long time, whether in a building or in any other form of transportation, may also develop this condition. Prolonged air travel poses an especially high risk, however, in part because movement is particularly restricted.

- A single relatively minor risk factor or a general tendency to develop leg swelling indicates only a very low risk. Elastic stockings and other compressive devices should be worn.

- Patients with a significant risk (eg post-thrombosis status, known malignancy, general or leg immobility or postoperative status) should be given low-molecular-weight heparin before boarding and again upon arrival.

- High risk patients (eg with multiple thrombosis or pulmonary embolism in their medical history) should be considered for full oral anticoagulation (pregnancy excepted). If this treatment is not possible, the patient should be advised to abandon the trip.

Low-Molecular-Weight Heparin (LMWH)
The dose of nadroparin (Fraxiparine), an LMWH, required for adequate prophylaxis without increased bleeding varies depending on risk factors. A typical regimen is 0.3ml SC qd (ie 2,850 anti-Xa IU). The first dose should be given 2-4 hours before boarding. Treatment should continue until the patient is actively ambulant or is no longer at risk of deep vein thrombosis, which usually means a few days after a long haul.

Warfarin
Taking a few warfarin tablets before a flight is a BAD idea. Warfarin must be accompanied by heparin in the first few days, or a hypercoagulable state occurs, resulting in an even higher risk for developing DVT.

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HYPERLIPIDAEMIA

Hypercholesterolaemia

- Hypercholesterolaemia is one of the most important risk factors for coronary heart disease. 10% increase in serum cholesterol is associated with a 20-30% increase in the risk for CHD, which killed > 5,300 Hong Kong people in 2003 (ie 14 deaths/day) and made itself the 2nd top killer in the locality.
- Lipid lowering drugs (LLD) reduce not only cholesterol level by 10% but also coronary death by 15%. Treatment for > 5 years yields a 25% reduction in coronary events.
- Serum total cholesterol reflects mainly endogenous cholesterol production, and fasting is not required for its determination. However, it may be influenced by acute illnesses (eg total cholesterol starts to fall 24 hours after myocardial infarction).
- Total cholesterol consists of sub-fractions of the protective HDL-cholesterol (HDL-C) and the atherogenic LDL-cholesterol (LDL-C).
- Direct LDL-C testing, although more expensive, is more accurate and preferred over the calculated LDL-C level.

Hypertriglyceridaemia

- Reflects exogenous fat consumption, hence fasting value is required
- Related to insulin resistance, DM, metabolic syndrome, hypothyroidism, renal diseases and increased alcohol intake
- Inversely proportional to HDL-cholesterol
- Increased risk of pancreatitis if >10 mmol/l
- Independent risk factor for CHD in the presence of a high LDL-C
- Rx: Lifestyle change -> +niacin -> +fibrates

LDL-C, the Lower the Better

The **Adult Treatment Panel (ATP) Guidelines** published by the National Cholesterol Educational Program (NCEP) in the US has been published 3 times over the past 20 years. While the key phrase of the original ATP III is "PRIMARY PREVENTION", that of the 2004 update is "LDL-C, THE LOWER THE BETTER". The update also emphasizes cholesterol lowering in those > 65 years. To summarize the guidelines:-

LDL-C is the lower the better in patients at high risk* for CHD, with a recommended treatment target of < 2.6 mmol/L. For the very high-risk patients, lowering LDL-C to < 1.8 mmol/L is now a therapeutic option.**

** CHD **or** CVD or DM or those with 2 or more CV risk factors*
*** CHD **and** risk factors, including DM, metabolic syndrome, or severe or poorly controlled risk factors*

Treatment

Although traditionally it is recommended that non-pharmacological means should first be used for 2-3 months before embarking on drug treatment, the practical situation in Hong Kong dictates that both measures should probably be employed right from the beginning.

Non-pharmacological treatment

- Weight reduction if overweight
- Regular exercise (2-3 times per week, 20-60 min each time using major muscle groups)

- Diet management (American Heart Association, ie AHA, recommends a dietary intake of < 300 mg of cholesterol per day):
    - Reduce dietary fat intake < 20% of total caloric intake (saturated fat < 10%)
    - Reduce intake of animal fats, fatty cuts of meat (veal, beef, chicken skin), dairy products (butter, cheese, ice cream), egg yolks, seafood (prawns, oysters, lobsters, crabs, squids), organ meats (brains, liver, heart), fast food (hamburgers, pizzas), coconut oil, egg noodles, baked food (cakes, cookies, biscuits)
    - Reduce frequency of eating out
    - Eliminate trans fat in the diet

Lipid lowering drugs (LLD)

There are at least 6 groups of anti-lipid drugs:

- Statins
- Fibrates
- Cholesterol absorption inhibitors
- Anion-exchange resins
- Nicotinic acids
- Fish oils.

If a patient has extremely high lipids, or if initial therapy with a single group is unsuccessful, drugs from more than one of the above groups may be combined. However, combining statins with either nicotinic acid or a fibrate increases the risk of myopathy, including myositis, rhabdomyolysis.

Hepatotoxicity is another complication that has raised concern and monitoring LFT on initiation of LLD and on increasing dosage is advised. LLD should be discontinued when liver enzymes increase >3 times the upper limit of normal. Since the effect is probably dose-related, the drug can be re-started at a lower dose after LFT becomes normal. LFT change usually occurs within the first 18 months and permanent impairment is rare after discontinuation of therapy.

Omega-3 fish oil preparations are able to reduce elevated triglycerides but may aggravate cholesterol levels. They are considered a relatively weak treatment and a large dosage would be needed to treat hypertriglyceridaemia.

All in all, LLD such as statins are safe and efficacious; the benefits far outweigh the risks of treatment. It is however a good practice to inform patients of possible risk of myopathy and hepatotoxicity on initiation of therapy and instruct patients to report events while on therapy.

Others

- Optimising glycaemic control
- Stopping beta-blockers and diuretics may improve hyperlipidaemia.
- Alcohol restriction especially in predominant hypertriglyceridaemia

Trans Fats

Trans fats (or trans fatty acids) are created in an industrial process that adds hydrogen to liquid vegetable oils to make them more solid. It is also known as "partially hydrogenated oils.

Companies like using trans fats in their foods because they are easy to use, inexpensive to produce and last a long time. Trans fats give foods a desirable taste and texture. Many restaurants and fast-food outlets use trans fats to deep-fry foods because oils with trans fats can be used many times in commercial fryers.

Trans fats raise LDL-C and lower HDL-C levels, thus increasing risk of cardiovascular events and stroke. It is also associated with a higher risk of developing type 2 DM.

Small amounts of trans fats occur naturally in some meat and dairy products, including beef, lamb and butter. It is not clear however if these naturally occurring trans fats have the same adverse effects on cholesterol levels as their industrially manufactured counterpart.

AHA recommends limiting the amount of dietary trans fats to < 1 % of our total daily calories. That means if one needs 2,000 calories a day, < 20 of those calories should come from trans fats. That is < 2 gms of trans fats a day. Given the amount of naturally occurring trans fats one probably eats every day, this leaves virtually no room at all for industrially manufactured trans fats.

Statins

Statin is the inhibitor of HMG-CoA reductase, the rate-limiting enzyme in liver cholesterol synthesis. It reduces LDL by 18-60%, triglycerides by 7-37%, and raises HDL by 5-15%. They are by far the most widely used LLD. The main drawbacks are:-

- Gastrointestinal discomfort, myalgia and raised liver enzymes are not uncommon in higher doses.
- Myopathy and rhabdomyolysis have been reported (a few cases for each statin over the world in the last 10 years). The risk of myopathy is increased with other drugs that share the cytochrome P450 system (eg niacin, fibrates, cyclosporine, imdazoles, macrolides, amiodarone, verapamil…etc).
- "Rule of Six" – each doubling of a statin dose results in only an additional 6% reduction in LDL. A potent statin in a lower dose is therefore preferred over a less potent one in higher dose.

Crestor

- The latest and most potent statin
- Usual dosage: 5-10 mg qd (a lot of patients can meet target with just 5mg daily)
- Lowering LDL by up to 63%
- High tolerability

Fibrates

As of October 2010, the Committee for Medicinal Products for Human Use (CHMP) of the European Medicines Agency (EMA) has concluded that the benefits of the 4 fibrates (bezafibrate, ciprofibrate, fenoflbrate and gemfibrozil) continue to outweigh their risks in the treatment of patients with blood lipid disorders. However, doctors should not prescribe them to newly-diagnosed patients with blood lipid disorders as first-line treatment, except for patients with severe hypertriglyceridaemia or patients who cannot take statins. For fenofibrate, the Committee noted additional new data and recommended that it can also be used together with a statin in some circumstances when a statin on its own cannot produce the desired results.

Ezetrol (Ezetimibe)

- First of a new class of lipid lowering agent – cholesterol absorption inhibitors (reduced delivery of intestinal cholesterol to the liver -> reduction of hepatic cholesterolstores -> increased clearance of blood cholesterol)
- As cholesterol in the body comes from 2 major sources – absorption from the intestine (approx 2/3 from biliary secretions and 1/3 from the diet) and synthesis in the liver and peripheral tissues, dual inhibition with ezetimibe and a statin should theoretically address both of these sources, and provide greater reductions in LDL-C.

- Vytorin (Zocor/Ezetrol) is such a medication, and available at different combinations of the two components. **However, results of the ENHANCE study comparing the effect of Vytorin to simvastatin alone showed little difference between the two medications when it came to plaque size in the arteries. The results suggest that Zocor, or its generic form may be as effective as the more expensive Vytorin.**

Niaspan (Nicotinic Acid)

- A once daily, sustained-release formulation of nicotinic acid, aka Niacin, an HDL-elevating agent
- Less hepatotoxicity and flushing compared to the regular-release nicotinic acid
- Reducing LDL-C by up to 20% in monotherapy, and up to 50% in combined Niaspan-statin therapy

Ω-3 Fatty Acids

Omega-3 polyunsaturated fatty acids are found in oil from certain types of fish*, vegetables, and other plant sources. These fatty acids are not made by the body and must be consumed in the diet. Clinical studies have determined that the main benefits of omega-3 fish oil are derived from DHA (DocosaHexaenoic Acid) and to a lesser extent EPA (EicosaPentaenoic Acid).

Fish or flax seed?
It is well known that the omega-3 in flax seed oil is ALA (Alpha Linolenic Acid), not the more active DHA and EPA. Although there is conversion of ALA to EPA and DHA in our body, the effectiveness is rather disappointing, especially in the elderly. Therefore, only fish oil provides DHA in a natural form that our body can quickly and easily utilize.

Supermarket-grade fish oil capsules:
The potential drawbacks of generic fish oil preparations include the following

- the ingredients are questionable
- the organic source might contain all kinds of contaminants in the form of PCB's (PolyChlorinated Biphenyls), and heavy metals including mercury.
- the extraction and processing uses heat and may reduce the levels of DHA.
- the fish oil usually does not come from the same country where it is processed; it is transported in tankers where the necessary additives and preservatives may damage the ingredients.

Recognizing these drawbacks, Solvay has produced Omacor, the only pharmaceutical grade omega-3 fish oil preparation that addressed these problems.

Dosage
For secondary prevention of cardiovascular disease, 1 gm per day of fish oil has shown to reduce overall and cardiovascular mortality, myocardial infarction, and sudden cardiac death. Higher doses (2-4 gm per day) may be used for its triglyceride-lowering effects and for patients with rheumatoid arthritis to reduce NSAID use. Ω-3 fatty acid supplementation of infant formula has shown benefit in infant neural growth and development. With the potential health benefits of fish, women of childbearing age should be encouraged to eat 1 to 2 low-mercury fish meals per week.

**The most common sources are mackerel (Atlantic), salmon (Atlantic) trout and Arctic char.*

HYPERTENSION

Hypertension (HT) is defined as BP > 140/90 measured on at least **2** different occasions at least 1 week apart. In HK the prevalence of HT was about 10% in a community hospital base study. In 1,513 Chinese employees of a local public utility company, the prevalence of HT, defined as systolic blood pressure > 140 or diastolic pressure > 90, was 17% in men and 5% in women.

Aim of Treatment

The minimal treatment goal set by WHO-ISH* 1999 and JNC VII** is both at < 140/90 mmHg. The levels are stricter for patients with DM or chronic renal disease (WHO-ISH* 1999: < 130/85 mmHg; JNC VII: < 130/80 mmHg). The optimal BP set by WHO-ISH 1999 and JNC VII is the same at 120/80 mmHg.

Benefits of treatment

A reduction of systolic pressure of 10 mmHg and diastolic pressure of 6 mmHg reduces the risk of stroke by 1/3 and the risk of a major coronary event by 1/6. Contrary to the old belief, systolic BP is a better predictor for cardiovascular disease, cerebrovascular disease, end-stage renal disease than diastolic BP.

'White coat' hypertension

Many patients are fearful of doctors and develop an alarm reaction. In these patients ambulatory 24-hour blood pressure recording may show a marked reduction in BP while the patient is at home.

** World Health Organization – International Society of Hypertension*
*** The Seventh Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure*

Diagnosis

History, physical examination and laboratory investigations aim at diagnosing:

(1) **secondary causes (in 'young' hypertensive; do not forget sleep apnoea)**
(2) **associated medical conditions:** DM, hyperlipidaemia, gout…
(3) **target organ damage:** do not forget the eyes, the teeth and the feet

Routine tests

- Blood for CBP, ESR, LFT, RFT, FBS, lipids, uric acid
- Urinalysis
- CXR
- Resting ECG

Tests looking for secondary causes

- Blood for TSH
- 24-hour urine for creatinine clearance
- 24-hour urine for VMA
- Renal MRA

Tests looking for target organ damage

- 24-hour urine for creatinine clearance
- Cardiac CT

Non-Pharmacological Treatment

- Stop smoking
- Reduce weight
- Lower alcohol consumption
- Decrease salt intake
- Adopt a less stressful lifestyle

Pharmacological Treatment

Offer patients with isolated systolic hypertension (SBP > 160 mmHg) the same treatment as those with both raised systolic and diastolic BP.

- STEP 1 : Start with an ARB. If BP control is not satisfactory ->
- STEP 2 : Add a CCB. If CCB is not suitable, eg because of oedema or intolerance, or in case of heart failure, offer a thiazide diuretic. If BP control is not satisfactory ->
- STEP 3 : ARB+CCB+Thiazide. If BP control is not satisfactory -> Refer.

Angiotensin receptor blockers (ARB)
These have become widely used as the first line treatment because of their multiple end organ benefits, eg increasing insulin sensitivity, improving renal function, enhancing cardiac performance, reducing risks of cardiovascular and cerebrovascular events and stroke. They are almost as well tolerated as placebo (no coughing). Contraindications are the same as for ACEI. Do not combine an ARB with an ACEI.

Calcium channel blockers (CCB)
CCBs decrease myocardial contractility and cause vasodilatation, and are used for treating HT, angina, and cardiac arrhythmias. Amlodipine (Norvasc) have very little effect on heart rate and contraction and so is safer to use in individuals who have heart failure or bradycardia.

Thiazide diuretics
Thiazide diuretics is now used mainly as an adjunctive medication when a combination of ARB and CCB cannot produce the desired anti-hypertensive result. Fixed-dose combinations of ARB, CCB and thiazide diuretic (eg Co-Diovan HCT) have gained increasing popularity. Its major drawbacks are its propensity for hyperglycaemia, hyperlipidaemia, hyperuricaemia, hypokalaemia and hyponatraemia.

Thiazides are sulphonamides and should not be given to patients allergic to sulphur drugs.

Beta-blockers
Use a once daily selective agent, eg atenolol 50mg or Betaloc ZOK 100mg in a relatively low dose.

Avoid beta-blockers in the elderly, who are prone to more frequent side-effects, especially tiredness, and in whom they seem less effective. Do not use beta-blockers in smokers and avoid using them in hyperlipidaemia. Observe the usual contraindications, especially a history of airways obstruction.

Antihypertensives & Pregnancy

- As BP falls in early pregnancy, decreasing or even discontinuing anti-hypertensive medications is often possible in women with mild hypertension.
- Acceptable anti-hypertensives: **methyldopa**, **labetalol**, and **nifedipine**.
- ACEI and ARB are contraindicated in all trimesters.
- Thiazide diuretics can decrease placental perfusion and adversely affect the fetus.

- Atenolol use should be avoided as it has been associated with lower birth weights.

Antihypertensives & Breast Feeding

- **Methyldopa** is generally considered safe.
- Atenolol and metoprolol are concentrated in breast milk, possibly to levels that could affect the infant; by contrast, exposure to labetalol and propranolol seems low.
- Although milk concentrations of diuretics are low and considered safe, these agents can decrease milk production significantly.
- There are reports of CCB going into breast milk, apparently without adverse effects.
- Sufficient data exist for the safety of two ACEI, captopril and enalapril; the American Academy of Pediatrics deems these drugs compatible with breast feeding.
- ARB: varied animal data show detectable milk levels, and recommendation regarding their safety cannot be given.

PALPITATIONS

Palpitations may be described as increased awareness of the normal heartbeat, or the sensation of rapid, slow, irregular or forceful heart rhythm.

Cardiac Causes

- Ventricular tachycardia
- Supraventricular tachycardia
- AF
- Extrasystoles

Non-Cardiac Causes

- Anxiety (commonest)
- Anaemia
- Thyrotoxicosis
- Fever

History

It is important to *clarify what exactly palpitations means to the patient*. A useful way to assess the rate and regularity is to ask the patient to mimic their rhythm by finger tapping. Careful analysis of the characteristics of palpitations helps to narrow the differential diagnoses. Associated symptoms (eg syncope, chest pain) are important. The drug history and past health should be assessed.

ECG

To look for - short PR intervals with delta waves (WPW syndrome), pathological Q waves (old infarct), and long QT intervals (Long QT syndrome)…etc.

Further investigations

Holter monitoring is one of the most important tools for diagnosis of palpitations. Invasive electrophysiology study is reserved for high risk patients (eg palpitations preceding a syncope, sustained and poorly tolerated palpitations with heart disease) and patients undergoing radiofrequency ablation. Echocardiogram, exercise-ECG, and cardiac MRI may be indicated.

Management

General advice

- Teach the patient how to take the pulse during palpitations
- Visit a doctor for ECG documentation during palpitations
- Avoid precipitating factors eg alcohol, drugs…etc

Specific treatment

- Ventricular tachycardia - REFER
- Supraventricular tachycardia (SVT) - vagotonic maneuvers, drugs, radiofrequency ablation
- AF - anticoagulation is most important in preventing stroke. Cardioversion may be used in newly onset AF. Amiodarone is best for maintaining sinus rhythm. Digoxin is effective in controlling the ventricular response at rest. Beta-blockers are used to control ventricular response during exercise.
- Symptomatic extrasystoles in normal heart - reassurance; may consider beta-blockers