Ischemic Heart Diseases

Epidemiology, Risk Factors, prevention

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2021 ESC Guidelines on cardiovascular disease prevention in clinical practice

Developed by the Task Force for cardiovascular disease prevention in clinical practice with representatives of the European Society of Cardiology and 12 medical societies

With the special contribution of the European Association of Preventive Cardiology (EAPC)

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Atherosclerotic cardiovascular disease (ASCVD) incidence and mortality rates are declining in many countries in Europe, but it is still a major cause of morbidity and mortality.

Over the past few decades,major ASCVD risk factors have been **identified**.

the prevalence of unhealthy lifestyle is still high, and ASCVD risk factors are often poorly treated,even in patients considered to be at high (residual) CVD risk

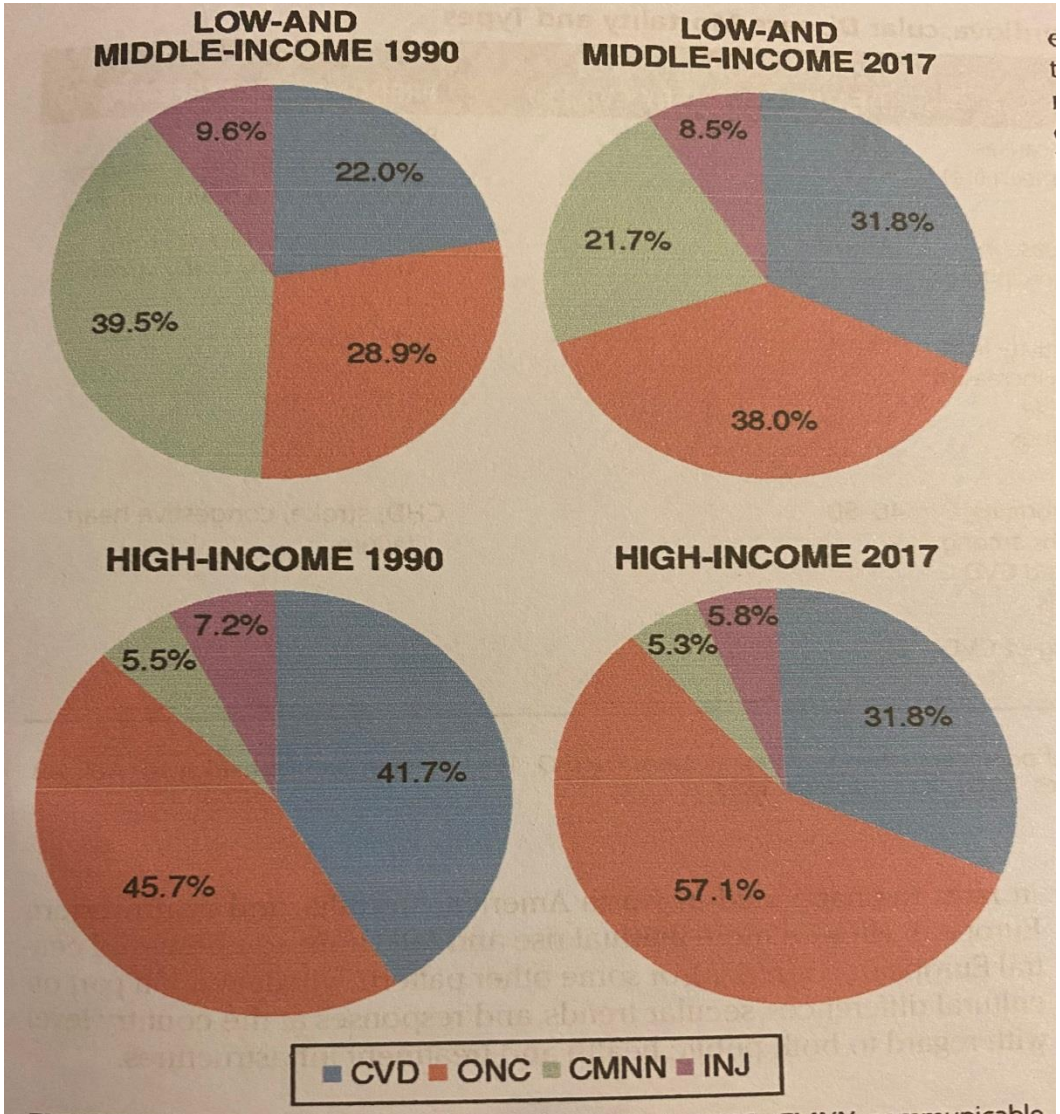


FIGURE 2.1 Changing pattern of mortality, 1990 to 2017. *CMNN*, communicable, maternal, neonatal, and nutritional diseases; *CVD*, cardiovascular disease; *INJ*, injury; *ONC*, other noncommunicable diseases. (From Global Burden of Disease Study 2017. *Age-sex Specific All-cause and Cause-specific Mortality, 1990–2017*. Seattle: Institute for Health Metrics and Evaluation; 2017.)

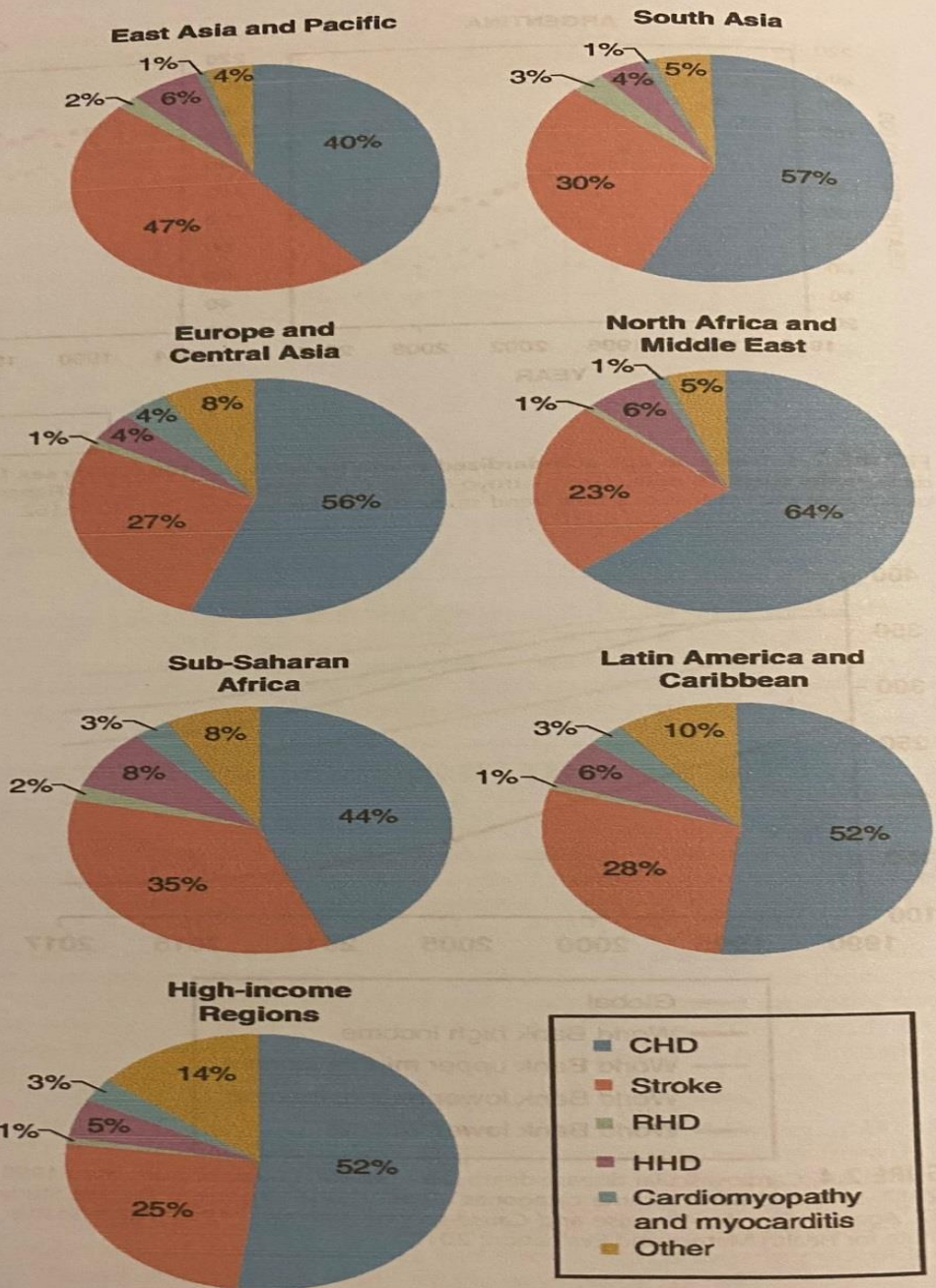


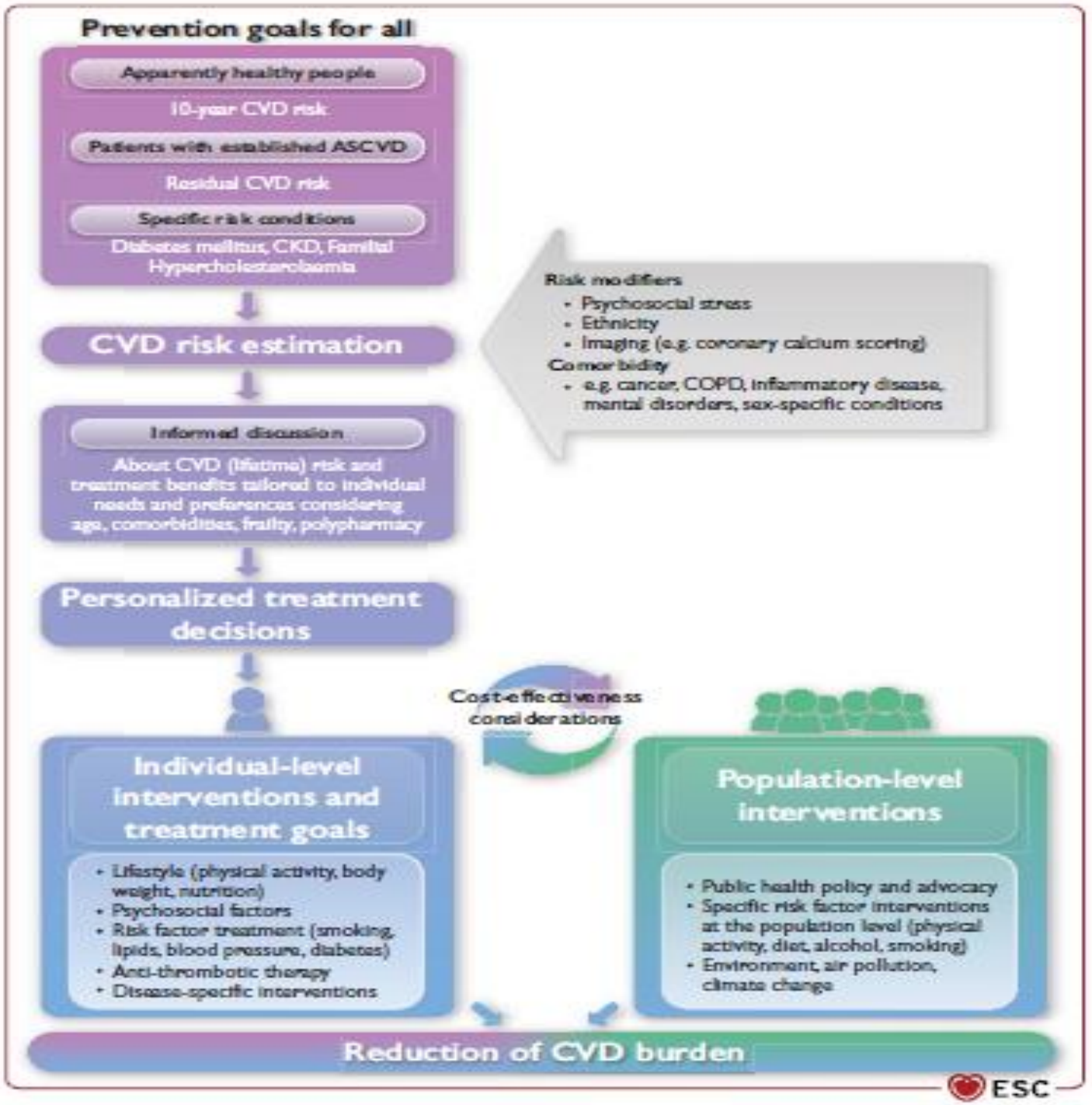
FIGURE 2.7 Cardiovascular disease death by specific cause and region. *CHD,* Coronary heart disease; *HHD,* hypertensive heart disease; *RHD,* rheumatic heart disease. (From Global Burden of Disease Study 2017. *Age-sex Specific All-cause and Cause-specific Mortality, 1990–2017.* Seattle: Institute for Health Metrics and Evaluation; 2017.)

Target population for assessing cardiovascular disease risk

CVD risk assessment or screening can be done opportunistically or systematically.

Systematic CVD risk assessment assessment in the general population(adult men >40 and women >50 years of age) with no known CV risk factors **appears not cost-effective** in reducing subsequent vascular events and premature death, at least in short-term follow-up, but does increase detection of CV risk factors.

Risk assessment is not a **one-time** event; it should be repeated, for example, every 5 years, although there are no empirical data to guide intervals.


Prevention goals for all
Apparently healthy people
10-year CVD risk
Patients with established ASCVD
Residual CVD risk
Specific risk conditions
Diabetes mellitus, CKD, Familial Hypercholesterolaemia
CVD risk estimation
Risk modifiers
• Psychosocial stress
• Ethnicity
• Imaging (e.g. coronary calcium scoring)
Comorbidity
• e.g. cancer, COPD, inflammatory disease, mental disorders, sex-specific conditions
Informed discussion
About CVD (lifetime) risk and treatment benefits tailored to individual needs and preferences considering age, comorbidities, frailty, polypharmacy
Personalized treatment decisions
Cost-effectiveness considerations
Individual-level interventions and treatment goals
• Lifestyle (physical activity, body weight, nutrition)
• Psychosocial factors
• Risk factor treatment (smoking, lipids, blood pressure, diabetes)
• Anti-thrombotic therapy
• Disease-specific interventions
Population-level interventions
• Public health policy and advocacy
• Specific risk factor interventions at the population level (physical activity, diet, alcohol, smoking)
• Environment, air pollution, climate change
Reduction of CVD burden
ESC

Recommendations for CVD risk assessment

Recommendations	Class[a]	Level[b]
Systematic global CVD risk assessment is recommended in individuals with any major vascular risk factor (i.e. family history of premature CVD, FH, CVD risk factors such as smoking, arterial hypertension, DM, raised lipid level, obesity, or comorbidities increasing CVD risk).	I	C
Systematic or opportunistic CV risk assessment in the general population in men >40 years of age and in women >50 years of age or postmenopausal with no known ASCVD risk factors may be considered.[9]	IIb	C
In those individuals who have undergone CVD risk assessment in the context of opportunistic screening, a repetition of screening after 5 years (or sooner if risk was close to treatment thresholds) may be considered.	IIb	C
Opportunistic screening of BP in adults at risk for the development of hypertension, such as those who are overweight or with a known family history of hypertension, should be considered.[19]	IIa	B
Systematic CVD risk assessment in men <40 years of age and women <50 years of age with no known CV risk factors is not recommended.[9]	III	C

ASCVD = atherosclerotic cardiovascular disease; BP = blood pressure; CV = cardiovascular; CVD = cardiovascular disease; DM = diabetes mellitus; FH = familial hypercholesterolaemia.

[a]Class of recommendation.

[b]Level of evidence.

Risk factors

The main causal and modifiable ASCVD risk factors are blood apolipoprotein-B-containing lipoproteins [of which low-density lipoprotein (LDL) is most abundant], high BP, cigarette smoking, and DM.

Another important risk factor is adiposity, which increases CVD risk via both major conventional risk factors and other mechanisms.

In addition to these, there are many other relevant risk factors, modifiers, and clinical conditions, which are addressed under riskmodifiers and clinical conditions (comorbids).

cholesterol

-The causal role of LDL-C, and other apo-B-containing lipoproteins, in the development of ASCVD is demonstrated beyond any doubt by genetic, observational, and interventional studies.

-Non-high-density lipoprotein cholesterol (HDL-C) encompasses all atherogenic (apo-B-containing) lipoproteins, and is calculated as: total cholesterol HDL-C = non-HDL-C.

The relationship between **non-HDL-C** and CV risk is at least *as strong as* the relationship with **LDL-C**. Non-HDL-C levels contain, in essence, the same information as a measurement of apo-B plasma concentration. Non-HDL-C is used as an input in the Systemic Coronary Risk Estimation 2 (SCORE2) and SCORE2-Older Persons (SCORE2-OP) risk algorithms.

-HDL-C is inversely associated with CVD risk. Very high HDL-C levels may signal an increased CVD risk. There is, however, no evidence from Mendelian randomization studies, or randomized trials of cholesteryl ester transfer protein inhibitors, that raising plasma HDL-C reduces CVD risk.

Blood pressure

Longitudinal studies, genetic epidemiological studies, and RCTs have shown that raised BP is a major cause of both ASCVD and nonatherosclerotic CVD [particularly heart failure (HF)], accounting for **9.4** million deaths and **7%** of global disability adjusted life-years.

Elevated BP is a risk factor for the development of coronary artery disease (CAD), HF, cerebrovascular disease, lower extremity arterial disease (LEAD), chronic kidney disease (CKD), and atrial fibrillation (AF).

The risk of death from either CAD or stroke increases linearly from BP levels as low as **90** mmHg systolic and **75** mmHg diastolic upwards.The absolute benefit of reducing systolic BP (SBP) depends on absolute risk and the absolute reduction in SBP, except that lower limits of SBP are imposed by tolerability and safety considerations.

Management is determined by the category of hypertension (optimal, normal, high-normal, stages 1 to 3, and isolated systolic hypertension), defined according to seated office BP, ambulatory BP monitoring (ABPM), or home BP average values.

Evidence suggests that lifetime BP **evolution differs** in women compared to men, potentially resulting in an increased CVD risk at lower BP thresholds.

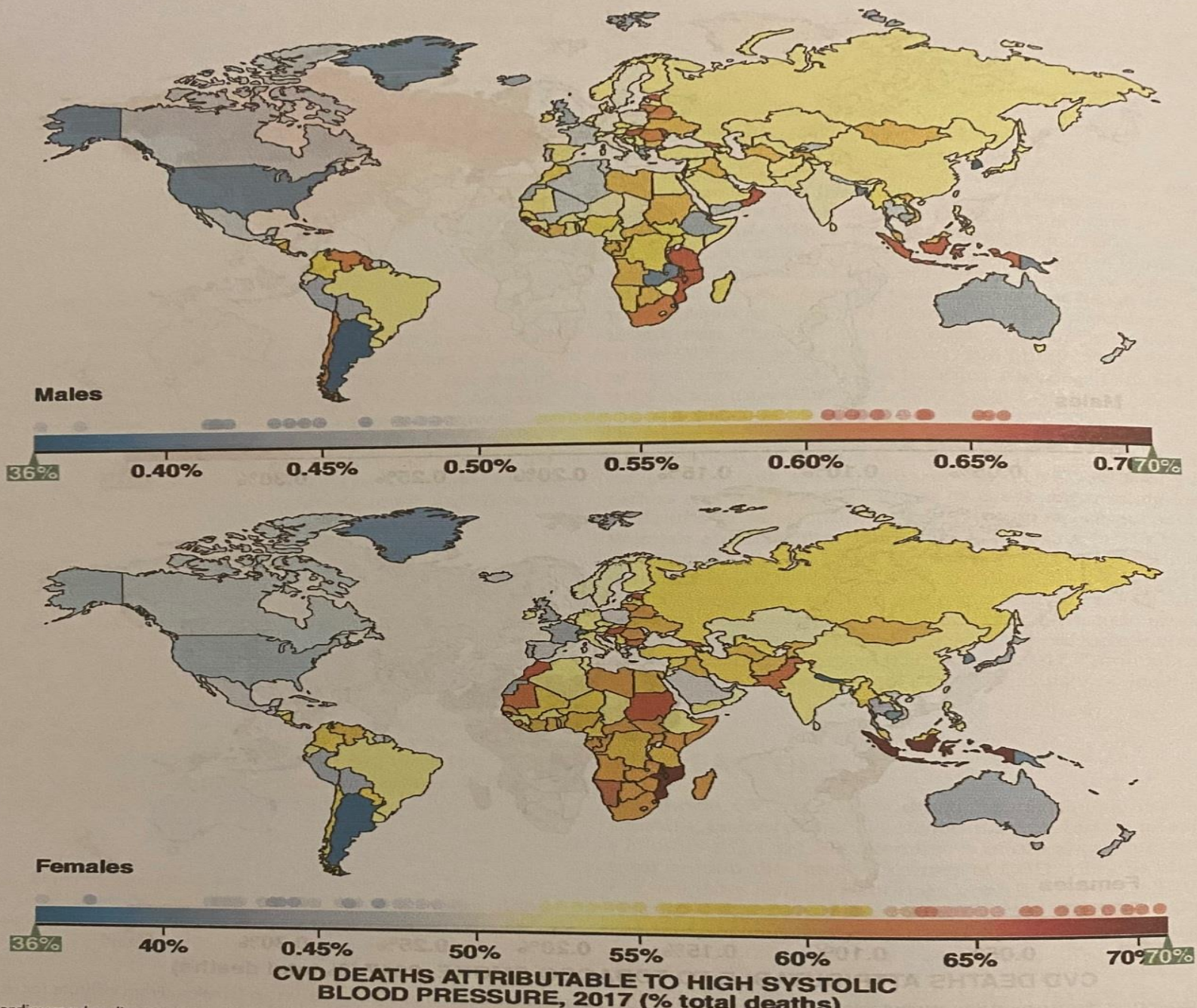


FIGURE 2.9 Cardiovascular disease mortality attributable to high systolic blood pressure in 2017, percentage of total deaths, males versus females. (From Institute for Health etrics and Evaluation (IHME). *GBD Compare*. Seattle: IHME, University of Washington; 2017. http://vizhub.healthdata.org/gbd-compare.)

Cigarette smoking

Cigarette smoking is responsible for **50%** of all avoidable deaths in smokers, with **half** of these due to ASCVD.

A lifetime smoker has a **50%** probability of dying due to smoking, and on average will lose **10** years of life.

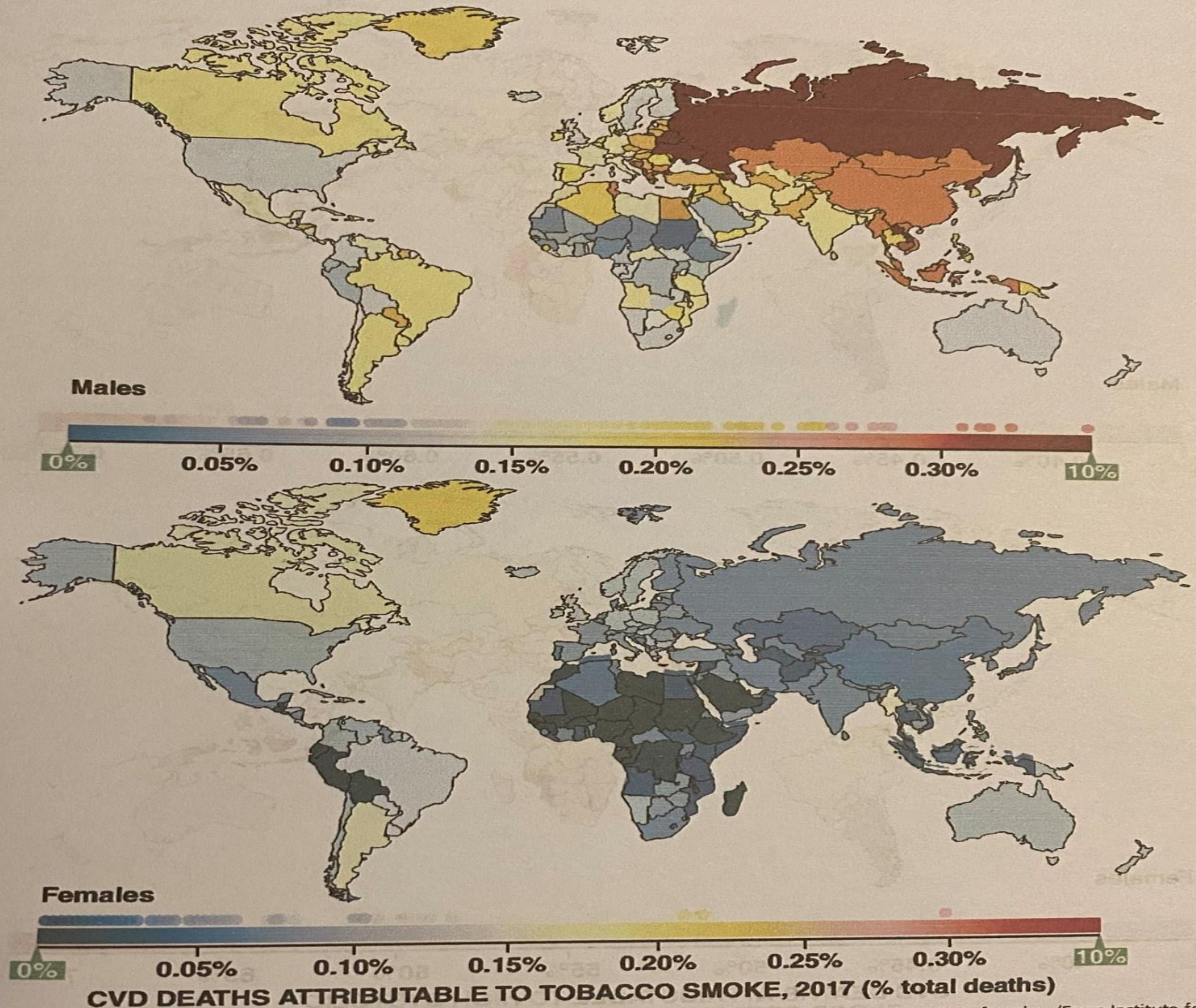
The CVD risk in smokers <50 years of age is **five-fold** higher than in non-smokers.

Prolonged smoking is more hazardous for women than for men.

Worldwide, after high SBP, smoking is the leading risk factor for disability adjusted life-years.

Second-hand smoke is associated with an increase in CVD risk.

Some smokeless tobacco is also associated with increased risk of CVD



GURE 2.8 Cardiovascular disease mortality attributable to tobacco smoke in 2017, percentage of total deaths, males versus females. (From Institute for Health M aluation (IHME). *GBD Compare*. Seattle: IHME, University of Washington; 2017. http://vizhub.healthdata.org/gbd-compare.)

Diabetes mellitus

Type 1 DM, **type 2 DM**, and **prediabetes** are independent risk factors for ASCVD, increasing risk of ASCVD by about two-fold, depending on the population and therapeutic control.

Women with type 2 DM appear to have a particularly higher risk for **stroke**.

Patients with type **2 DM** are likely to have **multiple ASCVD risk factors**(including dyslipidaemia and hypertension), each of which mediates an increase in risk of both ASCVD and non-ASCVD

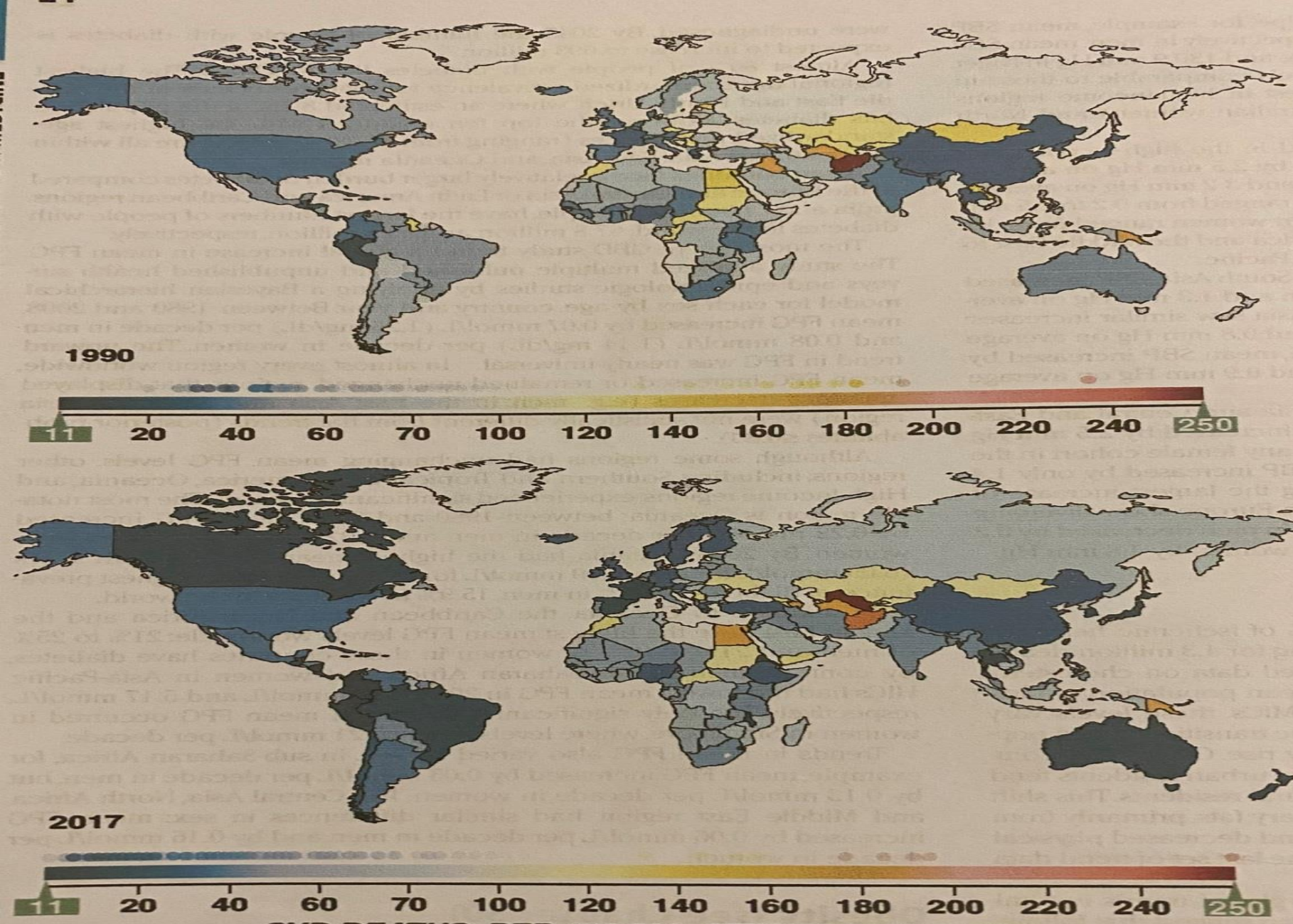


FIGURE 2.10 Cardiovascular disease mortality attributable to high fasting plasma glucose, deaths per 100,000, 1990 versus 2017. (From Institute for Health Metrics and Evaluation (IHME). *GBD Compare*. Seattle: IHME, University of Washington; 2017. http://vizhub.healthdata.org/gbd-compare.)

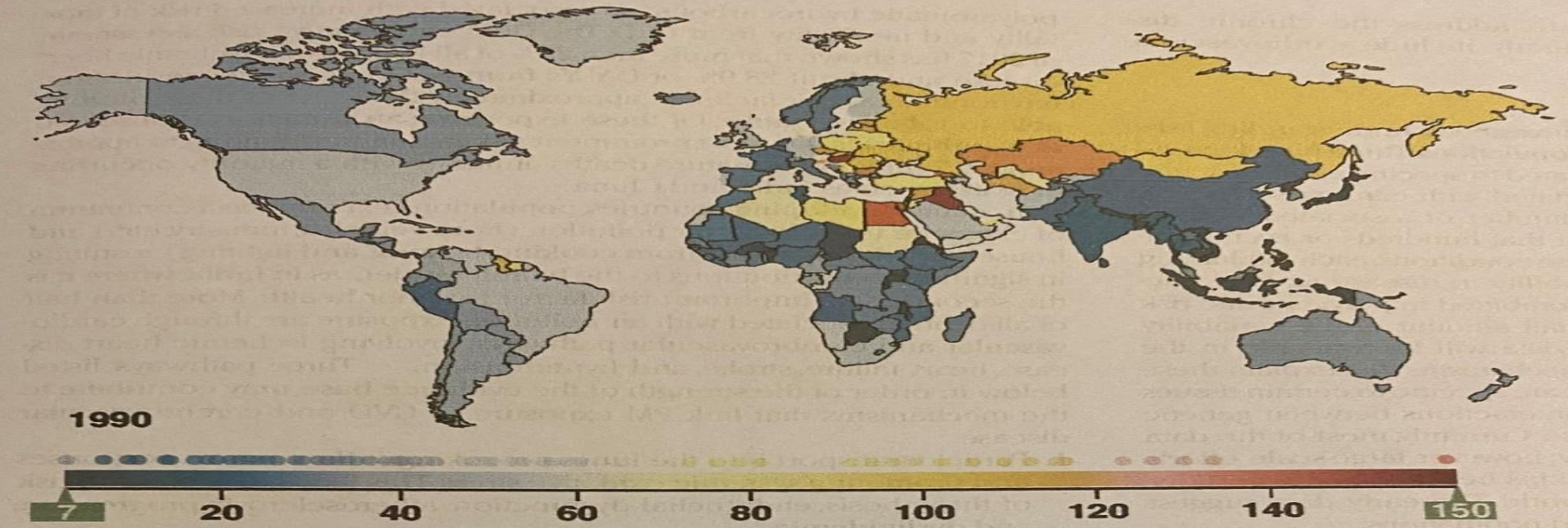
Adiposity

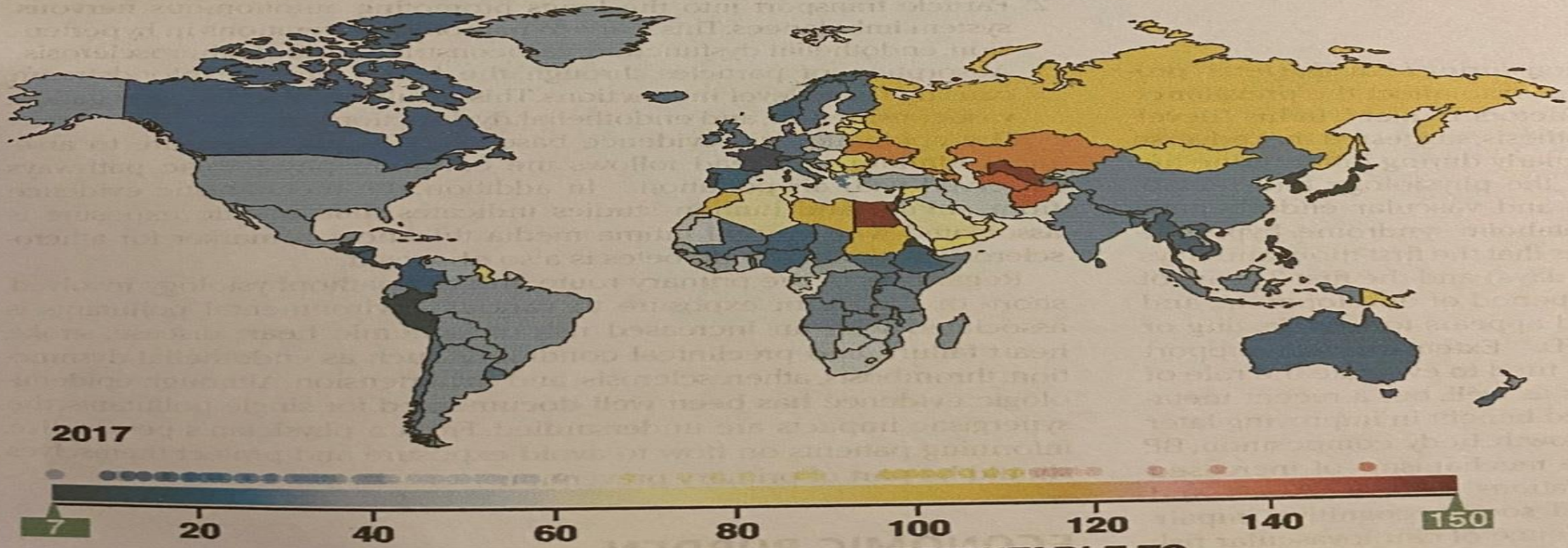
Mendelian randomization analyses suggest a linear relation between BMI and mortality in non-smokers and a J-shaped relation in ever-smokers.

All-cause mortality is lowest at a BMI of **20-25** kg/m_2 in apparently healthy people, with a J-shaped or U-shaped relation.

In HF patients, there is evidence for an **obesity paradox**, with lower mortality risk in patients with higher BMI.

A meta-analysis concluded that **both BMI and waist circumference** are similarly, strongly, and continuously associated with **ASCVD and type 2 DM**





CVD DEATHS, PER 100,000, ATTRIBUTABLE TO HIGH BODY MASS INDEX, 1990 vs. 2017

FIGURE 2.11 Cardiovascular disease mortality attributable to high body-mass index, deaths per 100,000, 1990 versus 2017. (From Institute for Health Metrics and Evaluation (IHME). *GBD Compare*. Seattle: IHME, University of Washington; 2017. http://vizhub.healthdata.org/gbd-compare.)

Sex and gender and their impact on health

Where evidence exists on the risk modifying effect of sex or where **sex-specific clinical conditions** and clinical management strategies exist, this has been included in these guidelines.

The influence of gender on an **individual's experience** and **access to healthcare** is paramount.

Epigenetic effects of social constructs appear to condition the **translation of biological sex into disease pathophysiology.**

Furthermore, social constructs can also be determinants of health access, healthcare utilization, disease perception, decision-making, and perhaps therapeutic response, including in the field of CVD **and** ASCVD prevention.

Examples of specific topics regarding physiological, pathological, and clinical differences related to sex and gender that have been studied include left ventricular (LV) ejection fraction (LVEF), adverse drug reactions, trends in ASCVD risk factors and awareness, sex disparitiesin the management of and outcomes after acute coronary syndromes (ACS).

Furthermore, CVD health after **menopause** transition, **pregnancy disorders,** and **gynaecologic condition**s have recently been reviewed

Cardiovascular disease risk classification

Age is the major driver of CVD risk. Women below 50 years and men below 40 years of age are almost invariably at **low 10-year** CVD risk, but may have unfavourable modifiable risk factors that sharply increase their longer-term CVD risk.

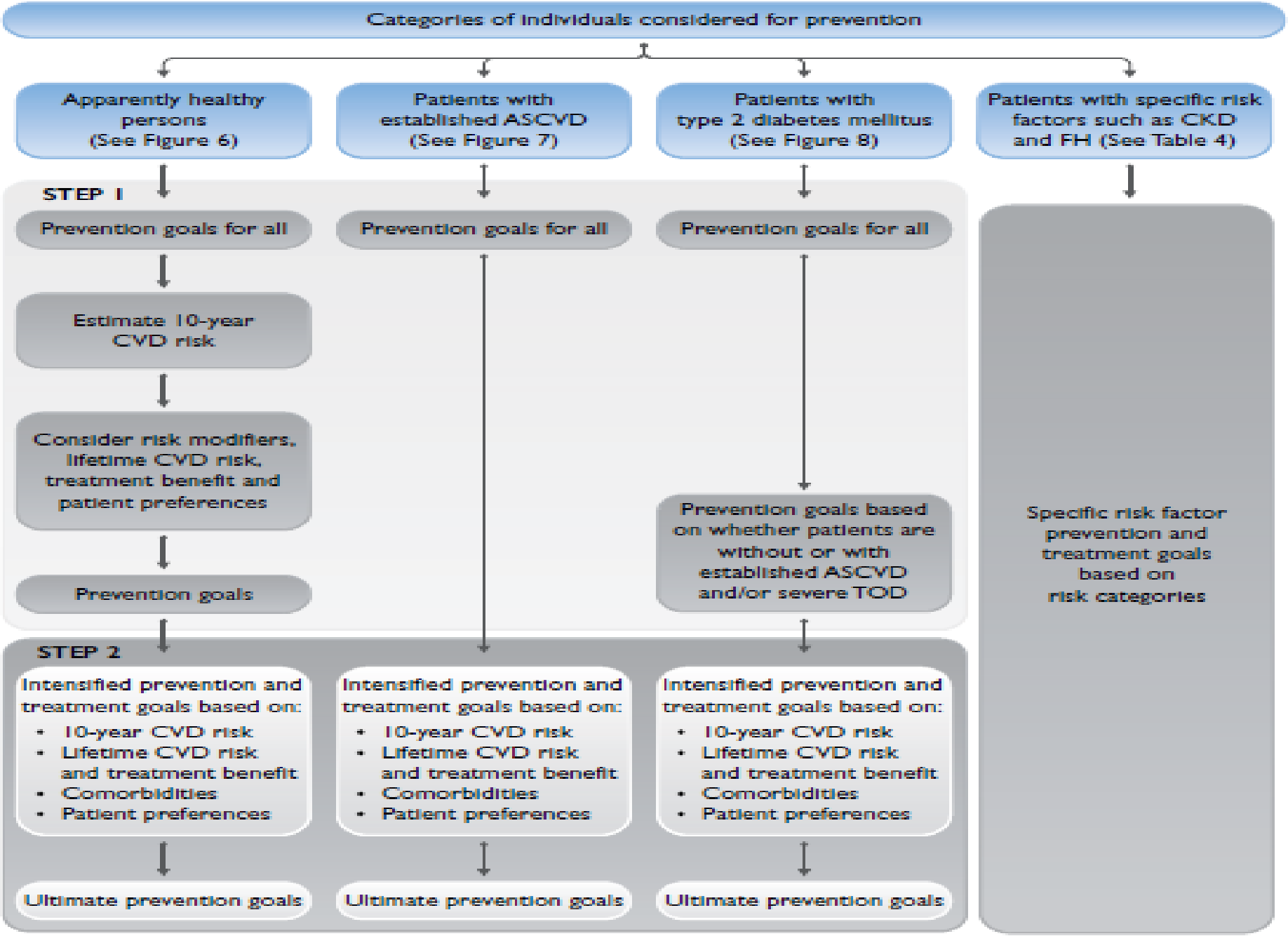
Conversely, men over 65 years and women over 75 years of age are almost always at **high** 10-year CVD risk.

Only between the ages of 55 and 75 years in women and 40 and 65 years in men does the 10-year CVD risk **vary** around commonly used thresholds for intervention. The age categories <50, 50-69, and >70 years should be used with common sense and **flexibility.**

Patient categories and associated cardiovascular disease risk.

Patient category	Subgroups	Risk categories	CVD risk and therapy benefit estimation
Apparently healthy persons			
Persons without established ASCVD, diabetes mellitus, CKD, Familial Hypercholesterolemia	<50 years	**Low- to high-risk**	10-year CVD risk estimation (SCORE2). Lifetime risk and benefit estimation of risk factor treatment (e.g. with the LIFE-CVD lifetime model) to facilitate the communication of CVD risk and treatment benefits.
	50-69 years	**Low- to very high-risk**	10-year CVD risk estimation (SCORE2). Lifetime benefit estimation of risk factor treatment (e.g. with the LIFE-CVD lifetime model) to facilitate the communication of treatment benefits.
	≥70 years	**Low- to very high-risk**	10-year CVD risk estimation (SCORE2-OP). Lifetime benefit estimation of risk factor treatment (e.g. with the LIFE-CVD lifetime model) to facilitate the communication of treatment benefits.
Patients with CKD			
CKD without diabetes or ASCVD	Moderate CKD (eGFR 30–44 mL/min/1.73 m^2 and ACR <30 **or** eGFR 45–59 mL/min/1.73 m^2 and ACR 30–300 **or** eGFR ≥60 mL/min/1.73 m^2 and ACR >300)	**High-risk**	N/A
	Severe CKD (eGFR<30 mL/min/1.73 m^2 **or** eGFR 30–44 mL/min/1.73 m^2 and ACR >30)	**Very high-risk**	N/A
Familial Hypercholesterolemia			
Associated with markedly elevated cholesterol levels	N/A	**High-risk**	N/A

Patients with type 2 diabetes mellitus			
Patients with type 1 DM above 40 years of age may also be classified according to these criteria	Patients with well controlled short-standing DM (e.g. <10 years), no evidence of TOD and no additional ASCVD risk factors	**Moderate-risk**	N/A
	Patients with DM without ASCVD and/or severe TOD, and not fulfilling the moderate risk criteria.	**High-risk**	Residual 10-year CVD risk estimation after general prevention goals (e.g. with the ADVANCE risk score or DIAL model). Consider lifetime CVD risk and benefit estimation of risk factor treatment (e.g. DIAL model).
	Patients with DM with established ASCVD and/or severe TOD:[87, 93-95] • eGFR <45 mL/min/1.73 m^2 irrespective of albuminuria • eGFR 45-59 mL/min/1.73 m^2 and microalbuminuria (ACR 30 -300 mg/g) • Proteinuria (ACR >300 mg/g) • Presence of microvascular disease in at least 3 different sites (e.g. microalbuminuria plus retinopathy plus neuropathy)	**Very high-risk**	Residual 10-year CVD risk estimation after general prevention goals (e.g. with the SMART risk score for established CVD or with the ADVANCE risk score or with the DIAL model). Consider lifetime CVD risk and benefit estimation of risk factor treatment (e.g. DIAL model).
Patients with established ASCVD			
Documented ASCVD, clinical or unequivocal on imaging. Documented clinical ASCVD includes previous AMI, ACS, coronary revascularization and other arterial revascularization procedures, stroke and TIA, aortic aneurysm and PAD. Unequivocally documented ASCVD on imaging includes plaque on coronary angiography or carotid ultrasound or on CTA. It does NOT include some increase in continuous imaging parameters such as intima–media thickness of the carotid artery.	N/A	**Very high-risk**	Residual CVD risk estimation after general prevention goals (e.g. 10-year risk with the SMART risk score for patients with established CVD or 1- or 2-year risk with EUROASPIRE risk score for patients with CHD). Consider lifetime CVD risk and benefit estimation of risk factor treatment (e.g. SMART-REACH model; or DIAL model if diabetes).


Categories of individuals considered for prevention
Apparently healthy persons (See Figure 6)
Patients with established ASCVD (See Figure 7)
Patients with type 2 diabetes mellitus (See Figure 8)
Patients with specific risk factors such as CKD and FH (See Table 4)
STEP 1
Prevention goals for all
Prevention goals for all
Prevention goals for all
Estimate 10-year CVD risk
Consider risk modifiers, lifetime CVD risk, treatment benefit and patient preferences
Prevention goals
Prevention goals based on whether patients are without or with established ASCVD and/or severe TOD
Specific risk factor prevention and treatment goals based on risk categories
STEP 2
Intensified prevention and treatment goals based on:
• 10-year CVD risk
• Lifetime CVD risk and treatment benefit
• Comorbidities
• Patient preferences
Intensified prevention and treatment goals based on:
• 10-year CVD risk
• Lifetime CVD risk and treatment benefit
• Comorbidities
• Patient preferences
Intensified prevention and treatment goals based on:
• 10-year CVD risk
• Lifetime CVD risk and treatment benefit
• Comorbidities
• Patient preferences
Ultimate prevention goals
Ultimate prevention goals
Ultimate prevention goals

four clusters of countries (low, moderate, high, and very high CVD risk) that are grouped based on national CVD mortality rates published by the WHO.

Low-risk countries:

Belgium, Denmark, France, Israel, Luxembourg, Norway, Spain, Switzerland, the Netherlands, and the United Kingdom (UK).

Moderate-risk countries:

Austria, Cyprus, Finland, Germany, Greece, Iceland, Ireland, Italy, Malta, Portugal, San Marino, Slovenia, and Sweden.

High-risk countries:

Albania, Bosnia and Herzegovina, Croatia, Czech Republic, Estonia, Hungary, Kazakhstan, Poland, Slovakia, and Turkey.

Very high-risk countries:

Algeria, Armenia, Azerbaijan, Belarus, Bulgaria, Egypt, Georgia, Kyrgyzstan, Latvia, Lebanon, Libya, Lithuania, Montenegro, Morocco, Republic of Moldova, Romania, Russian Federation, Serbia, Syria, The Former Yugoslav Republic (Macedonia), Tunisia, Ukraine, and Uzbekistan.

To estimate a person's 10-year risk of total CVD events, one must first identify the correct cluster of countries and the accompanying risk table for their sex, smoking status, and (nearest) age.Within that table, one then finds the cell nearest to the person's BP and non- HDL-C. Risk estimates then need to be adjusted upwards as the person approaches the next age category.

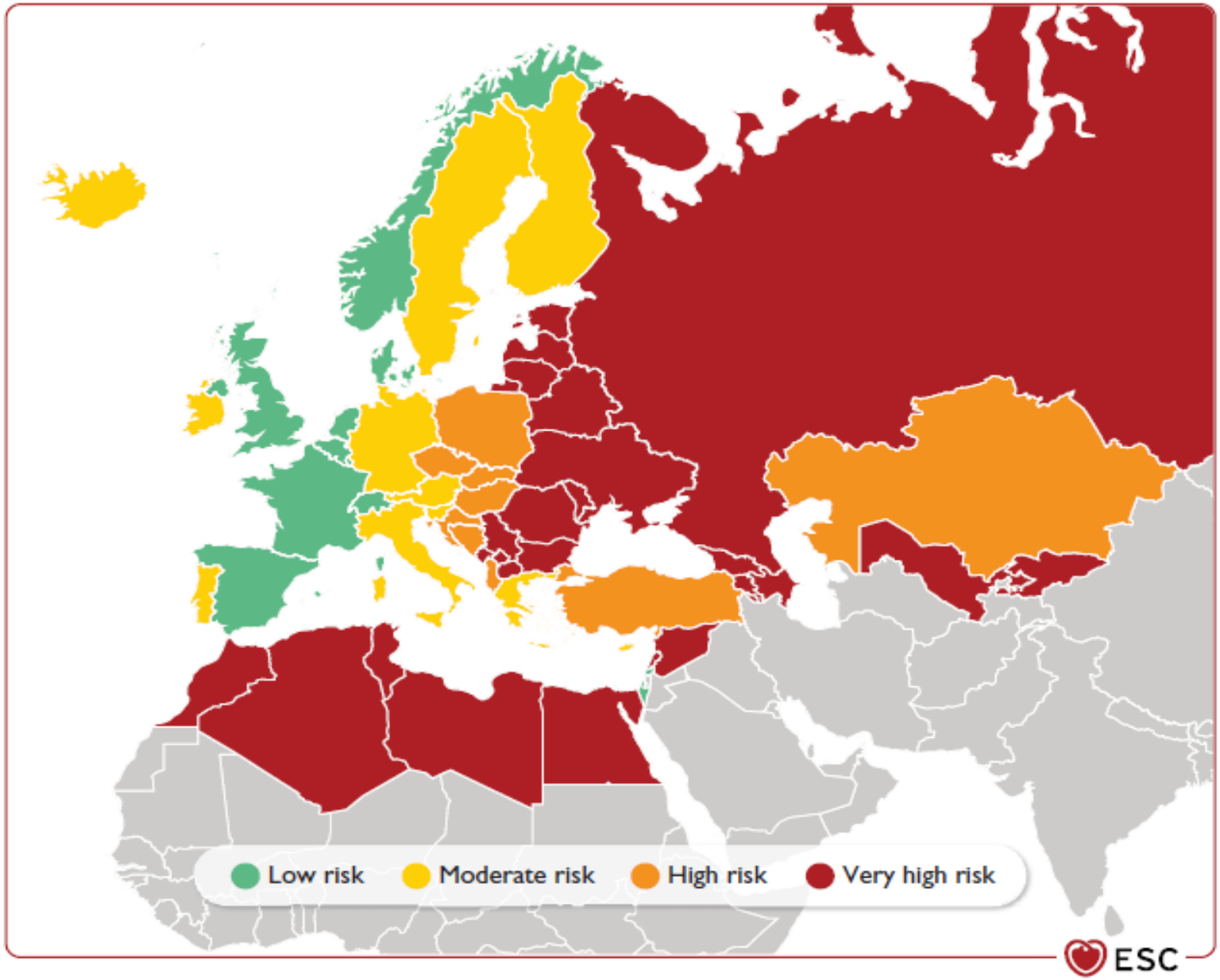


Figure 4 Risk regions based on World Health Organization cardiovascular mortality rates.[68,72,73]

Table 5 Cardiovascular disease risk categories based on SCORE2 and SCORE2-OP in apparently healthy people according to age

	<50 years	50–69 years	≥70 years[a]
Low-to-moderate CVD risk: risk factor treatment generally not recommended	<2.5%	<5%	<7.5%
High CVD risk: risk factor treatment should be considered	2.5 to <7.5%	5 to <10%	7.5 to <15%
Very high CVD risk: risk factor treatment generally recommended[a]	≥7.5%	≥10%	≥15%

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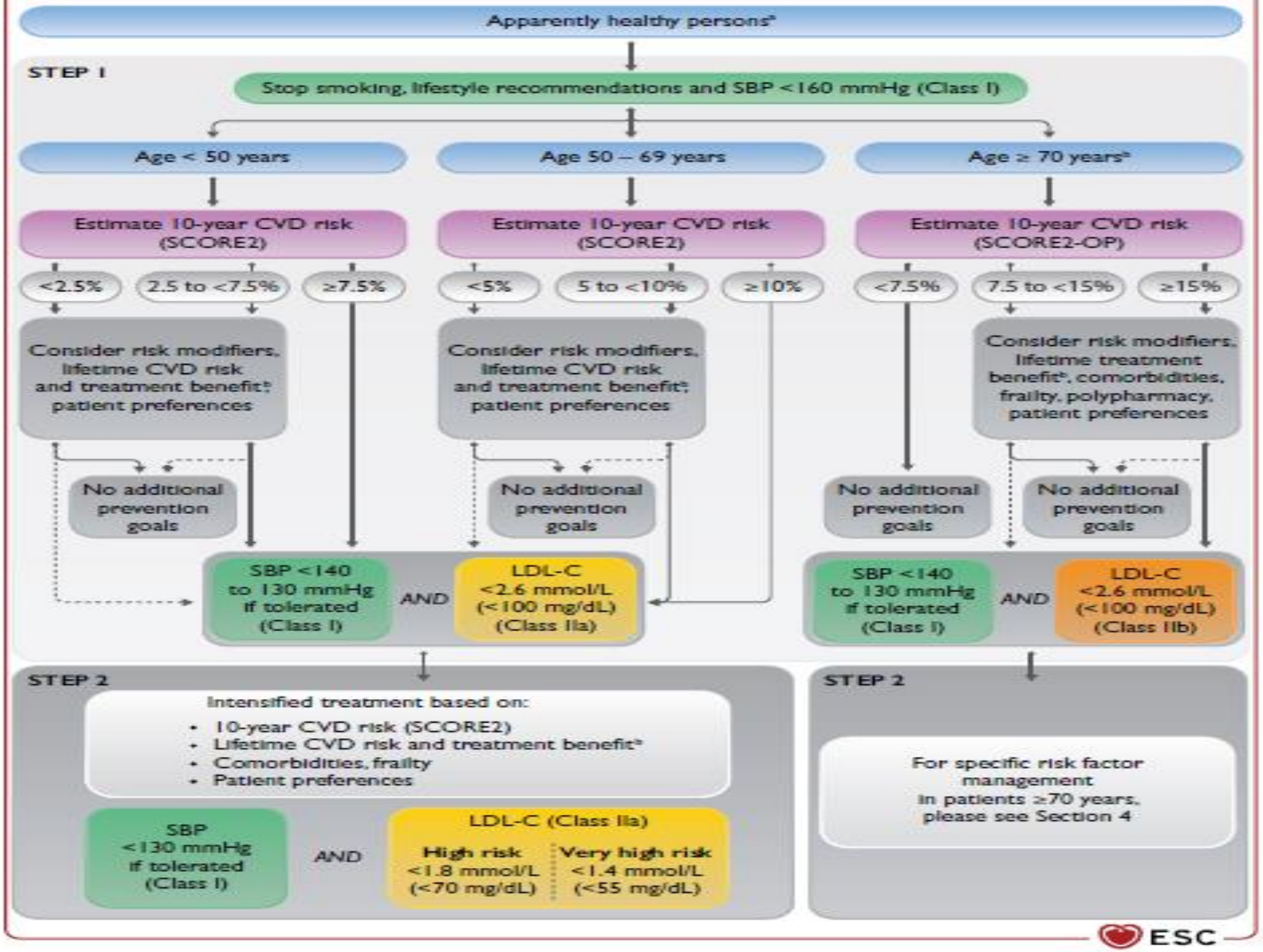
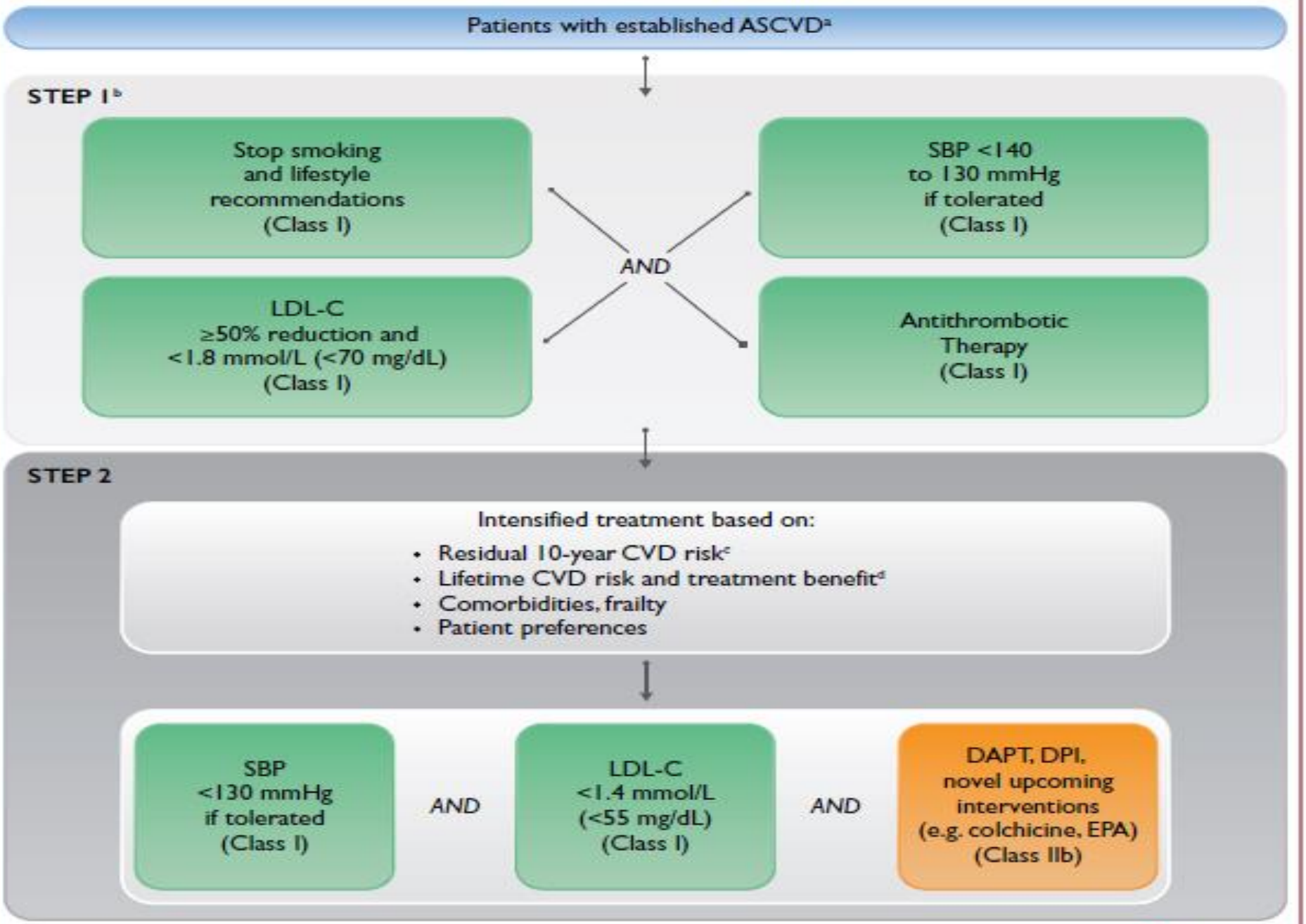
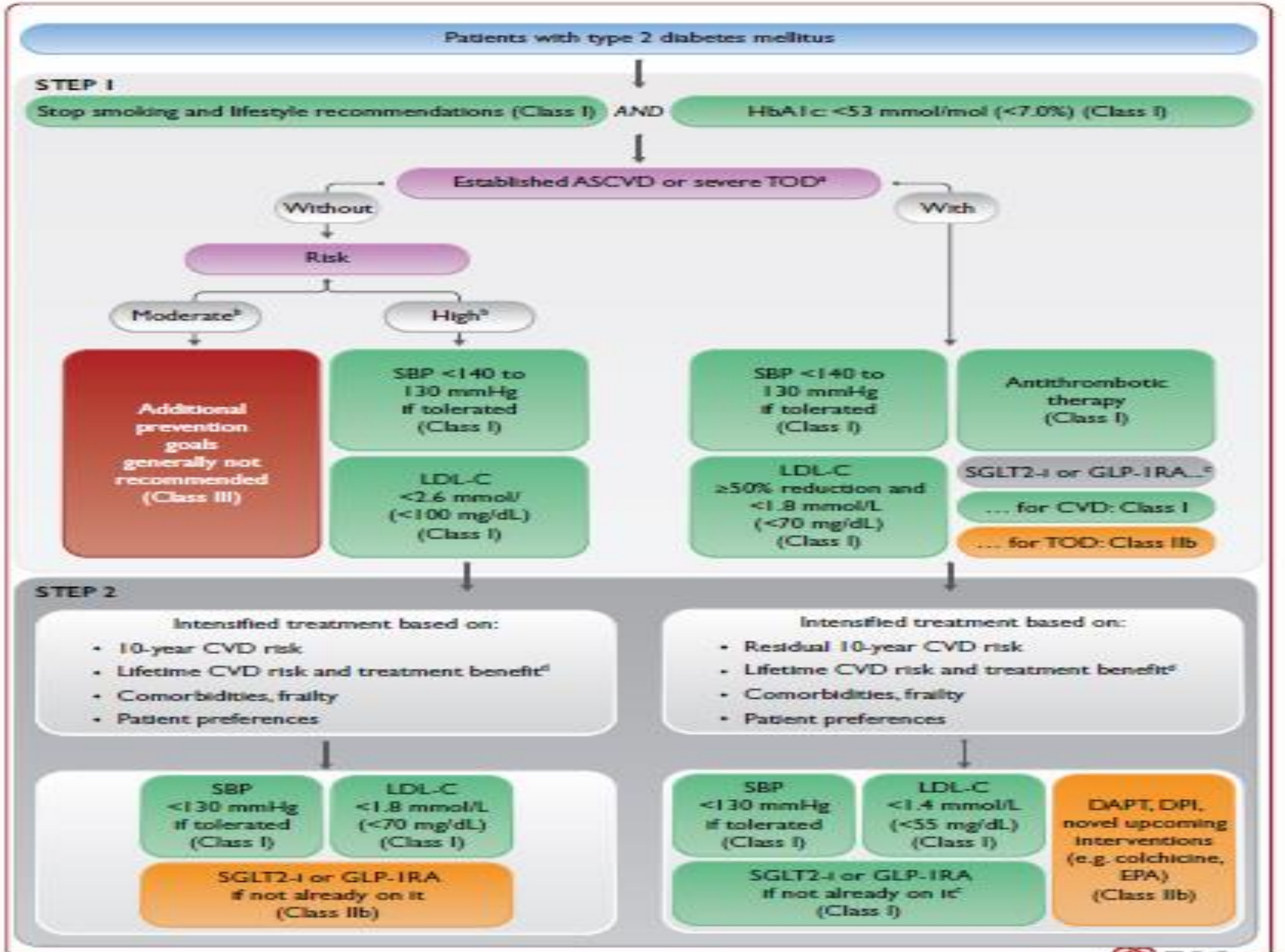
CVD = cardiovascular disease.

[a]In apparently healthy people ≥70 years old, the treatment recommendation for lipid-lowering drugs is Class IIb ('may be considered').

The division of the population into three distinct age groups (<50, 50–69, and ≥70 years) results in a discontinuous increase in risk thresholds for low-to-moderate, high, and very high risk. In reality, age is obviously continuous, and a sensible application of the thresholds in clinical practice would require some flexibility in handling these risk thresholds as patients move towards the next age group, or recently passed the age cut-off. *Figure 5* illustrates how a continuous increase in age relates to increasing risk thresholds, and may be used as a guide for daily practice.


Apparently healthy persons[a]
STEP 1
Stop smoking, lifestyle recommendations and SBP <160 mmHg (Class I)
Age < 50 years
Age 50 – 69 years
Age ≥ 70 years[a]
Estimate 10-year CVD risk (SCORE2)
Estimate 10-year CVD risk (SCORE2)
Estimate 10-year CVD risk (SCORE2-OP)
<2.5%
2.5 to <7.5%
≥7.5%
<5%
5 to <10%
≥10%
<7.5%
7.5 to <15%
≥15%
Consider risk modifiers, lifetime CVD risk and treatment benefit[b]; patient preferences
Consider risk modifiers, lifetime CVD risk and treatment benefit[b]; patient preferences
Consider risk modifiers, lifetime treatment benefit[b], comorbidities, frailty, polypharmacy, patient preferences
No additional prevention goals
No additional prevention goals
No additional prevention goals
No additional prevention goals
SBP <140 to 130 mmHg if tolerated (Class I)
AND
LDL-C <2.6 mmol/L (<100 mg/dL) (Class IIa)
SBP <140 to 130 mmHg if tolerated (Class I)
AND
LDL-C <2.6 mmol/L (<100 mg/dL) (Class IIb)
STEP 2
Intensified treatment based on:
• 10-year CVD risk (SCORE2)
• Lifetime CVD risk and treatment benefit[b]
• Comorbidities, frailty
• Patient preferences
SBP <130 mmHg if tolerated (Class I)
AND
LDL-C (Class IIa)
High risk <1.8 mmol/L (<70 mg/dL)
Very high risk <1.4 mmol/L (<55 mg/dL)
STEP 2
For specific risk factor management in patients ≥70 years, please see Section 4


Patients with established ASCVD[a]
STEP 1[b]
Stop smoking and lifestyle recommendations (Class I)
SBP <140 to 130 mmHg if tolerated (Class I)
AND
LDL-C ≥50% reduction and <1.8 mmol/L (<70 mg/dL) (Class I)
Antithrombotic Therapy (Class I)
STEP 2
Intensified treatment based on:
• Residual 10-year CVD risk[c]
• Lifetime CVD risk and treatment benefit[d]
• Comorbidities, frailty
• Patient preferences
SBP <130 mmHg if tolerated (Class I)
AND
LDL-C <1.4 mmol/L (<55 mg/dL) (Class I)
AND
DAPT, DPI, novel upcoming interventions (e.g. colchicine, EPA) (Class IIb)


Patients with type 2 diabetes mellitus
STEP 1
Stop smoking and lifestyle recommendations (Class I)
AND
HbA1c <53 mmol/mol (<7.0%) (Class I)
Established ASCVD or severe TOD[a]
Without
With
Risk
Moderate[b]
High[b]
Additional prevention goals generally not recommended (Class III)
SBP <140 to 130 mmHg if tolerated (Class I)
LDL-C <2.6 mmol/l (<100 mg/dL) (Class I)
SBP <140 to 130 mmHg if tolerated (Class I)
LDL-C ≥50% reduction and <1.8 mmol/L (<70 mg/dL) (Class I)
Antithrombotic therapy (Class I)
SGLT2-i or GLP-1RA...[c]
... for CVD: Class I
... for TOD: Class IIb
STEP 2
Intensified treatment based on:
• 10-year CVD risk
• Lifetime CVD risk and treatment benefit[d]
• Comorbidities, frailty
• Patient preferences
SBP <130 mmHg if tolerated (Class I)
LDL-C <1.8 mmol/L (<70 mg/dL) (Class I)
SGLT2-i or GLP-1RA if not already on it (Class IIb)
Intensified treatment based on:
• Residual 10-year CVD risk
• Lifetime CVD risk and treatment benefit[d]
• Comorbidities, frailty
• Patient preferences
SBP <130 mmHg if tolerated (Class I)
LDL-C <1.4 mmol/L (<55 mg/dL) (Class I)
SGLT2-i or GLP-1RA if not already on it[c] (Class I)
DAPT, DPI, novel upcoming interventions (e.g. colchicine, EPA) (Class IIb)
ESC

Potential risk modifiers

1-Psychosocial factors

Psychosocial stress is associated, in a **dose-response pattern**, with the development and progression of ASCVD, independently of conventional risk factors and sex.

Psychosocial stress has **direct** biological effects, but is also highly correlated with socioeconomic and behavioural risk factors (e.g. smoking, poor adherence).

Owing to the importance of stress symptoms among ASCVD patients, several guidelines and scientific statements recommend screening of ASCVD patients for psychological stress.

A recent prospective cohort study with a median follow-up of 8.4 years reported favourable effects of screening for **depression** on major ASCVD events

Box 2. Core topics for psychosocial assessment

Simultaneous diagnostic assessment	At least one in five patients carries a diagnosis of a mental disorder, usually presenting with bodily symptoms (e.g. chest tightness, shortness of breath). Therefore, physicians should be equally attentive to somatic as to emotional causes of symptoms.
Screening	Screening instruments assessing depression, anxiety, and insomnia are recommended (e.g. Patient Health Questionnaire,[116] see *Supplementary Table 5*).[117,118]
Stressors	There are simple questions to get into a conversation about significant stressors[112]: Are you bothered by stress at work, financial problems, difficulties in the family, loneliness, or any stressful events?
Need for mental health support	Are you interested in a referral to a psychotherapist or mental health service?

2-Ethnicity

Immigrants from South Asia (notably India and Pakistan) present higher CVD rates independent of other risk factors, whereas adjusted CVD risks appear lower in most other ethnic groups.

- Southern Asian: multiply the risk by 1.3 for Indians and Bangladeshis, and 1.7 for Pakistanis.
- Other Asian: multiply the risk by 1.1.
- Black Caribbean: multiply the risk by 0.85.
- Black African and Chinese: multiply the risk by 0.7.

3-Imaging

3.1 Coronary artery calcium

Coronary artery calcium (CAC) scoring can reclassify CVD risk upwards and downwards in addition to conventional risk factors, and may thus be considered in men and women with calculated risks around decision thresholds.

If CAC is detected, its extent should be compared with what would be expected for a patient of the **same sex and age**.

Higher-than-expected CAC increases the person's calculated risk, whereas absent or lower-than-expected CAC is associated with lower than calculated risk.

CAC scoring does **not** provide direct information on total plaque burden or stenosis severity, and can be low or even zero in middle-aged patients with soft non-calcified plaque.

3.2 Contrast computed tomography coronary angiography

CCTA allows identification of coronary stenoses and predicts cardiac events.

In the SCOT-HEART (Scottish Computed Tomography of the Heart) study, 5-year rates of coronary death or myocardial infarction were reduced when CCTA was used in patients with stable chest pain.

The relative reduction in myocardial infarction was similar in patients with non-cardiac chest pain.

Whether CCTA improves risk classification or adds prognostic value over CAC scoring is unknown.

3.3 Carotid ultrasound

Systematic use of intima-media thickness (IMT) to improve risk assessment **is not recommended** due to the lack of methodological standardization, and the absence of added value of IMT in predicting future CVD events, even in the intermediate-risk group.

Plaque is defined as the presence of a focal wall thickening that is **>50%** greater than the surrounding vessel wall, or as a focal region with an IMT measurement **>1.5 mm** that protrudes into the lumen.

Although the evidence is less extensive than it is for CAC, carotid artery plaque assessment using ultrasonography probably also reclassifies CVD risk, and may be considered as a risk modifier in patients at intermediate risk when a CAC score is not feasible.

3.4 Arterial stiffness

Arterial stiffness is commonly measured using either **aortic pulse wave velocity** or **arterial augmentation index**.

Studies suggest that arterial stiffness predicts future CVD risk and improves risk classification.

However, measurement difficulties and substantial publication bias argue against widespread use.

3.5 Ankle brachial index

Estimates are that 12-27% of middle-aged individuals have an ankle brachial index (ABI) <0.9, around 50-89% of whom do not have typical claudication.

An individual patient data metaanalysis concluded that the **reclassification potential of ABI was limited**, perhaps with the exception of women at **intermediate risk**.

4.Frailty

Frailty is a multidimensional state, independent of age and multimorbidity, that makes the individual more vulnerable to the effect of stressors.

It constitutes a functional risk factor for unfavourable outcomes, including both high CV and non-CV morbidity and mortality.

Frailty is not the same as **ageing** and the two should not be confused.

The incidence of frailty increases with *age*, but people of the same chronological age *can differ* significantly in terms of health status and vitality.

'Biological age' is much more important in the context of clinical status (including frailty features) and hard clinical outcomes (including CVD events).

Similarly, although the presence of **comorbidities** can exacerbate frailty within an individual, frailty is not the same as multimorbidity

Frailty is a **potential modifier** of global CVD risk.

The impact of frailty on CVD risk has been demonstrated across the spectrum of ASCVD, including people with ASCVD risk factors, patients with subclinical ASCVD, stable ASCVD, acute cerebral and coronary syndromes, and HF, with frailty itself rather than classical CVD risk factors predicting both all-cause and CVD mortality in the very old.

Importantly, the ability of frailty measures to improve CVD risk prediction has not been formally assessed.

Hence, **we do not recommendm** that frailty measures are integrated into formal CVD risk assessment.

Importantly, frailty may influence treatment. Non-pharmacological interventions (e.g. balanced nutrition, micronutrient supplementation, exercise training, social activation) aiming to prevent, attenuate, or reverse frailty are of utmost importance.

In terms of pharmacotherapy and device implantations, frailty assessment is not a method to determine the eligibility for any particular treatment, but rather serves to build an individualized care plan with predefined priorities.

Frail individuals often have comorbidities, polypharmacy, and may be more susceptible to drug side-effects and serious complications during invasive and surgical procedures.

5. Family history

Family history of premature CVD is a simple indicator of CVD risk, reflecting the genetic and environment interplay.

In the few studies that simultaneously assessed the effects of family history and genetics, family history **remained** significantly associated with CVD after adjusting for genetic scores.

However, family history only **marginally** improves the prediction of CVD risk beyond conventional ASCVD risk factors.

A family history of premature CVD is simple, inexpensive information that can trigger comprehensive risk assessment in individuals with a family history of premature CVD.

6. Genetics

The aetiology of ASCVD has a genetic component, but this information **is not** currently used in preventive approaches.

Advances on polygenic risk scores for risk stratification could increase the use of genetics in prevention.For ASCVD, there is, however, a lack of consensus regarding which genes and corresponding single nucleotide polymorphisms should be included, and whether to use risk factor-specific or outcome-specific polygenic risk scores.

Polygenic risk scoring has shown some potential to improve ASCVD risk prediction for primary prevention, but the incremental prediction accuracy is relatively modest and needs further evaluation in both men and women.

Additional evidence is also needed to evaluate the clinical utility of polygenic risk scores in other clinical settings, such as in patients with pre-existing ASCVD

7. Socioeconomic determinants

Low socioeconomic status and work stress are independently associated with ASCVD development and prognosis in both sexes.

The strongest association has been found between low income and CVD mortality, with a RR of 1.76 [95% confidence interval (CI) 1.452.14].

Work stress is determined by job strain (i.e. the combination of high demands and low control at work) and effort-reward imbalance.

There is preliminary evidence that the detrimental impact of work stress on ASCVD health is independent of conventional risk factors and their treatment.

8. Environmental exposure

Environmental exposures with CVD risk modifying potential include air and soil and water pollution as well as above-threshold noise levels.

Evaluating individual **cumulative** exposure to pollutants and noise remains challenging, but when available, might impact on individual risk assessment.

Components of outdoor air pollution include airborne particulate matter [PM; ranging in size from coarse particles 2.510 mm in diameter, to fine (<2.5 mm; PM2.5), and ultrafine (<0.1 mm)] and gaseous pollutants (e.g. ozone, nitrogen dioxide, volatile organic compounds, carbon monoxide, sulphur dioxide), produced primarily by combustion

of fossil fuels.

Soil and water pollutions are also CVD risk modifiers; increased exposure to lead, arsenic, and cadmium is associated with multiple CVD outcomes including hypertension, coronary heart disease (CHD), stroke, and CVD mortality.

Ambient PM pollution recently ranked as a leading modifiable mortality risk factor and also responsible for attributable disability adjusted life-years at the global level.

A recent model estimated that loss of life expectancy due to ambient air pollution is similar to, if not exceeding, that due to tobacco smoking, and accounts for a global excess mortality estimated at 8.8 million/year.

The short-term attributable effects on mortality are linked primarily to exposure to PM, nitrogen dioxide, and ozone, with an average **1.0% increase of all-cause mortality** for an increment of 10 lg/m$_3$in exposure to PM; the long-term effects are associated mainly with PM.

The evidence linking exposure to PM and CVD events is based on large-scale epidemiological studies and experimental studies.

Associations with ASCVD mortality vary, but the majority of cohort studies link long-term air pollution with an increased risk of fatal or non-fatal CAD, and with subclinical atherosclerosis.

Evidence suggests that reduction of PM is associated with improvements in inflammation, thrombosis, and oxidative stress, and a decrease in death from ischaemic heart disease.

As sufficiently precise individual exposure estimates are hard to obtain, formal risk reclassification is difficult to quantify at present.

Recommendations for cardiovascular disease risk related to air pollution

Recommendations	Class[a]	Level[b]
Patients at (very) high risk for CVD may be encouraged to try to avoid long-term exposure to regions with high air pollution.	IIb	C
In regions where people have long-term exposure to high levels of air pollution, (opportunistic) CVD risk screening programmes may be considered.	IIb	C

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CVD = cardiovascular disease.
[a]Class of recommendation.
[b]Level of evidence.

9. Biomarkers in blood or urine

Many biomarkers have been suggested to improve risk stratification.

Some may be causal [e.g. lipoprotein(a), reflecting a pathogenic lipid fraction], whereas others may reflect underlying mechanisms (e.g. Creactive protein reflecting inflammation) or indicate early cardiac damage (e.g. **natriuretic peptides or high-sensitivity cardiac troponin).**

In the 2016 Guidelines,we recommended against the routine use of biomarkers because most do not improve risk prediction, and publication bias seriously distorts the evidence.

New studies confirm that C-reactive protein has limited additional value.

There is renewed interest in lipoprotein(a), but it too provides limited additional value in terms of reclassification potential.

Cardiac biomarkers are promising,but further work is needed.

10. Body composition

In observational studies, all-cause mortality is minimal at a BMI of 20-25 kg/m_2, with a J- or U-shaped relation in current smokers.

Mendelian randomization analyses suggest a linear relation between BMI and mortality in never-smokers and a J-shaped relation in ever-smokers.

A meta-analysis concluded that both BMI and waist circumference are similarly strongly and continuously associated with ASCVD in the elderly and the young and in men and women.

Among those with established ASCVD, the evidence is contradictory.

Systematic reviews of patients with ACS or HF have suggested an 'obesity paradox' whereby obesity appears protective.

Which index of obesity is the best predictor of cardiovascular risk?

BMI can be measured easily and is used extensively to define categories of body weight.

Several measures of global and abdominal fat are available, of which waist circumference is the simplest to measure.

The WHO thresholds for waist circumference are widely accepted in Europe.

Two action levels are recommended:

- Waist circumference >94 cm in men and >80 cm in women: no further weight gain
- Waist circumference >102 cm in men and >88 cm in women: weight reduction advised.

The phenotype of 'metabolically healthy obesity', defined by the presence of obesity in the absence of metabolic risk factors, has gained interest. Long-term results support the notion that metabolically healthy obesity is a transient phase moving towards glucometabolic abnormalities rather than a specific 'state

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The clinical presentation of acute coronary syndromes (ACS) is broad.

from silent ischemia to cardiac arrest,electrical or haemodynamic instability with cardiogenic shock (CS) due to ongoing ischaemia or mechanical complications such as severe mitral regurgitation.

The leading symptom initiating the diagnostic and therapeutic cascade in patients with suspected ACS is acute **chest discomfort** described as pain, pressure, tightness, and burning

Chest pain-**equivalent** symptoms may include:

dyspnoea, syncope, LOC, epigastric pain, and pain in the left arm.

Based on the electrocardiogram (ECG), two groups of patients should be differentiated:

1- Patients with acute chest pain and persistent (>20 min) ST-segment elevation(STE-ACS).

2- Patients with acute chest discomfort but no persistent ST-segment elevation [non-ST-segment elevation ACS (NSTE-ACS)]

1- Patients with acute chest pain and persistent (>20 min) ST-segment elevation.

-This condition is termed ST-segment elevation ACS

-generally reflects an acute total or subtotal coronary occlusion.

-Most patients will ultimately develop ST-segment elevation myocardial infarction (STEMI).

-treatment in these patients is immediate reperfusion by:

a-primary percutaneous coronary intervention (P.PCI)

b-or, if not available in a timely manner, by fibrinolytic therapy.

2- Patients with acute chest discomfort but no persistent ST-segment elevation [non-ST-segment elevation ACS (NSTEACS)]

exhibit ECG changes that may include:

transient ST-segment elevation, persistent or transient ST-segment depression, T-wave inversion, flat T waves, or pseudonormalization of T waves; or the ECG may be normal.

The pathological correlate at the myocardial level is cardiomyocyte **necrosis** [non-ST-segment elevation myocardial infarction (NSTEMI)] or, less frequently, myocardial **ischaemia** without cell damage (unstable angina).

Although the main treatment in this group is angioplasty, the timing is different from STEMI.

A small proportion of patients may present with ongoing myocardial ischaemia, characterized by one or more of the following:

-recurrent or ongoing chest pain

-marked ST-segment depression on 12-lead ECG

-heart failure

-haemodynamic or electrical instability.

Due to immediate coronary angiography and, if appropriate, revascularization are indicated:

-the amount of myocardium in jeopardy and the risk of developing CS *and/or*

-malignant ventricular arrhythmias

Universal definition of myocardial infarction

Acute myocardial infarction (AMI) defines cardiomyocyte necrosis in a clinical setting consistent with acute myocardial ischaemia.

the detection of an increase and /or decrease of a cardiac biomarker, preferably high-sensitivity cardiac troponin (hs-cTn) T or I, with at least one value above the 99th percentile of the upper reference limit and at least one of the following:

(1) Symptoms of myocardial ischaemia.
(2) New ischaemic ECG changes.
(3) Development of pathologicalQ waves on ECG.
(4) Imaging evidence of loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology.
(5) Intracoronary thrombus detected on angiography or autopsy.

Type 1 myocardial infarction

Type 1 myocardial infarction (MI) is characterized by atherosclerotic plaque rupture, ulceration, fissure, or erosion with resulting intraluminal thrombus in one or more coronary arteries leading to decreased myocardial blood flow and/or distal embolization and subsequent myocardial necrosis.

The patient may have underlying severe coronary artery disease (CAD) but, on occasion (10% of cases), there may be non-obstructive coronary atherosclerosis or no angiographic evidence of CAD, particularly in women.

Type 2 myocardial infarction

Type 2 MI is myocardial necrosis in which a condition other than coronary plaque instability causes an imbalance between myocardial oxygen supply and demand.

Mechanisms include hypotension, hypertension, tachyarrhythmias, bradyarrhythmias, anaemia, hypoxaemia, but also by definition, coronary artery spasm, spontaneous coronary artery dissection (SCAD), coronary embolism, and coronary microvascular dysfunction.

Types 3-5 myocardial infarction

type 3 MI (MI resulting in death when biomarkers are not available) and

types 4 and 5 MI [related to PCI and coronary artery bypass grafting (CABG), respectively]

MI, even presenting as STEMI, also occurs in the absence of obstructive coronary artery disease (CAD) on angiography.

This type of MI is termed ‘myocardial infarction with non-obstructive coronary arteries’ (**MINOCA**).

Despite the fact that the majority of STEMI patients are classified as a type 1MI (with evidence of a coronary thrombus), some STEMIs fall into other MI types

Epidemiology of ST-segment elevation myocardial infarction and other ACSs

Worldwide, ischaemic heart disease is the single most common cause of death and its frequency is increasing.

In the last decade, cardiovascular disease has become the most important cause of death in the world and is known as a global epidemic.

Cardiovascular disease is reported to cause 30% of deaths and 11% of DALYs.

in Europe, there has been an overall trend for a reduction in ischaemic heart disease mortality over the past three decades.

Ischaemic heart disease now accounts for almost 1.8 million annual deaths, or 20% of all deaths in Europe, although with large variations between countries.

The relative incidences of STEMI and NSTEMI are decreasing and increasing, respectively.

The proportion of patients with NSTEMI in MI surveys increased from one third in 1995 to more than half in 2015, mainly accounted for by a refinement in the operational diagnosis of NSTEMI.

As opposed to STEMI, no significant changes are observed in the baseline characteristics of the NSTEMI population with respect to age and smoking, while diabetes, hypertension, and obesity increased substantially.

The mortality in STEMI patients is influenced by many factors, among them **advanced age**, **Killip class**, time **delay to treatment**, presence of emergency medical system (EMS)-based STEMI networks, treatment strategy, history of MI, diabetes mellitus, renal failure, number of diseased coronary arteries, and left ventricular ejection fraction (LVEF).

Several recent studies have highlighted a fall in acute and long-term mortality following STEMI in parallel with *greater use of reperfusion therapy*, *primary percutaneous coronary intervention* (PCI), *modern antithrombotic therapy*, and *secondary prevention*.

These guidelines aim to highlight the fact that women and men receive equal benefit from a reperfusion strategy and STEMI related therapy, and that both genders must be managed in a similar fashion.

Emergency care

in patients with acute ST- Elevation MI

The importance:

TIME is MUSCLE

The Best Strategy is

PPCI

Initial diagnosis

Management—including diagnosis and treatment—of STEMI starts from the point of first medical contact (FMC) by Faciliated early STEMI diagnisis and triage.

In this system with suspicion of myocardial ischaemia and ST segment elevation, reperfusion therapy needs to be initiated as soon as possible.

This is usually based on symptoms consistent with myocardial ischaemia (i.e. persistent chest pain) and signs [i.e. 12-lead electrocardiogram (ECG)].

Important clues are a history of CAD and radiation of pain to the neck, lower jaw, or left arm.

Some patients present with less-typical symptoms such as shortness of breath, nausea/vomiting, fatigue, palpitations, or syncope.

A reduction in chest pain after nitroglycerin (glyceryl trinitrate) administration can be misleading and is not recommended as a diagnostic manoeuvre.

In cases of symptom relief after nitroglycerin administration, another 12-lead ECG must be obtained.

A complete normalization of the ST-segment elevation after nitroglycerin administration, along with complete relief of symptoms, is suggestive of coronary spasm, with or without associated MI.

In these cases, an early coronary angiography (within 24 h) is recommended.

In cases of recurrent episodes of ST-segment elevation or chest pain, immediate angiography is required

If the ECG is equivocal or does not show evidence to support the clinical suspicion of MI, ECGs should be repeated and, when possible compared with previous recordings.

If interpretation of pre-hospital ECG is not possible on-site, field transmission of the ECG is recommended.

at least two contiguous leads with ST-segment elevation 2.5mm in men < 40 years, 2mm in men 40 years, or 1.5mm in women in leads V2–V3 and/or 1mm in the other leads [in the absence of left ventricular (LV) hypertrophy or left bundle branch block LBBB)].

In patients with inferior MI, it is recommended to record right precordial leads (V3R and V4R) seeking ST-segment elevation, to identify concomitant right ventricular (RV) infarction.

Likewise, ST-segment depression in leads V1–V3 suggests myocardial ischaemia, especially when the terminal T-wave is positive (ST-segment elevation equivalent), and confirmation by concomitant ST-segment elevation 0.5mm recorded in leads V7–V9 should be considered as a means to identify posterior MI.

The presence of a Q-wave on the ECG should not necessarily change the reperfusion strategy decision

Recommendations	Class[a]	Level[b]
ECG monitoring		
12-lead ECG recording and interpretation is indicated as soon as possible at the point of FMC, with a maximum target delay of 10 min.[36,38]	I	B
ECG monitoring with defibrillator capacity is indicated as soon as possible in all patients with suspected STEMI.[44,45]	I	B
The use of additional posterior chest wall leads (V_7–V_9) in patients with high suspicion of posterior MI (circumflex occlusion) should be considered.[8,46–49]	IIa	B
The use of additional right precordial leads (V_3R and V_4R) in patients with inferior MI should be considered to identify concomitant RV infarction.[8,43]	IIa	B
Blood sampling		
Routine blood sampling for serum markers is indicated as soon as possible in the acute phase but should not delay reperfusion treatment.[8]	I	C

In patients with LBBB, The presence of concordant ST-segment elevation (i.e. in leads with positive QRS deflections) appears to be one of the best indicators of ongoing MI with an occluded infarct artery.

Patients with a clinical suspicion of ongoing myocardial ischaemia and LBBB should be managed in a way similar to STEMI patients, regardless of whether the LBBB is previously known.

It is important to remark that the presence of a (presumed) new LBBB does not predict an MI per se.

Patients with MI and right bundle branch block (RBBB) have a poor prognosis.

It may be difficult to detect transmural ischaemia in patients with chest pain and RBBB.

Therefore, a primary PCI strategy(emergent coronary angiography and PCI if indicated) should be considered when persistent ischaemic symptoms occur in the presence of RBBB.

Non-diagnostic ECG:

Some patients with an acute coronary occlusion and ongoing MI may have an initial ECG without ST-segment elevation:

-very early after symptom onset (in which case, one should look for hyper-acute T-waves, which may precede ST-segment elevation).

-those with an occluded circumflex coronary artery

-acute occlusion of a vein graft, or

-left main disease

It is important to repeat the ECG or monitor for dynamic ST-segment changes.

Extending the standard 12-lead ECG with V7–V9 leads may identify some of these patients.

In any case, suspicion of ongoing myocardial ischaemia is an indication for a primary PCI strategy even in patients without diagnostic ST-segment elevation.

Table 3 lists the atypical ECG presentations

that should prompt a primary PCI strategy in patients with ongoing symptoms consistent with myocardial ischaemia.

Table 3 Atypical electrocardiographic presentations that should prompt a primary percutaneous coronary intervention strategy in patients with ongoing symptoms consistent with myocardial ischaemia

Bundle branch block Criteria that can be used to improve the diagnostic accuracy of STEMI in LBBB[50]: • Concordant ST-segment elevation ≥1 mm in leads with a positive QRS complex • Concordant ST-segment depression ≥1 mm in V_1–V_3 • Discordant ST-segment elevation ≥5 mm in leads with a negative QRS complex The presence of RBBB may confound the diagnosis of STEMI
Ventricular paced rhythm During RV pacing, the ECG also shows LBBB and the above rules also apply for the diagnosis of myocardial infarction during pacing; however, they are less specific
Isolated posterior myocardial infarction Isolated ST depression ≥0.5 mm in leads V_1–V_3 and ST-segment elevation (≥0.5 mm) in posterior chest wall leads V_7–V_9
Ischaemia due to left main coronary artery occlusion or multivessel disease ST depression ≥1 mm in eight or more surface leads, coupled with ST-segment elevation in aVR and/or V_1, suggests left main-, or left main equivalent- coronary obstruction, or severe three vessel ischaemia

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Isolated posterior MI:

In AMI of the inferior and basal portion of the heart, often corresponding to the left circumflex territory, isolated ST-segment depression 0.5 mm in leads V1–V3 represents the dominant finding. These should be managed as a STEMI.

The use of additional posterior chest wall leads [elevation V7–V9 0.5mm (1mm in men, 40 years old)] is recommended to detect STsegment elevation consistent with inferior and basal MI.

Left main coronary obstruction:

The presence of ST depression ımm in eight or more surface leads (inferolateral ST depression), coupled with ST-segment elevation in aVR and/or V1, suggests multivessel ischemia or left main coronary artery obstruction, particularly if the patient presents with haemodynamic compromise.

Table 10 Diagnostic criteria for myocardial infarction with non-obstructive coronary arteries (adapted from Agewall et *al*[12])

The diagnosis of MINOCA is made immediately upon coronary angiography in a patient presenting with features consistent with an AMI, as detailed by the following criteria:
(1) Universal AMI criteria[8]
(2) Non-obstructive coronary arteries on angiography, defined as no coronary artery stenosis ≥50% in any potential IRA
(3) No clinically overt specific cause for the acute presentation

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AMI = acute myocardial infarction; IRA = infarct-related artery; MINOCA = myocardial infarction with non-obstructive coronary arteries.

Blood sampling for serum markers is routinely carried out in the acute phase. This is indicated, **but should not delay the reperfusion strategy/treatment**.

If in doubt regarding the possibility of acute evolving MI, emergency imaging aids the provision of timely reperfusion therapy to these patients.

Recommendations for the use of **echocardiography** for initial diagnosis are described in later slides

Summary of indications for imaging and stress testi in ST-elevation myocardial infarction patients

Recommendations	Class[a]	Level[b]
At presentation		
Emergency echocardiography is indicated in patients with cardiogenic shock and/or haemodynamic instability or suspected mechanical complications without delaying angiography.[295]	I	C
Emergency echocardiography before coronary angiography should be considered if the diagnosis is uncertain.[295]	IIa	C
Routine echocardiography that delays emergency angiography is not recommended.[295]	III	C
Coronary CT angiography is not recommended	III	C
During hospital stay (after primary PCI)		
Routine echocardiography to assess resting LV and RV function, detect early post-MI mechanical complications, and exclude LV thrombus is recommended in all patients.[296,297]	I	B
Emergency echocardiography is indicated in haemodynamically unstable patients.[295]	I	C

During hospital stay (after primary PCI)		
Routine echocardiography to assess resting LV and RV function, detect early post-MI mechanical complications, and exclude LV thrombus is recommended in all patients.[296,297]	I	B
Emergency echocardiography is indicated in haemodynamically unstable patients.[295]	I	C
When echocardiography is suboptimal/inconclusive, an alternative imaging method (CMR preferably) should be considered.	IIa	C
Either stress echo, CMR, SPECT, or PET may be used to assess myocardial ischaemia and viability, including in multivessel CAD.[1,298–300]	IIb	C
After discharge		
In patients with pre-discharge LVEF ≤40%, repeat echocardiography 6–12 weeks after MI, and after complete revascularization and optimal medical therapy, is recommended to assess the potential need for primary prevention ICD implantation.[3,296]	I	C
When echo is suboptimal or inconclusive, alternative imaging methods (CMR preferably) should be considered to assess LV function.	IIa	C

If echocardiography is not available or if doubts persist after echo, a primary PCI strategy is indicated

(including immediate transfer to a PCI centre if the patient is being treated in a non-PCI centre).

In the STEMI emergency setting, there is no role for routine computed tomography (CT).

Use of CT should be confined to selected cases where acute aortic dissection or pulmonary embolism is suspected, but CT is not recommended if STEMI diagnosis is likely.

Some non-AMI conditions can present with symptoms and ECG findings similar to STEMI.

An emergency coronary angiography is therefore indicated in these cases.

Pre-hospital logistics of care:
-Delays

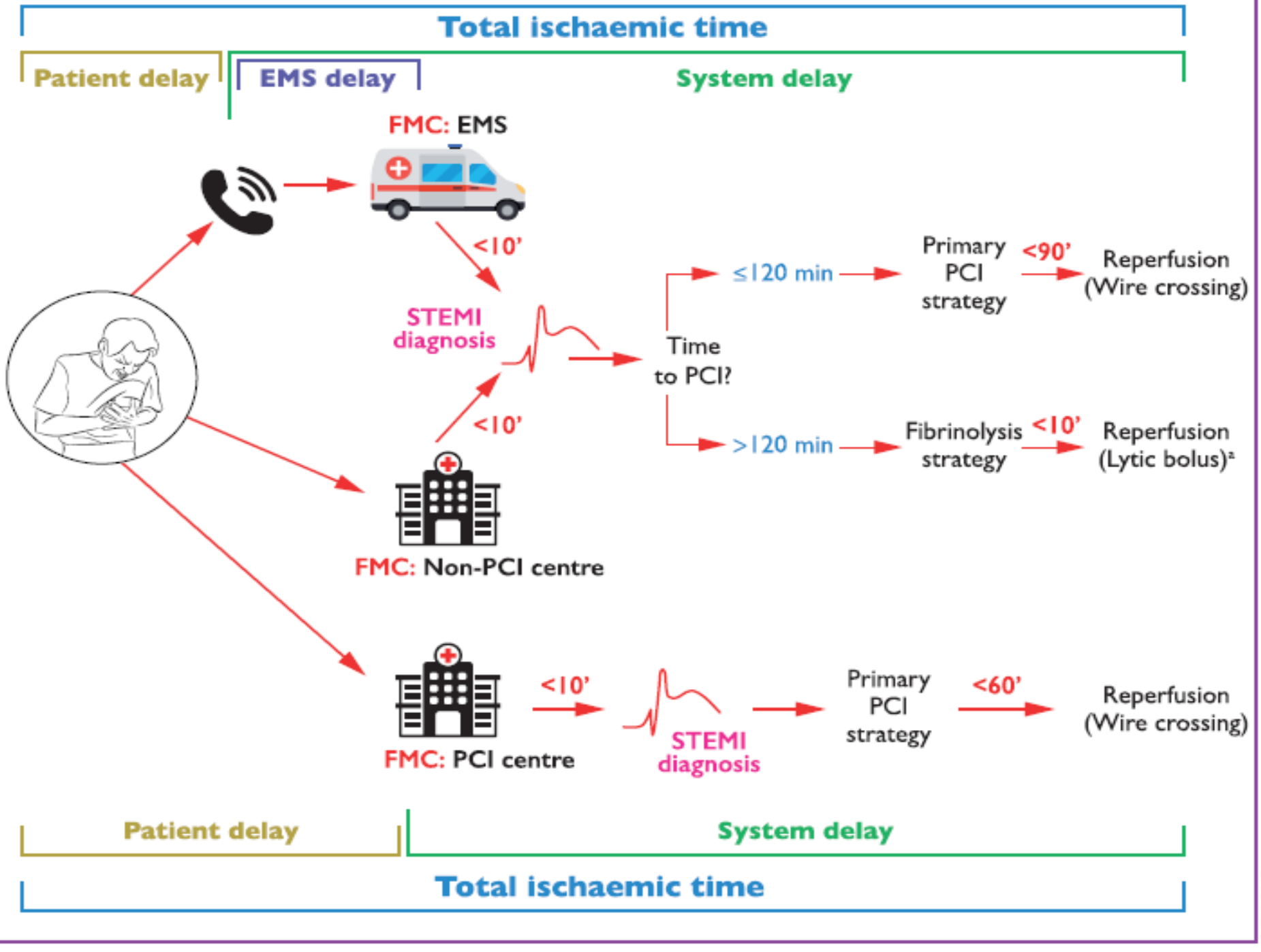
Treatment delays are the most easily audited index of quality of care in STEMI

they should be recorded in every system providing care to STEMI patients and be reviewed regularly.

Components of the ischaemic time:

1- delays of initial diagnosis(patient&EMS&hospital delay)

2-delay of selection of reperfusion strategy


Total ischaemic time
Patient delay
EMS delay
System delay
FMC: EMS
<10'
STEMI diagnosis
Time to PCI?
≤120 min
Primary PCI strategy
<90'
Reperfusion (Wire crossing)
>120 min
Fibrinolysis strategy
<10'
Reperfusion (Lytic bolus)[a]
<10'
FMC: Non-PCI centre
FMC: PCI centre
<10'
STEMI diagnosis
Primary PCI strategy
<60'
Reperfusion (Wire crossing)
Patient delay
System delay
Total ischaemic time

To minimize delays:

recod of tims carefully and regularly check them

1- Patient delay: increase public awareness of how to recognize common symptoms of AMI and to call the emergency services(the most difficaut).

2- diagnosis delay: In hospitals and EMS participating in the care of STEMI patients, the goal is to reduce the delay between FMC and STEMI diagnosis to <_ 10 min.

3-treatment delay: STEMI diagnosis refers to the time when the ECG is interpreted as ST-segment elevation or equivalent and it is the time zero to guide appropriate therapy.

4-immediate activation of the catheterization laborator When STEMI diagnosis is made in the pre-hospital setting (EMS)

5-bypass the emergency department and bring the patient straight to the catheterization laboratory(20 min).

6-For patients presenting in a non-PCI centre, **door-in to door-out time**(duration between arrival of the patient at the hospital to discharge of the patient in an ambulance en route to the PCI centre)<_30min .

Emergency medical system

An EMS with an easily recalled and well publicized unique medical dispatching number (112 for most medical emergencies across Europe and 115 in Iran) is important to speed up activation.

Parallel circuits for referral and transport of patients with a STEMI that bypass the EMS should be avoided.

The ambulance system has a critical role in the early management of STEMI patients and it is not only a mode of transport but also a system to enhance early initial diagnosis, triage, and treatment.

It is indicated that all ambulances in the EMS are equipped with ECG recorders, defibrillators, and at least one person trained in advanced life support.

The quality of the care provided depends on the training of the staff involved.

It is indicated that all ambulance personnel are trained to recognize the symptoms of an AMI, administer oxygen when appropriate, relieve pain, and provide basic life support.

Ambulance staff should be able to:

- record an ECG for diagnostic purposes
- either interpret it
- transmit it
- so that it can be reviewed by experienced staff in a coronary care unit (CCU)/ ICCU or elsewhere and establish a STEMI diagnosis.

Paramedics trained to administer fibrinolytics do so safely and effectively.

As pre-hospital fibrinolysis is indicated in patients presenting early when anticipated STEMI diagnosis to PCI-mediated reperfusion time is > 120min, ongoing training of paramedics to undertake.

these functions is recommended, even in the current setting of primary PCI.

Organization of ST-segment elevation myocardial infarction treatment in networks

Optimal treatment of STEMI should be based on the implementation of networks between hospitals ('hub' and 'spoke') with various levels of technology, linked by a prioritized and efficient ambulance service.

The goal of these networks is to provide optimal care while minimizing delays, thereby improving clinical outcomes.

Cardiologists should actively collaborate with all stakeholders, particularly emergency physicians, in establishing such networks.

The main features of such a network are:

- Clear definition of geographic areas of responsibility

- Shared written protocols, based on risk stratification and transportation by a trained physician, nurse, or paramedic staff in appropriately equipped ambulances or helicopters.

Pre-hospital triage of STEMI patients to the appropriate institution, bypassing non-PCI hospitals or hospitals without a 24 h a day, 7 days a week (24/7) primary PCI programme.

On arrival at the appropriate hospital, the patient should immediately be taken to the catheterization laboratory, bypassing the emergency department.

Patients presenting to a non-PCI-capable hospital and awaiting transportation for primary or rescue PCI must be attended in an appropriately monitored and staffed area.

If the diagnosis of STEMI has not been made by the ambulance crew and the ambulance arrives at a non-PCI-capable hospital, the ambulance should await the diagnosis and, if a STEMI diagnosis is made, should continue to a PCI-capable hospital.

To maximize staff experience, primary PCI centres should perform the procedure systematically on a 24/7 basis for all STEMI patients.

Othermodels, although not ideal,may include weekly or daily rotation of primary PCI centres or multiple primary PCI centres in the same region.

Hospitals that cannot offer a 24/7 service for primary PCI should be allowed to perform primary PCI in patients already admitted for another reason who develop STEMI during their hospital stay.

However, these hospitals should be discouraged from initiating a service limited to daytime- or within-hours primary PCI, as this may generate confusion with the EMS operators and may affect the STEMI diagnosis-to-reperfusion time and the quality of intervention of focused 24/7 true primary PCI centres.

Therefore,

it is indicated that the EMS transports STEMI patients to hospitals with an established interventional cardiology programme available 24/7, if necessary bypassing a non-PCI-capable hospital (if the transfer time is within the recommended time-windows for primary PCI; see Figure 3).

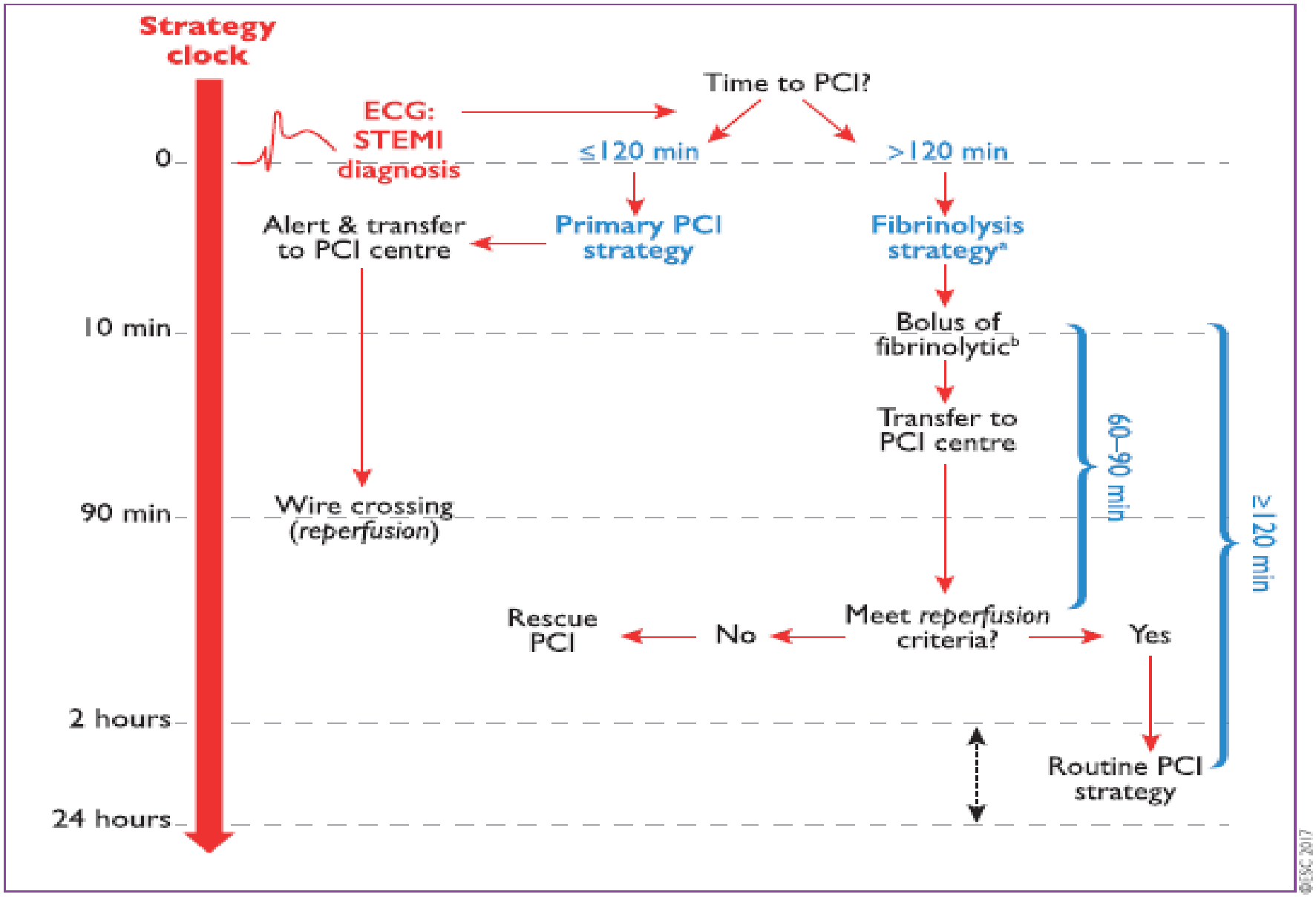


Figure 3 Maximum target times according to reperfusion strategy selection in patients presenting via EMS or in a non-PCI centre. ECG = electro-

Geographic areas where the expected transfer time to the primary PCI centre makes it impossible to achieve the maximal allowable delays indicated in the recommendations (Figure 2, Total ischemic time slide) should develop systems for rapid fibrinolysis, at the place of STEMI diagnosis, with subsequent immediate transfer to primary PCI centres.

Such networks increase the proportion of patients receiving reperfusion with the shortest possible treatment delay.

The quality of care, time delays, and patient outcomes should be measured and compared at regular intervals for improvement.

General practitioners

In some countries, general practitioners play a role in the early care of patients with AMI and are often the first to be contacted by the patients.

If general practitioners respond quickly they can be very effective, as they usually know the patient and can perform and interpret the ECG.

Their first task after the STEMI diagnosis should be to alert the EMS.

In addition, they can administer opioids and antithrombotic drugs (including fibrinolytics, if that management strategy is indicated),and can undertake defibrillation if needed.

However, in most settings, consultation with a general practitioner—instead of a direct call to the EMS—will increase pre-hospital delay.

Therefore,

in general,

the public should be educated to call the EMS

rather than

the primary care physician for symptoms

suggestive of MI.

Logistics of pre-hospital care

Recommendations	Class[a]	Level[b]
It is recommended that the pre-hospital management of STEMI patients is based on regional networks designed to deliver reperfusion therapy expeditiously and effectively, with efforts made to make primary PCI available to as many patients as possible.[100]	I	B
It is recommended that primary PCI-capable centres deliver a 24/7 service and are able to perform primary PCI without delay.[18,103,104]	I	B
It is recommended that patients transferred to a PCI-capable centre for primary PCI bypass the emergency department and CCU/ICCU and are transferred directly to the catheterization laboratory.[92,107–110]	I	B
It is recommended that ambulance teams are trained and equipped to identify STEMI (with use of ECG recorders and telemetry as necessary) and administer initial therapy, including fibrinolysis when applicable.[95]	I	C

It is recommended that ambulance teams are trained and equipped to identify STEMI (with use of ECG recorders and telemetry as necessary) and administer initial therapy, including fibrinolysis when applicable.[95]	I	C
It is recommended that all hospitals and EMS participating in the care of patients with STEMI record and audit delay times and work to achieve and maintain quality targets.[105–107]	I	C
It is recommended that EMS transfer STEMI patients to a PCI-capable centre, bypassing non-PCI centres.	I	C
It is recommended that EMS, emergency departments, and CCU/ICCU have a written updated STEMI management protocol, preferably shared within geographic networks.	I	C
It is recommended that patients presenting to a non-PCI-capable hospital and awaiting transportation for primary or rescue PCI are attended in an appropriately monitored area (e.g. the emergency department, CCU/ICCU, or intermediate care unit).	I	C

Table 5 Summary of important time targets

Intervals	Time targets
Maximum time from FMC to ECG and diagnosis[a]	≤10 min
Maximum expected delay from STEMI diagnosis to primary PCI (wire crossing) to choose primary PCI strategy over fibrinolysis (if this target time cannot be met, consider fibrinolysis)	≤120 min
Maximum time from STEMI diagnosis to wire crossing in patients presenting at primary PCI hospitals	≤60 min
Maximum time from STEMI diagnosis to wire crossing in transferred patients	≤90 min
Maximum time from STEMI diagnosis to bolus or infusion start of fibrinolysis in patients unable to meet primary PCI target times	≤10 min
Time delay from start of fibrinolysis to evaluation of its efficacy (success or failure)	60–90 min
Time delay from start of fibrinolysis to angiography (if fibrinolysis is successful)	2–24 hours

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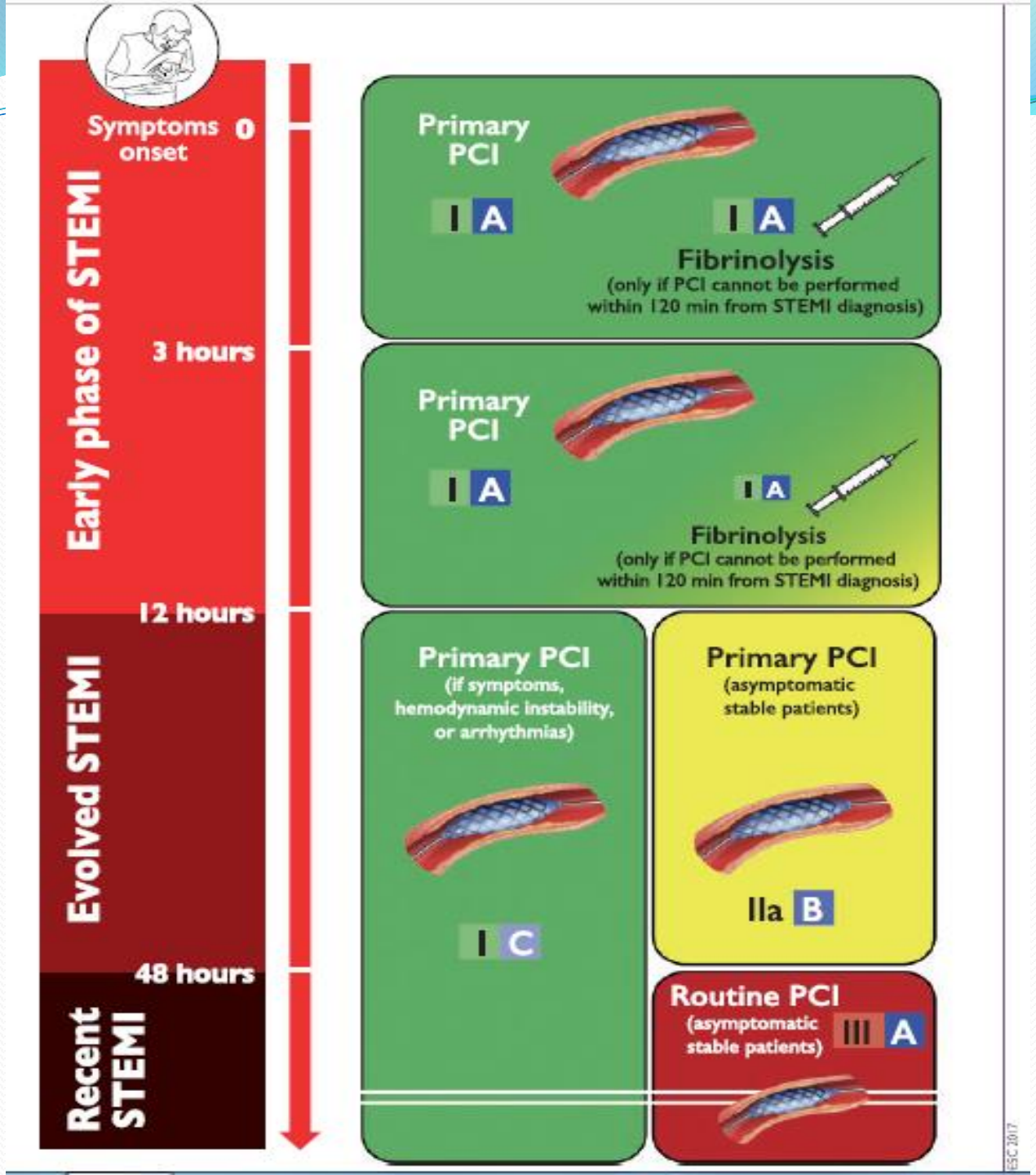

Symptoms onset
0
Early phase of STEMI
3 hours
Evolved STEMI
12 hours
Recent STEMI
48 hours
Primary PCI
I A
I A
Fibrinolysis
(only if PCI cannot be performed within 120 min from STEMI diagnosis)
Primary PCI
I A
I A
Fibrinolysis
(only if PCI cannot be performed within 120 min from STEMI diagnosis)
Primary PCI
(if symptoms, hemodynamic instability, or arrhythmias)
I C
Primary PCI
(asymptomatic stable patients)
IIa B
Routine PCI
(asymptomatic stable patients)
III A
ESC 2017

Figure 4 Reperfusion strategies in the infarct-related artery according to time from symptoms onset. PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction.

In early presenters (i.e. those with STEMI diagnosis within 3 hours from symptoms onset), a primary PCI strategy is the reperfusion strategy of choice. If the anticipated time from STEMI diagnosis to PCI-mediated reperfusion is > 120 min, then immediate fibrinolysis is indicated. After 3 hours (and up to 12 hours) of symptoms onset, the later the patient presents, the more consideration should be given to a primary PCI strategy as opposed to administering fibrinolytic therapy. In evolved STEMI (12–48 hours after symptoms onset), a routine primary PCI strategy (urgent angiography and subsequent PCI if indicated) should be considered in all patients. After 48 hours (recent STEMI) angiography should be performed but routine PCI of a total occluded IRA is not recommended. Regardless of the time from symptoms onset, the presence of ongoing symptoms suggestive of ischaemia, haemodynamic instability, or lifethreatening arrhythmias is an indication for a primary PCI strategy.

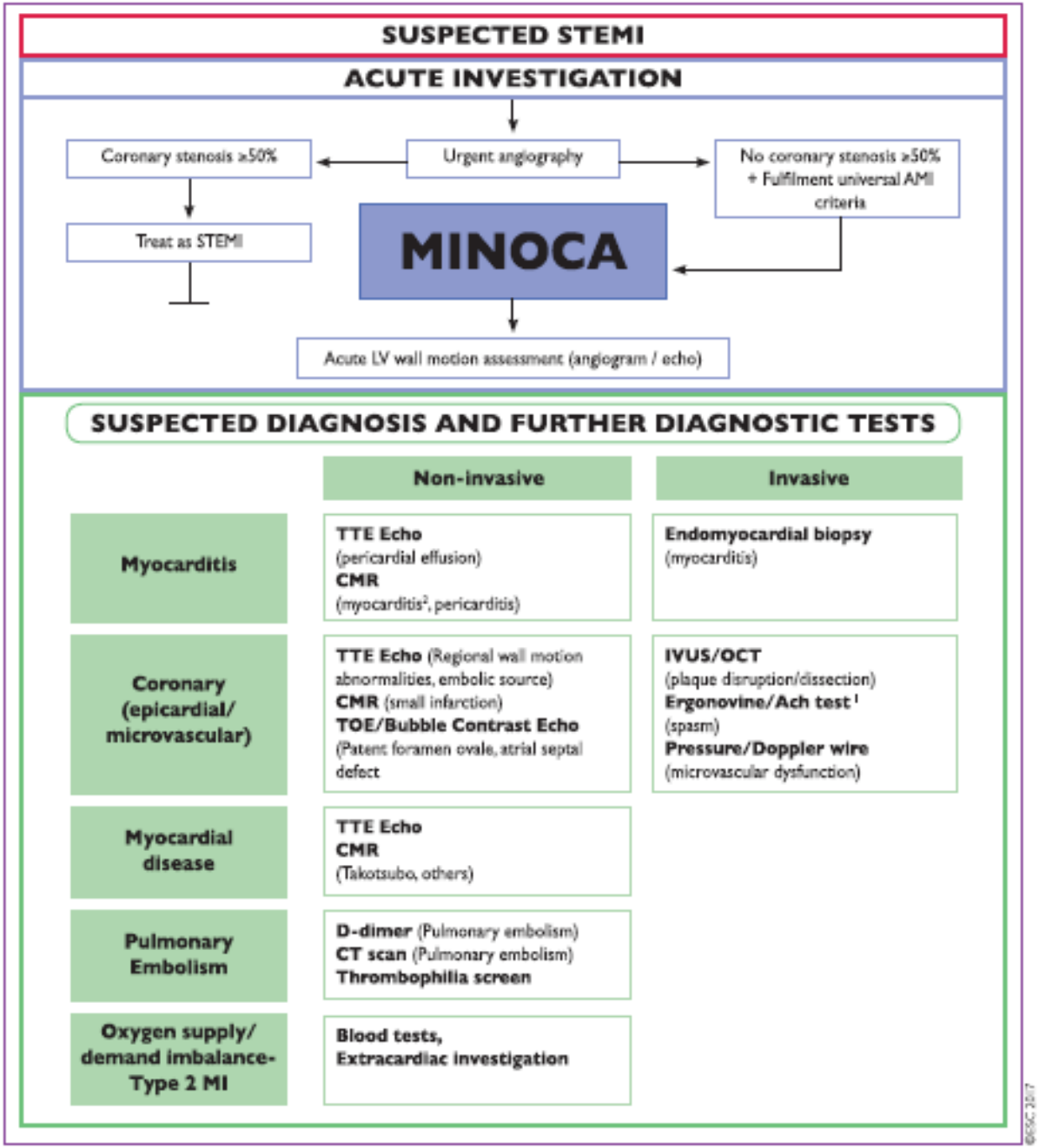

SUSPECTED STEMI
ACUTE INVESTIGATION
Coronary stenosis ≥50%
Urgent angiography
No coronary stenosis ≥50%
+ Fulfilment universal AMI criteria
Treat as STEMI
MINOCA
Acute LV wall motion assessment (angiogram / echo)
SUSPECTED DIAGNOSIS AND FURTHER DIAGNOSTIC TESTS
Non-invasive
Invasive
Myocarditis
TTE Echo (pericardial effusion)
CMR (myocarditis[2], pericarditis)
Endomyocardial biopsy (myocarditis)
Coronary (epicardial/ microvascular)
TTE Echo (Regional wall motion abnormalities, embolic source)
CMR (small infarction)
TOE/Bubble Contrast Echo (Patent foramen ovale, atrial septal defect
IVUS/OCT (plaque disruption/dissection)
Ergonovine/Ach test[1] (spasm)
Pressure/Doppler wire (microvascular dysfunction)
Myocardial disease
TTE Echo
CMR
(Takotsubo, others)
Pulmonary Embolism
D-dimer (Pulmonary embolism)
CT scan (Pulmonary embolism)
Thrombophilia screen
Oxygen supply/ demand imbalance- Type 2 MI
Blood tests,
Extracardiac investigation

Figure 7 Diagnostic test flow chart in MINOCA. CMR = Cardiac Magnetic Resonance; IVUS = IntraVascular UltraSound; LV = Left Ventricle; MINOCA = Myocardial Infarction with Non-Obstructed Coronary Arteries; OCT = Optical Coherence Tomography; STEMI = ST segment Elevation Myocardial Infarction; TOE = Trans-Oesofageal Echocardiography; TTE = Trans-Thoracic Echocardiography. Takotsubo syndrome cannot be diagnosed with certainty in the acute phase as the definition requires follow up imaging to document recovery of left ventricular function. IVUS and OCT frequently show more atherosclerotic plaque than may be appreciated on angiography. They also increase sensitivity for dissection. If intracoronary imaging is to be performed, it is appropriate to carry out this imaging at the time of the acute cardiac catheterization, after diagnostic angiography. Patients should be made aware of the additional information the test can provide and the small increase in risk associated with intracoronary imaging.

1 • Provocative testing for coronary artery spasm might be considered in selected patients with a recent AMI with suspected vasospastic angina. Provocative manoeuvres have to be always performed by operators with experience and not necessarily in the acute phase of STEMI.

2 • Clinically suspected myocarditis by ESC Task Force criteria = No angiographic stenosis ≥50% plus non ischemic pattern on CMR. Definite myocarditis by ESC Task Force criteria = No angiographic stenosis ≥50% plus endomyocardial biopsy confirmation (histology, immunohistology, polymerase-chain reaction based techniques to search for genome of infectious agents, mainly viruses).

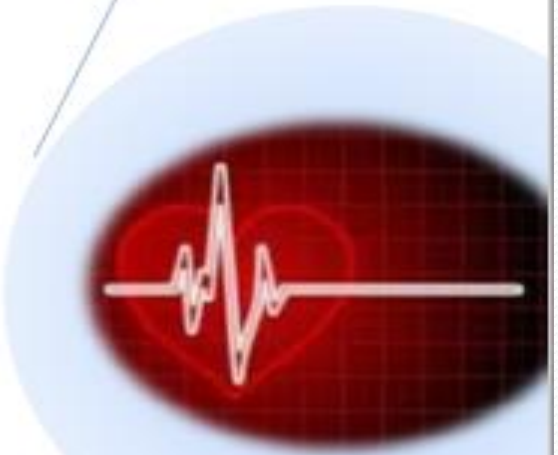
جمهوری اسلامی ایران
وزارت بهداشت، درمان و آموزش پزشکی

شناسنامه استاندارد خدمات

مدیریت درمان سکته حاد قلبی

تهیه شده در کارگروه تخصصی کمیته علمی مدیریت درمان سکته حاد قلبی

معاونت درمان
دفتر مدیریت بیمارستانی وتعالی خدمات بالینی
مرکز مدیریت حوادث و فوریت های پزشکی
ویرایش دوم- مهر ۱۳۹۴



در دهه گذشته بیماری های قلبی عروقی به عنوان مهم ترین علت مرگ ومیر در دنیا مطرح و به صورت اپیدمی جهانی شناخته شده است.

بیماری های قلبی عروقی علت 30 % از مرگ ها و 11 % از موارد DALYsگزارش شده است.

در این راستا بیماری عروق کرونر به عنوان شایع ترین بیماری قلبی در بزرگسالان اهمیت ویژه ای دارد.

بیماری های عروق کرونر براساس پاتولوژی زمینه ای به انواع زیر تقسیم می شوند:

1- بیماری مزمن قلبی عروقی

2- سندرم های حاد کرونری:

STEMI

NSEMI

UA

3- مرگ ناگهانی

سکته قلبی که با بالا رفتن قطعه STدر نوار قلبی همراه باشند، اصطلاحاً STEMIنامیده می شود؛ در اثر انسداد کامل یک رگ اصلی کرونر به وسیله لخته خونی رخ می دهد. درسایراشکال تنگی عروق کرونرناکامل است

این فرم وخیم ترین تظاهر سندرم حاد کرونری و تهدید کننده حیات می باشد.

طبق آخرین آمارهای منتشر شده جهانی تقریباً 40 - 25 % موارد از سکته حاد قلبی همراه با بالا رفتن قطعه STهستند.

چون در STEMI تنگی رگ کامل وجریان خون آن بخش از میوکارد قطع است درمان استاندارد بدون شک شامل باز کردن رگ وبرقراری مجدد جریان خون در عروق بسته شده **(ری پرفیوژن)** به صورت فوری می باشد.

هدف از این درمان جلوگیری از نکروز میوکارد و نجات میوکارد در معرض خطر، کاهش بروزنارسایی قلبی و نهایتاً افزایش طول عمر بیمار می باشد.

با توجه به این که عملکرد سلول های قلبی درصورت انسداد پایداررگ درگیر کاهش می یابد، شروع سریع درمان با رعایت استانداردهای درمانی و برقراری مجدد جریان خون در منطقه انفارکت الزامی است

مهمترین چیز در حفظ میوکارد و پیشگیری ازمرگ و میر وعوارض مهم سکته قلبی مانند نارسایی قلبی **زمان شروع درمان** است که هرچه زودتر جریان خون مجددا برقرار شود سلول عضله کمتری خواهد مرد وبخش بیشتری از عملکرد انقباضی قلب جفظ خواهد شد.

از نظر کیفیت زندگی EFمعادل 45% و30% بسیار متفاوتند

به همین دلیل تشخیص وشروع درمان سکته های قلبی بویژه از نوع STEMI باید با سرعت انجام شود.

ری پرفیوژن با دو روش **فیبرینولیز** و **آنژیوپلاستی** اولیه انجام می شود.

در هر یک ازروش های ری پرفیوژن هر چقدر زمان ایسکمی طولانی تر شود؛ میزان از دست رفتن عضله قلبی واحتمال مرگ بالاتر می رود و جمله مصداق عینی پیدا می کند:

زمان عضله است

TIME IS MUSCLE

در سال های اخیر درمان آن از روش **فارماکولوژیک** به روش عمدتاً **مکانیکال (آنژیوپلاستی)** تغییر یافته که این موضوع به همراه پیشرفت های ایجاد شده در درمان دارویی باعث **کاهش** قابل توجه در میزان مرگ ومیر آن شده است.

چرا؟

1- در صورت وجود امکانات لازم جهت انجام آنژیوپلاستی اولیه تقریباً هیچ اندیکاسیونی برای درمان ترومبولیتیک وجود ندارد.

2- فیبرینولیزکنترااندیکاسیون هایی دارد که در صورت وجود، می تواند باعث افزایش خطر خونریزی شود؛ این عارضه با وجود نادر بودن(حدود 1 %) مهم می باشد

3- احتمال رسیدن به TIMI Flow grade III درروش آنژیوپلاستی اولیه به طور معنی داری از روش ترومبولیز بیشتر است.

4- اگرچه در نگاه اجمالی به نظر می رسد آنژیوپلاستی اولیه هزینه بالاتری نسبت به ترومبولیتیک تراپی دارد ولی با درنظر گرفتن موارد زیر این روش کاملاً هزینه اثر بخش می باشد:

- تعداد بیمارانی که بعد از گرفتن ترومبولیتیک نیاز به اینترونشن پیدا می کنند

- طول مدت بستری بیشتر در درمان با ترومبولیتیک ها

- وقوع بیشتر نارسایی قلبی

پس درمان انتخابی برای سکته های حاد قلبی **ری پرفیوزن با تعبیه استنت** است(PCI: Percutaneous coronary intervention)

لکن مهم این است که بایدهرچه سریعتر انجام شود تا سکته قلبی بیمار کامل نگردد. به این روش(PPCI: Primary PCI) می گویند که در حال حاضربهترین روش است.

در این روش بیمار باعلائم سکته قلبی در اورژانس پذیرش شده ومستقیما به کتلب می رود وتحت PPCIقرار می گیرد

شروع پروسه PPCI در بیمارستان دارای شرایط وبافعال کردن تیم کد247 صورت می گیرد

بدیهی است هرچه تیم اورژانس وکد247 سریع تر وحرفه ای تر عمل کنند نتایج مطلوب تری حاصل خواهد شد

به همین منظور باید اورژانس های بیمارستان ها دارای تجهیزات لازم بوده، وبعلاوه بهتر است **مستقیما** به کتلب راه داشته باشند

بیماردربخش اورژانس سریعا اکوشده، توسط پرستار IV بیمار که توسط 115 گرفته شده بود مطمئن شده یا حتی دورگ گرفته می شود ویا حتی بسته به شرایط ورید مرکزی گرفته می شود، نمونه ها ارسال می گردد وسپس به کتلب فرستاده می شود.

شرایط انتقال: پس از اطمینان از حضورتیم اینترونشن درکتلب وروشن شدن دستگاه آنژیوگرافی، تحت مانیتورینگ، همراه دوپرستاریا یک پرستار و پزشک یااینترن، الکتروشوک آماده واز کمترین فاصله ممکن

نکته مهم:

بزرگترین خطا واردکردن بیمار به بخش آنژیوگرافی ورها کردنش توسط پرسنل اورژانس است. تا بیمار روی تخت آنژیو نخوابیده نباید رها شود.

بخش مهم دیگری که در این پروسه موثرند پرسنل فوریت ها هستند:

انجام صحیح اقدامات اولیه شامل رگ گیری سریع ومناسب، دادن داروهای اولیه، شروع CBRوCM از منزل، شیوه صحیح حمل منجمله دمای مناسب داخل امبولانس، تسلط بر استفاده صحیح وبموقع از داروهای انتی اریتمی، شیوه صحیح وسرعت در تحویل بیمار به اورژانس بیمارستان، همگی از اهمیت ویژه ای برخوردارند.

بیمارپس از انجام موفق پروسه وگذراندن دقایق تا ساعاتی در بخش یا قسمت پست کت(Post cath) به سی سی یو منتقل ودر انجا بستری خواهد شد.

يك نكته مهم ديگر در نتيجه گيری در اين پروسه تجهيزامبولانس پيش بيمارستانی به امكانات *دوراپزشكی* است.

اين تجهيزات امكان تشخيص سريع ودر صورت نياز مداخلات صحيح تحت امر پزشک متخصص قلب مستقر در مركزرا فراهم می كند

ناگفته پيداست نقش خود بيمار در بيان دقيق وزود هنگام علائم، پرهيزاز درمان های محلی وسنتی، بويژه داروهای مرسوم پنهان كننده علائم يعنی مواد مخدرواظهار نظرهای غير عالمانه اما مصرانه اطرافيان بخصوص وقتی بيمار خانم است دربموقع بودن اقدامات بسيار مهم هستند

سلسله اقدامات ضروری برای درمان مناسب سکته حاد قلبی، باید به صورت زنجیره ای و مرتبط با هم تعریف شوند و بطور خلاصه به شرح زیر می باشند:

- آموزش دقیق ودرست به جامعه برای درک علایم سکته قلبی و تماس زود هنگام با سیستم درمانی

- هماهنگی سیستم اورژانس برای اقدامات درمانی به موقع قبل از بیمارستان

- انتقال بیمار مطابق با استانداردها به مراکز مجهز

- انجام اقدامات درمانی ری پرفیوژن با رعایت استانداردهای زمانی توسط یک تیم مجرب در بیمارستان

زمان های مهم در ارائه خدمت آنژیوپلاستی اولیه:

- **First Medical Contact Time:** زمان اولین ویزیت بیمار توسط پزشک یا تیم پزشکی
- **First ECG Time:** زمان اخذ اولین نوار قلب پس از ورود به بیمارستان
- **STEMI ECG Time:** زمان اخذ اولین نوار قلبی که تشخیص STEMIرا تایید می کند .
- **STEMI Verification Time:** زمان تشخیص STEMIتوسط پزشک مستقر در اورژانس
- **Door To Device Time:** فاصله زمانی بین ورود بیمار دچار STEMIبه یک بیمارستان با قابلیت ارائه خدمت آنژیوپلاستی اولیه (Door Time)تا عبور وایر از ضایعه کرونری مسوول سکته قلبی(DeviceTime)

جدول شماره ٦- مدت زمان استاندارد هر واحد خدمت آنژیوپلاستی				
ردیف	نوع خدمت	ارائه دهنده خدمت	مدت زمان مشارکت در فرایند ارائه خدمت	نوع مشارکت در قبل، حین و بعد از ارائه خدمت
۱	ویزیت مقدماتی واخذ شرح حال وبررسی ECG و فعال کردن کد ۲۴۷	پزشک مستقر در اورژانس با تصویب کمیته درمان سکته قلبی بیمارستان[۱۶]	۱۰دقیقه	حین ارائه خدمت
۲	آماده کردن بیمار و انتقال به کت لب	پرستاراورژانس	۱۵دقیقه	حین ارائه خدمت
۳	انجام آنژیوپلاستی اولیه و تکمیل فرم ثبت	اینترونشنال کاردیولوژیست	۶۰-۳۰ دقیقه	حین ارائه خدمت
۳	ترومبولیتیک تراپی در صورت نبود شرایط آنژیوپلاستی اولیه و تکمیل فرم ثبت با ذکر دلایل فیبرینولیتیک تراپی	کاردیولوژیست	باید ظرف ۳۰ دقیقه از زمان Door Time شروع شود و بسته به نوع دارو می تواند تا ۹۰ دقیقه هم به طول انجامد	حین ارائه خدمت
۴	اطمینان از تکمیل فرم ثبت STEMI و دریافت اطلاعات ترومبولیتیک تراپی	مسوول بخش کت لب با هماهنگی پرستار کت لب	۳۰ دقیقه روزانه	بعد از خدمت
۵	ثبت خدمات در سامانه مدیریت درمان STEMI	منشی بخش کت لب	۱۰ دقیقه	بعد از خدمت
۶	انتقال بیمار به سی سی یو	سوپروایزر	۱۵ دقیقه	بعد از خدمت
۷	مراقبت در سی سی یو و انتقال به بخش	کاردیولوژیست	۵-۳ روز	بعد از خدمت
۸	آموزش مراقبت های پس از ترخیص	پرستار	۱۵ دقیقه	بعد از خدمت

بیمارستان 247 :

بیمارستان با قابلیت ارائه خدمت آنژیوپلاستی اولیه که متعهد می شود خدمات را به صورت تمام وقت24 ساعته و 7 روز در هفته ارائه نماید.

جدول شماره ۳- عنوان و مشخصات سایر اعضای تیم ارائه خدمت درمان سکته حاد قلبی				
ردیف	عنوان تخصص	میزان تحصیلات موردنیاز	سابقه کار یا دوره آموزشی مصوب درصورت لزوم	نقش در فرآیند ارائه خدمت
۱	متخصص قلب و عروق متخصص طب اورژانس متخصص داخلی	دکترای تخصصی پزشکی	شرکت در دوره بازآموزی مصوب کمیته علمی هر ۲ سال یک بار	در بیمارستان دارای امکانات آنژیوپلاستی اولیه: تشخیص STEMI و فعال کردن کد ۲۴۷ در سایر بیمارستان ها: تجویز ترومبولیتیک برای STEMI
۲	دستیار قلب	دکترای پزشکی		
۳	پرستار آموزش دیده	کارشناسی	سابقه دو سال کار در سی سی یو، کت لب یا آی سی یو شرکت در دوره بازآموزی مصوب کمیته علمی هر ۲ سال یک بار	اطمینان از کارکرد مناسب تجهیزات تهیه و تدارک ملزومات مصرفی آماده کردن بیمار: تعبیه IV line و اخذ آزمایشات لازم مراقبت های پرستاری بیمار در حین خدمت هماهنگی با سوپروایزر برای تامین تخت سی سی یو برای انتقال بیمار پس از خدمت

جدول شماره ۳- عنوان و مشخصات سایر اعضای تیم ارائه خدمت درمان سکته حاد قلبی				
ردیف	عنوان تخصص	میزان تحصیلات موردنیاز	سابقه کار یا دوره آموزشی مصوب درصورت لزوم	نقش در فرآیند ارائه خدمت
				ثبت اقدامات و نظارت بر عملکرد تیم غیر پزشکی
٤	تکنسین	فوق دیپلم	۱.آشنایی با عملکرد دستگاه ۲.سابقه دو سال کار در کت لب شرکت در دوره بازآموزی مصوب کمیته علمی هر ۲ سال یک بار	کمک به تصویربرداری مناسب در حین آنژیوپلاستی اولیه
٥	منشی بخش کت لب	مطابق شرایط احراز تشکیلات بیمارستانی	شرکت در دوره آموزشی سیستم ثبت اطلاعات خدمت	ثبت اقدامات انجام شده براساس فرم تکمیل شده توسط پزشک
٦	مدیر بیمارستان	مطابق شرایط احراز تشکیلات بیمارستانی	شرکت دردوره آموزشی مدیریت درمان سکته حاد قلبی مصوب کمیته علمی هر ۲ سال یک بار	مشارکت در تدوین استانداردهای داخل بیمارستانی و نظارت بر اجرا
۷	مسوول آزمایشگاه	مطابق شرایط احراز تشکیلات بیمارستانی	آشنایی با فرآیند مصوب کمیته بیمارستانی	انجام آزمایش های ضروری در ارائه خدمت با دقت و سرعت مناسب
۸	کارپرداز	مطابق شرایط احراز تشکیلات بیمارستانی	آشنایی با فرآیند ارائه خدمت	تهیه و تدارک ملزومات مورد نیاز
۹	پرسنل تجهیزات پزشکی	مطابق شرایط احراز تشکیلات بیمارستانی	آشنایی با فرآیند ارائه خدمت	تهیه و تدارک و پشتیبانی تجهیزات مورد نیاز
۱۰	انتظامات بیمارستان	مطابق شرایط احراز تشکیلات بیمارستانی	آشنایی با فرآیند ارائه خدمت	راهنمایی مراجعین و تسریع در ارائه خدمت
۱۱	بیماربر	مطابق شرایط احراز تشکیلات بیمارستانی	آشنایی با فرآیند ارائه خدمت	انتقال بیمار از اورژانس به کت لب و سی سی یو
۱۲	نظافتچی	مطابق شرایط احراز تشکیلات بیمارستانی	آشنایی با فرآیند ارائه خدمت و اصول استریلیتی	حفظ نظافت و استریلیتی در محیط کت لب

کمیته کد 247

در هر بیمارستان مجری برنامه 24 ساعته و 7 روز در هفته مدیریت درمان سکته حاد قلبی، باید کمیته ای تحت عنوان سکته های قلبی تشکیل شود.

ریاست این کمیته به عهده رییس بیمارستان بوده، دبیرآن که یک اینترونشنال کاردیولوژیست است، توسط رییس بیمارستان انتخاب می گردد.

وظایف این کمیته به شرح زیر است:

- تنظیم برنامه عملیاتی دستیابی به الزامات برنامه مطابق چک لیست با هماهنگی معاون درمان دانشگاه
- امضای تفاهم نامه با معاونت درمان دانشگاه به منظور تعهد به حسن اجرای برنامه در بیمارستان
- تامین تجهیزات ونیروی انسانی آموزش دیده متناسب اجرای برنامه با هماهنگی معاون درمان دانشگاه
- تهیه ابلاغ مسوولیت افراد مسوول اجرای برنامه در بیمارستان
- ابلاغ شرح وظایف پرسنل درگیر در آنژیوپلاستی اولیه مطابق با الزامات بخش مشخصات فنی این دستورالعمل

- هماهنگی با اورژانس محلی و برگزاری دوره های آموزشی برای تکنسین های اورژانس 115
- تدوین فرآیند اعلام کد 247 در بیمارستان و اطمینان از آشنایی کلیه پرسنل با نحوه اجرای آن
- اطمینان از آموزش پرسنل درگیر در آنژیوپلاستی اولیه به ویژه دوره احیای قلبی پیشرفته و اصول کار با دستگاه بالن پمپ
- طراحی فرآیند کنترل کیفی روتین دستگاه ها و انجام کالیبراسیون های مورد نیاز
- نظارت دوره ای بر کنترل کیفی دستگاه ها و مرور داده های مربوط به کنترل کیفی روتین و یا انجام کنترل های کیفی خاص به صورت دوره ای و تطبیق با استانداردهای تکنیکی
- تامین زیرساخت پشتیبانی سامانه ثبت، نظارت بر ثبت و گزارش دهی و ارزیابی شاخص های مدیریتی برنامه
- تدوین و اجرای فرآیند رضایت سنجی مراجعین و ارزیابی گزارش های دوره ای آن
- طراحی و اجرای فرایند آموزش بیمار و همراهان وی درخصوص مراقبت های پس از خدمت و پیگیری درمان

حقوق اختصاصی بیماران مرتبط با خدمت دریافتی (با تاکید بر عوارض جانبی مرتبط با خدمت دریافتی):

۱. محرمانه بودن اطلاعات پزشکی اخذ شده از بیمار
۲. توضیح مراحل انجام کار
۳. بررسی دقیق اندیکاسیون و کنتراندیکاسیون ها
۴. پاسخ به پرسش های احتمالی بیمار وهمراهان بیمار
۵. ارائه تصاویر خدمت و گزارش کتبی خدمت
۶. ارائه توصیه های لازم در ارتباط با خدمت پس از ترخیص
۷. مراقبت از مدارک بیمار

پایش و ارزشیابی

الف- معیار های ارزیابی بخش پیش بیمارستانی

۱. آیا زمان تماس بیمار با شکایت درد حاد قفسه سینه، با سیستم اورژانس (first medical contact) توسط تکنسین اورژانس پیش بیمارستانی ثبت می شود؟

۲. آیا بیمار با درد حاد قفسه سینه به نزدیکترین بیمارستان معین منتقل می شود؟

۳. آیا دستگاه دفیبریلاتور (defibrillator) در آمبولانس موجود است؟

۴. آیا امکان تشخیص STEMI در آمبولانس وجود دارد؟

۵. آیا سیستم انتقال داده های بیمار به سیستم دیسپچ مرکزی وجود دارد؟

۶. آیا دارو درمانی اولیه در آمبولانس انجام می شود؟

۷. آیا شاخص های زیر هر ماه ثبت و گزارش دهی می شود: تعداد موارد بیمار با درد حاد قفسه سینه، تعداد موارد اثبات شده سکته حاد قلبی یا سندرم حاد کرونری از بین مراجعین با علایم درد قفسه سینه، میزان مرگ و میر پیش بیمارستانی بیمار با درد حاد قفسه سینه، موارد نیاز به احیا در بیمار درد حاد قفسه سینه و موفقیت/ عدم موفقیت احیا

ب- معیار های ارزیابی بخش اورژانس

۱. آیا اورژانس بیمارستان ۲۴۷ به بیمار با درد حاد قفسه سینه (ارجاعی توسط سیستم اورژانس یا مراجعه شخصی) پذیرش می دهد؟

۲. آیا زمان ورود بیمار به بیمارستان توسط پرستار تریاژ ثبت می شود؟

۳. آیا زمان شروع درد بیمار تا رسیدن بیمار به بیمارستان توسط پرستار تریاژ/اورژانس پرسش و ثبت می شود؟

۴. آیا فلوچارت فرآیند پذیرش و انتقال بیمار STEMI به بخش کت لب/ مراقبت های ویژه قلبی در اورژانس موجود و در محلی مناسب و قابل رویت بر روی تابلوی اعلانات نصب شده است؟

۵. آیا کد STEMI/247 در بیمارستان موجود است؟

۶. آیا سیستم فعال کردن کد سکته قلبی برای بیمار STEMI مطابق استانداردهای تشخیص و درمان انجام می شود؟

۷. بخش اورژانس در تمام اوقات شبانه روز و در تمامی روزهای هفته (۲۴ ساعته و ۷ روز درهفته) دسترسی مناسب به متخصص قلب اینترونشنیست دارد؟

۸. آیا اقدامات دارویی اولیه برای بیمار سکته حاد قلبی به موقع اجرا و در پرونده ثبت می شود؟

۹. در صورت اثبات STEMI آیا در حداقل زمان ممکن *کد سکته قلبی/کد* ۲۴۷ فعال می شود و *زمان اعلام کد۲۴۷* توسط پرستار اورژانس ثبت می‌شود؟

۱۰. آیا *انتقال بیمار به کت لب* در حداقل زمان ممکن انجام می شود؟

۱۱. آیا در بخش اورژانس یک کتابچه/ مجموعه توجیهی برای آشنایی پرسنل مربوطه در مورد چگونگی برخورد با بیمار سکته حاد قلبی موجود است؟

۱۲. آیا به بیماران بستری و همراهانشان توضیحات مناسب و قابل درک در مورد بیماری، نوع مراقبت درنظرگرفته شده، روش های جایگزین، پیامدهای احتمالی ناشی از درمان ارائه می شود؟

۱۳. آیا شاخص های زیر هر ماه در اورژانس ثبت و توسط مسئول اورژانس به کمیته بهبود کیفیت و کمیته مرگ و میر بیمارستان گزارش می شود؟

- میزان مرگ و میر داخل بیمارستانی (اورژانس) در بیمارSTEMI
- موارد اعلام کد احیاء برای بیماران STEMI
- موفقیت/ عدم موفقیت احیاء

ج- معیار های ارزیابی بخش آنژیوپلاستی (کت لب)

۱. آیا زمان رسیدن بیمار به کت لب توسط پرستار کت لب ثبت می شود؟

۲. با در نظر گرفتن و محاسبه زمان های ثبت شده در پرونده بیمار آیا زمان Door-To-Device - Time توسط پرستار کت لب برای بیمار STEMI محاسبه و ثبت می شود؟

۳. آیا خدمات پشتیبانی بخش کت لب (آزمایشگاه، خدمات دارویی و ...) به صورت شبانه روزی در دسترس می باشد؟

۴. آیا امکانات و تجهیزات مناسب برای دستیابی به اهداف مراقبتی بیماران در بخش کت لب وجود دارد؟

۵. آیا یک سیستم در بخش کت لب برای بازبینی و ارائه گزارش تصاویر توسط یک تصویربردار همراه با گزارش بالینی حداکثر ظرف ۲۴ ساعت وجود دارد؟

۶. آیا طبق مستندات پرستار مسئول کت لب از آماده، کامل و به روز بودن داروها و امکانات مورد نیاز آنژیوپلاستی، اطمینان حاصل می نماید؟

۷. آیا استانداردهای فضای فیزیکی بخش کاتتریزاسیون مطابق جداول موجود در شناسنامه تدوین استاندارد رعایت شده است؟

۸. آیا اینترونشنیست در زمان مناسب (با رعایت زمان استاندارد Door-To-Device - Time کمتر از ۹۰ دقیقه) در کت لب حاضر می شود؟

د- معیار های ارزیابی بخش مراقبت های ویژه قلبی (CCU)

۱. آیا بخش مراقبت های ویژه قلبی به بیماران سکته قلبی ارجاعی از کت لب پذیرش به موقع می دهد؟

۲. بخش مراقبت های ویژه قلبی در تمام اوقات شبانه روز و در تمامی روزهای هفته (۲۴ ساعته و ۷ روز در هفته) از حضور متخصص قلب مقیم برخوردار است؟

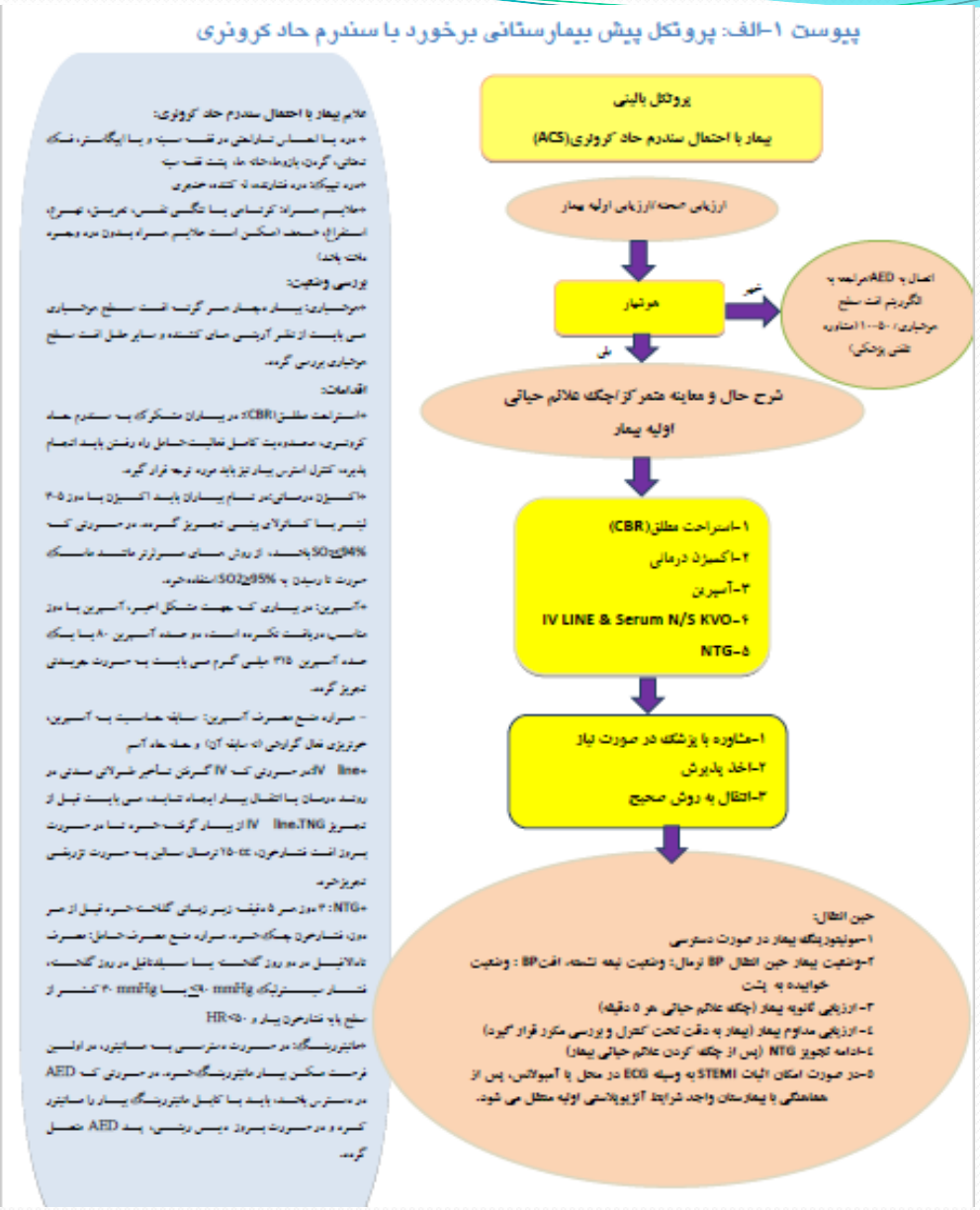
۳. آیا طبق مستندات موجود، اقدامات اصلاحی به منظور رفع نارسایی های شناسایی شده در برنامه آموزشی پرستار/ پرسنل درمانی و یا کمبود/نواقص تجهیزات و نیروی انسانی مرتبط با تشخیص و درمان بیمار سکته حاد قلبی توسط مسئول بخش مراقبت های ویژه قلبی انجام می گیرد؟

۴. آیا شاخص های زیر به صورت هر ماه یکبار برای بیماران STEMI ثبت و به کمیته بهبود کیفیت و کمیته مرگ و میر بیمارستان گزارش می شود؟ (میزان بهبودی و ترخیص از بیمارستان - میزان مرگ و میر بیمار - عوارض بیماری/ عوارض جانبی درمان)

۵. آیا در زمان ترخیص بیمار از بخش مراقبت های ویژه قلبی، ارزیابی خطر بیمار از نظر عوارض بیماری (وجود و درجه نارسایی قلبی، میزان عملکرد عضله قلب) انجام می شود؟

۶. آیا در زمان ترخیص بیمار از بخش مراقبت های ویژه قلبی، ، زمان پیگیری بعدی تعیین و توضیحات مربوطه به بیمار داده می شود؟

پیوست ۱-الف: پروتکل پیش بیمارستانی برخورد با سندرم حاد کرونری



پیوست ۱-ب: الگوریتم تریاژ تلفنی در مورد بیماران قلبی

سطوح اولویت اعزام آمبولانس	
قرمز	✓ افت هوشیاری/ عدم پاسخ ✓ شواهد تنفس ناکافی یا غیر موثر مانند سیانوز
زرد	✓ هوشیاری ناکامل یا بی قراری شدید ✓ نشانه های دیسترس حاد تنفسی شامل: صدادار شدن تنفس، تقلای تنفسی، بی قراری شدید، ناتوانی در تکلم، ناتوانی در بلع بزاق ، همراه با تنگی نفس ✓ غش، سیاهی رفتن چشم ها یا احساس سبکی سر و یا تعریق شدید همراه با تهوع/ استفراغ ✓ آنژین ناپایدار شامل: شروع درد در دو ماهه اخیر، درد در حالت استراحت، تغییر الگوی درد (افزایش مدت یا شدت درد، شروع درد با فعالیت کمتر نسبت به روزهای گذشته، عدم پاسخ به داروی موثر قبلی) ✓ مصرف داروها یا مواد محرک مانند اکستازی، شیشه و کوکائین ✓ شک قوی به ACS: علائم تیپیک سکته قلبی: درد قفسه سینه که می تواند به فک تحتانی،گردن، شانه یا بازو کشیده شود، درد فعالیتی (افزایش درد با فعالیت، استرس یا سرما) که ممکن است همراه با تعریق سرد، تهوع ، استفراغ یا تنگی نفس باشد. ✓ آنژین اکو والان: علائم غیر تیپیک(مانند: دیافورز، تهوع، تعریق سرد، تنگی نفس، سرگیجه و ...) در بیماران با بیماری زمینه ای مانند دیابت در صورت قضاوت بالینی پرستار تریاژ تلفنی
سبز	✓ سن بالای ۳۵ سال ✓ آنژین پایدار:درد تیپیک قلبی در مورد شناخته شده بیماری کرونر که هیچ کدام از معیارهای آنژین ناپایدار را ندارد و در حال حاضر فاقد علامت است.
سفید	✓ درد غیر تیپیک قلبی در بیمار زیر ۳۵ سال که در حال حاضر کاملاً رفع شده و هیچ یک از شواهد آنژین ناپایدار را ندارد و بیمار ریسک فاکتورهای دیابت، سابقه خانوادگی مثبت و بیماری قلبی را ندارد.
تعاریف رنگ ها	
قرمز	اعزام آمبولانس با اولویت بسیار بالا به همراه موتورآمبولانس پیشرو
زرد	اعزام آمبولانس با اولویت بالا، در صورت وجود ترافیک شهری اعزام موتورآمبولانس پیشرو
سبز	اعزام موتورآمبولانس در صورت فقدان موتورآمبولانس اعزام آمبولانس زمینی با اولویت کمتر
سفید	توصیه اکید مراجعه سرپایی در صورت درد مجدد یا تغییر وضعیت بیماری یا الگوی درد، مجدداً تماس بگیرد.

توصیه های قبل از رسیدن EMS
بیمار را در هر وضعیتی که راحت تر است قرار دهید،
کلیه لباسهای تنگ سر و گردن بیمار را آزاد کنید
اجازه هیچگونه فعالیت اضافه ای را به بیمار ندهید و محیط را برای او آرام کنید،
اجازه خوردن و آشامیدن را به بیمار ندهید،
در صورت امکان داروهای مصرفی بیمار را در کنار وی قرار دهید،
در صورت بروز مشکل جدید با من تماس بگیرید،