Università degli Studi di Ferrara

Anno Accademico 2018-2019
C.L. Infermieristica – C.L. Ostetricia

Corso di

Semeiotica Medica e Medicina Interna

Medicina Interna

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Università degli Studi di Ferrara

Malattie dell'apparato cardiovascolare

-Ipertensione arteriosa

-Patologia cardiovascolare aterosclerotica (cardiopatia ischemica, angina e infarto, sindrome Takotsubo)

-Scompenso cardiaco

Pressione arteriosa

Forma di energia potenziale che deriva dalla contrazione del cuore e serve per consentire al sangue di scorrere contro la resistenza offerta dalle arteriole periferiche.

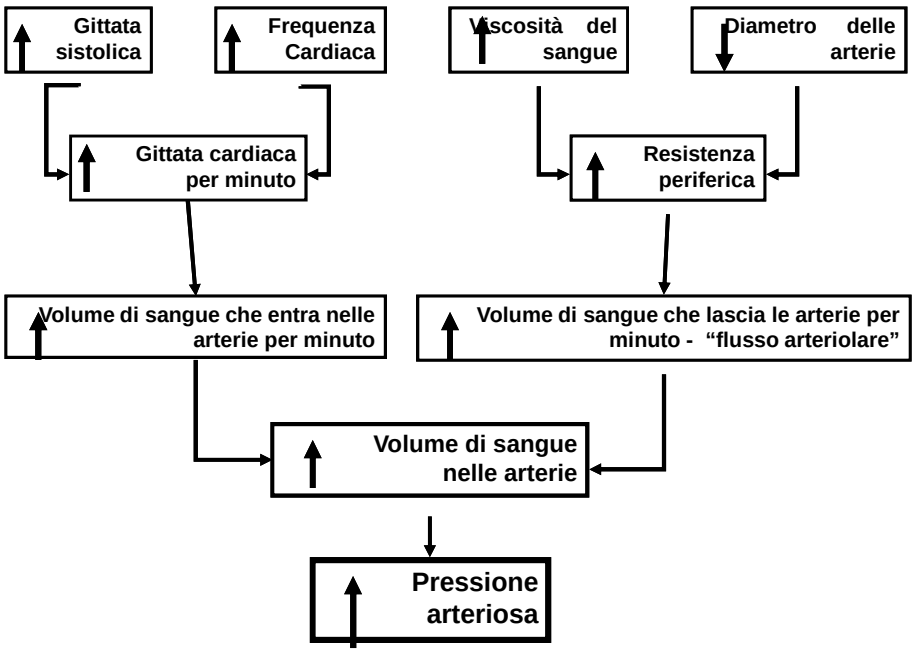
Viene espressa in mmHg: altezza raggiunta da colonna di mercurio, se si applica a questa la forza del sistema arterioso.

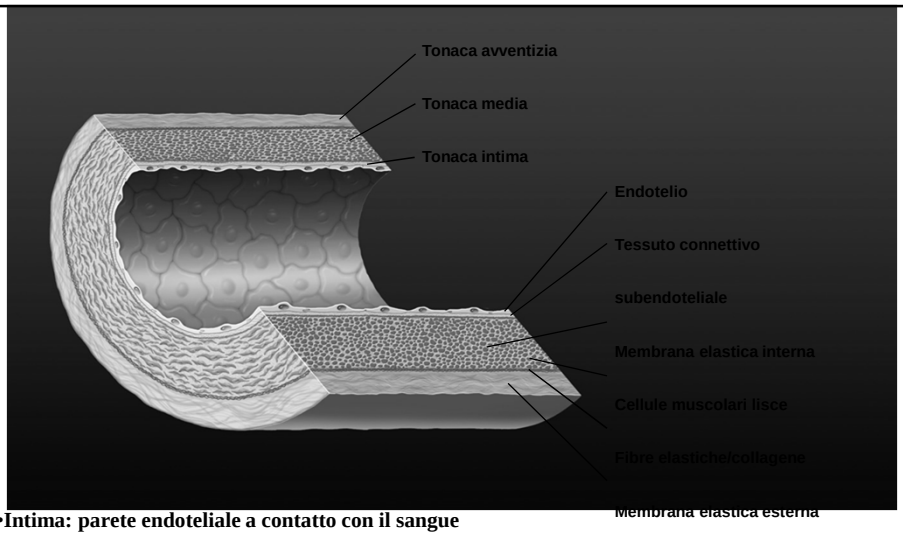
Da cosa è determinata?

$$P = V \times R$$

V= volume minuto cardiaco= vol sistolico x freq. cardiaca

R= resistenza periferica totale= lunghezza sistema arterioso x viscosità plasmatica/raggio del vaso^4





- **Intima: parete endoteliale a contatto con il sangue**
- **Media: parte centrale costituita da tessuto muscolare**
- **Avventizia: tessuto fibroso che ancora il vaso al connettivo circostante**

Il sangue espulso dal ventricolo sx colpisce e dilata le pareti dell'aorta che acquisisce energia potenziale. Tale energia viene liberata dal ritorno elastico che genera un flusso anterogrado

Pressione arteriosa

Pressione arteriosa sistolica (PAS): picco della pressione intravascolare raggiunto durante la sistole ventricolare per l'eiezione del sangue ventricolare

Pressione arteriosa diastolica (PAD): pressione intravascolare raggiunta alla fine della diastole ventricolare = resistenza vascolare periferica

Pressione differenziale: differenza tra le due

VARIAZIONI DELLA PRESSIONE ARTERIOSA LEGATE ALL'OSSERVATORE E AL SOGGETTO

Somma delle variazioni

Soggetto

- **Effetti ambientali**
- **Recente attività fisica**
- **Recente fumo di sigaretta**
- **Temperatura ambientale**
- **Abitudine alla misurazione**

Osservatore

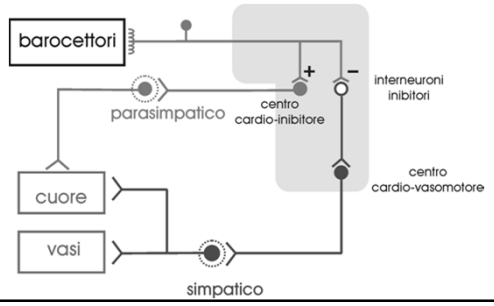
- **Validità dello strumento e stato di manutenzione**
- **Dimensioni del manicotto**
- **Tecnica di misurazione**
- **Criteri di valutazione**

Sistema nervoso autonomo

Regola l'omeostasi pressoria attraverso la modulazione:

1) **simpatica (recettori β renali), della diuresi e del volume circolante, sia direttamente che mediante il SRAA;**
2) **adrenergica (recettori α) del tono vasocostrittore periferico**
3) **parasimpatica e simpatica (recettori muscarinici sensibili all'acetilcolina (Ach) e recettori β sensibili alla noradrenalina (NA) della frequenza, della gittata cardiaca, dell'inotropismo cardiaco.**

   Il tono simpatico dipende da influenze nervose centrali, dalla regolazione riflessa barocettiva e chemocettiva e dalla secrezione periferica di NA

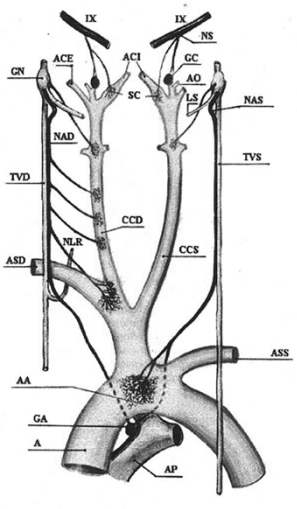


Sistema nervoso autonomo

I barocettori sono terminazioni sensitive localizzate nei seni carotidei e nell'arco aortico.

Captano variazioni pressorie e inviano stimoli ai centri nervosi cerebrali.

Se PA ↓ → ↑ scarica vagale sul nodo sinusale → ↑ FC e ↑ attività nervi simpatici → vasocostrizione periferica e rilascio renina → ↑ pressione arteriosa



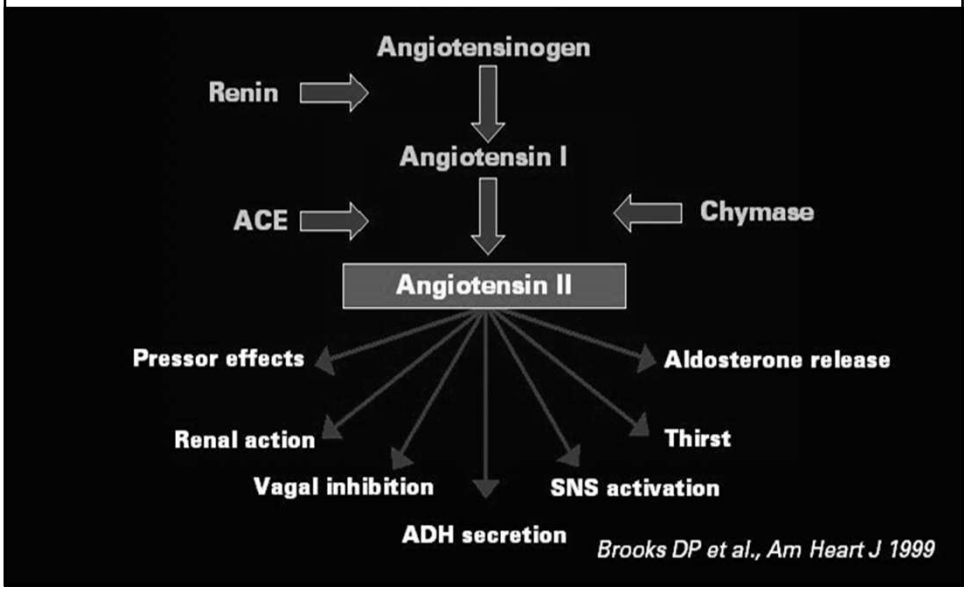
Sistema nervoso autonomo

I chemocettori sono localizzati nell'arco aortico e nei corpi carotidei. Sono attivati dall'ipossia, dall'acidosi e dall'ipocapnia nelle situazioni di ipotensione grave.

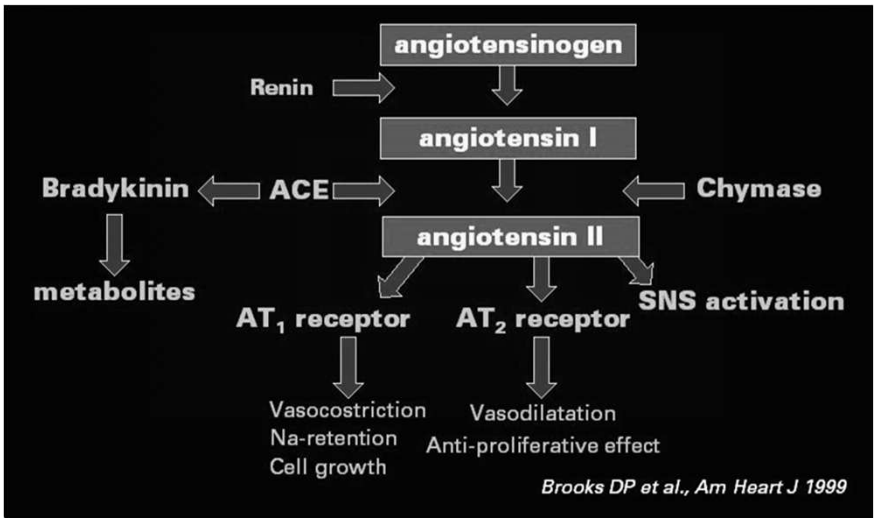
Inducono aumento della ventilazione se si riduce PO_2 nel sangue arterioso

Attivazione → ↑ scarica simpatica → vasocostrizione arteriolare→ ↑ FC → ↑ distensione polmonare

Sistema renina angiotensina aldosterone (SRAA)



Sistema renina angiotensina aldosterone (SRAA)



Endotelio vasale

Le cellule endoteliali secernono mediatori chimici ad azione paracrina che possono favorire o inibire la contrazione delle cellule muscolari liscie

Vasodilatatori: ossido nitrico (NO), prostaglandina I_2 (PGI_2), fattore iperpolarizzante endotelio attivato

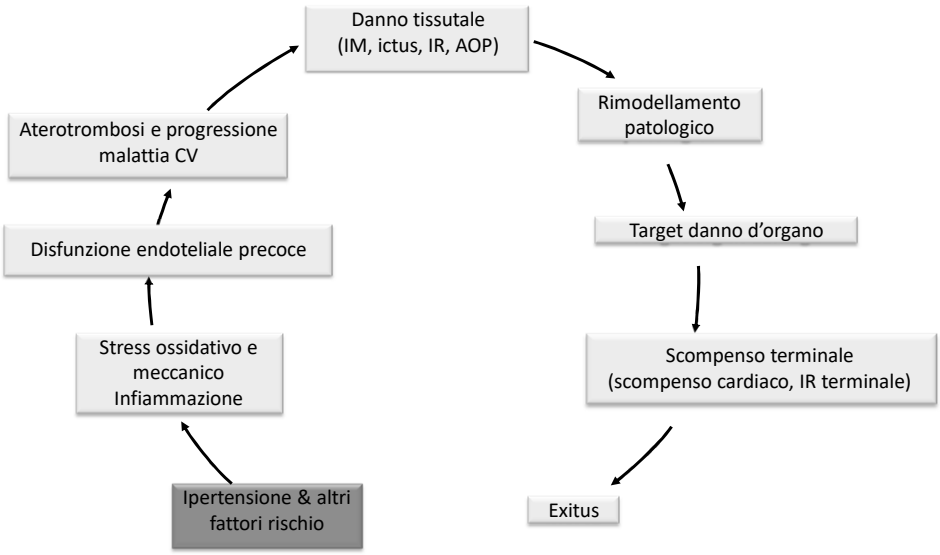
Vasocostrittori: endoteline (endotelina 1) trombossano A_2 (TXA_2), prostaglandina H_2 (PGH_2)

Ormone antidiuretico (ADH)

Prodotto dall'ipotalamo, agisce modificando la permeabilità dell'epitelio del nefrone distale all'acqua e dunque la concentrazione urinaria. La sua liberazione dipende da modificazioni dell'osmolalità e dell'ipovolemia.

↑ Permeabilità all'acqua → passaggio di acqua nell'interstizio → riduzione del volume urinario → ↑ concentrazione intratubulare di NaCl → ↑ riassorbimento di NaCl

Continuum fisiopatologico cardiovascolare e renale




Circulation

American Heart Association.

Heart Disease and Stroke Statistics—2017 Update: A Report From the American Heart Association

Emelia J. Benjamin, Michael J. Blaha, Stephanie E. Chiuve, Mary Cushman, Sandeep R. Das, Rajat Deo, Sarah D. de Ferranti, James Floyd, Myriam Fornage, Cathleen Gillespie, Carmen R. Isasi, Monik C. Jiménez, Lori Chaffin Jordan, Suzanne E. Judd, Daniel Lackland, Judith H. Lichtman, Lynda Lisabeth, Simin Liu, Chris T. Longenecker, Rachel H. Mackey, Kunihiro Matsushita, Dariush Mozaffarian, Michael E. Mussolino, Khurram Nasir, Robert W. Neumar, Latha Palaniappan, Dilip K. Pandey, Ravi R. Thiagarajan, Mathew J. Reeves, Matthew Ritchey, Carlos J. Rodriguez, Gregory A. Roth, Wayne D. Rosamond, Comilla Sasson, Amytis Towfighi, Connie W. Tsao, Melanie B. Turner, Salim S. Virani, Jenifer H. Voeks, Joshua Z. Willey, John T. Wilkins, Jason HY. Wu, Heather M. Alger, Sally S. Wong, Paul Muntner and On behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee

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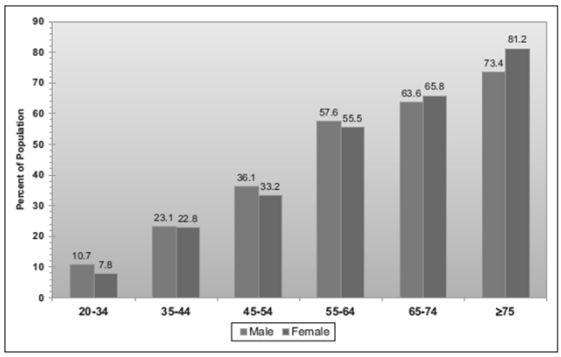
AHA STATISTICAL UPDATE

Heart Disease and Stroke Statistics—2017 Update

A Report From the American Heart Association

AHA Statistical 2016 Update, Circulation 2017

Prevalenza dell'ipertensione in adulti (NHANES 2007-2012)



AHA Statistical 2017 Update, Circulation 2017

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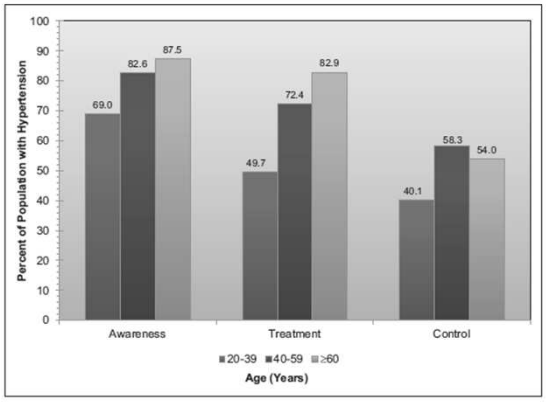
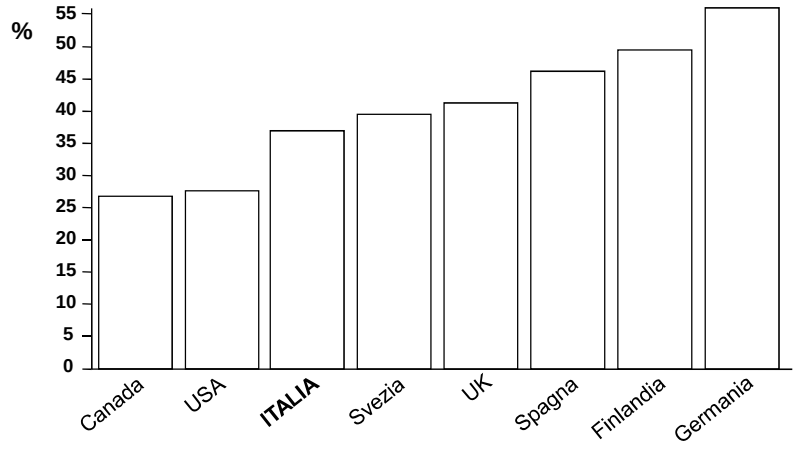


AHA Statistical 2017 Update, Circulation 2017

Prevalenza dell'ipertensione nella popolazione adulta italiana



Wolf-Maier K et al, JAMA 2003

IPERTENSIONE ARTERIOSA

⇩

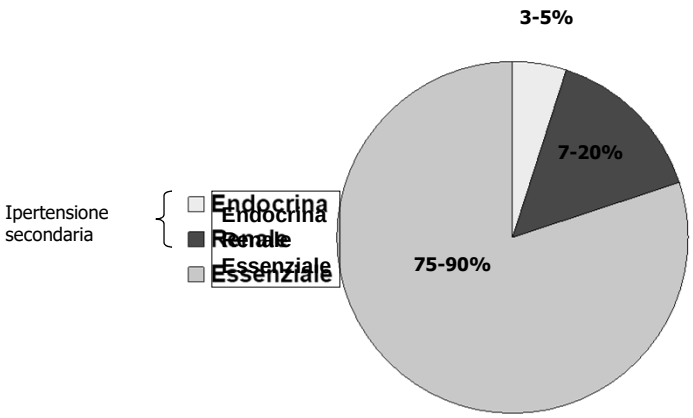
PREVALENZA

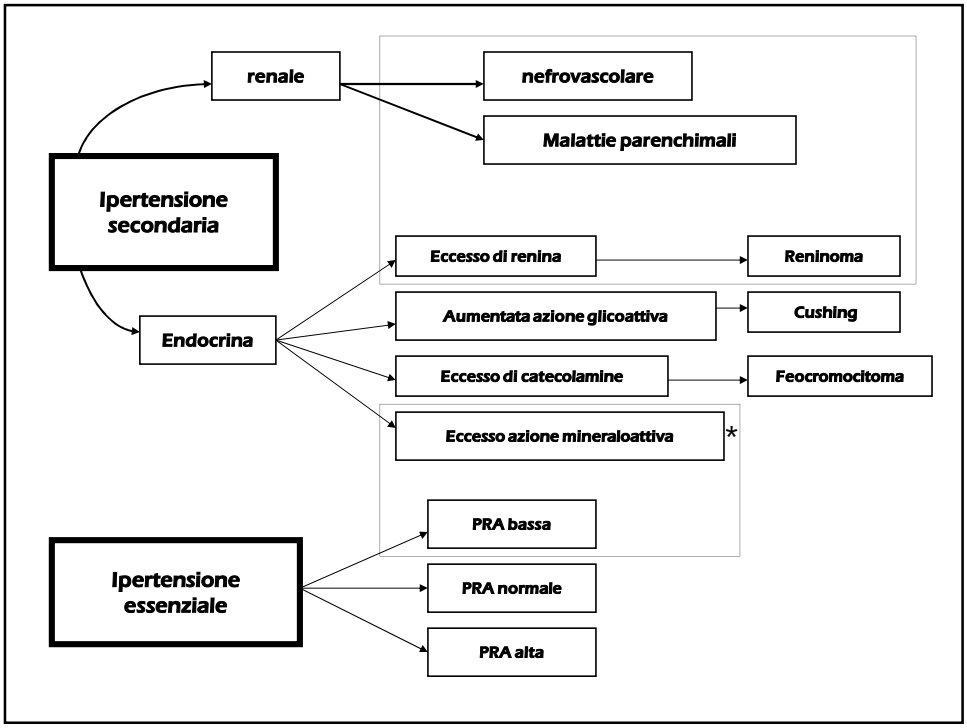
25 % DI TUTTI GLI ADULTI

60 % DEI SOGGETTI DI ETA' SUPERIORE A 60 ANNI

IPERTENSIONE ENDOCRINA ⇨ **> IL 5 % DELLE VARIE FORME IPERTENSIVE**

Cause di ipertensione





ESC European Society of Cardiology
European Heart Journal (2018) 39, 3021–3104
doi:10.1093/eurheartj/ehy339

ESC/ESH GUIDELINES

2018 ESC/ESH Guidelines for the management of arterial hypertension

The Task Force for the management of arterial hypertension of the European Society of Cardiology (ESC) and the European Society of Hypertension (ESH)

Table 1 ESC Classes of recommendations

Classes of recommendations	Definition	Suggested wording to use
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective.	Is recommended/is indicated
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.	
Class IIa	*Weight of evidence/opinion is in favour of usefulness/efficacy.*	Should be considered
Class IIb	*Usefulness/efficacy is less well established by evidence/opinion.*	May be considered
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective; and in some cases may be harmful.	Is not recommended

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Table 2 ESC Levels of evidence

Level of evidence A	Data derived from multiple randomized clinical trials or meta-analyses.
Level of evidence B	Data derived from a single randomized clinical trial or large non-randomized studies.
Level of evidence C	Consensus of opinion of the experts and/or small studies, retrospective studies, registries.

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2.1 What is new and what has changed in the 2018 ESC/ESH Arterial Hypertension Guidelines?

Changes in recommendations	
2013	**2018**
Diagnosis	**Diagnosis**
Office BP is recommended for screening and diagnosis of hypertension.	It is recommended to base the diagnosis of hypertension on: • Repeated office BP measurements; or • Out-of-office BP measurement with ABPM and/or HBPM if logistically and economically feasible.
Treatment thresholds **High-normal BP (130–139/85–89 mmHg):** Unless the necessary evidence is obtained, it is not recommended to initiate antihypertensive drug therapy at high–normal BP.	**Treatment thresholds** **High-normal BP (130–139/85–89 mmHg):** Drug treatment may be considered when CV risk is very high due to established CVD, especially CAD.
Treatment thresholds **Treatment of low-risk grade 1 hypertension:** Initiation of antihypertensive drug treatment should also be considered in grade 1 hypertensive patients at low–moderate-risk, when BP is within this range at several repeated visits or elevated by ambulatory BP criteria, and remains within this range despite a reasonable period of time with lifestyle measures.	**Treatment thresholds** **Treatment of low-risk grade 1 hypertension:** In patients with grade 1 hypertension at low–moderate-risk and without evidence of HMOD, BP-lowering drug treatment is recommended if the patient remains hypertensive after a period of lifestyle intervention.
Treatment thresholds **Older patients** Antihypertensive drug treatment may be considered in the elderly (at least when younger than 80 years) when SBP is in the 140–159 mmHg range, provided that antihypertensive treatment is well tolerated.	**Treatment thresholds** **Older patients** BP-lowering drug treatment and lifestyle intervention is recommended in fit older patients (>65 years but not >80 years) when SBP is in the grade 1 range (140–159 mmHg), provided that treatment is well tolerated.
BP treatment targets	**BP treatment targets**
An SBP goal of <140 mmHg is recommended.	• It is recommended that the first objective of treatment should be to lower BP to <140/90 mmHg *in all patients* and, provided that the treatment is well tolerated, treated BP values should be targeted to 130/80 mmHg or lower in most patients. • In patients <65 years it is recommended that SBP should be lowered to a BP range of 120–129 mmHg in most patients.

Recommendation Grading							
	Grade I		Grade IIa		Grade IIb		Grade III

2.1 What is new and what has changed in the 2018 ESC/ESH Arterial Hypertension Guidelines?

BP treatment targets in older patients (65–80 years)	**BP treatment targets in older patients (65–80 years)**
An SBP target of between 140–150 mmHg is recommended for older patients (65–80 years).	In older patients (≥65 years), it is recommended that SBP should be targeted to a BP range of 130–139 mmHg.
BP treatment targets in patients aged over 80 years	**BP treatment targets in patients aged over 80 years**
An SBP target between 140–150 mmHg should be considered in people older than 80 years, with an initial SBP ≥160 mmHg, provided that they are in good physical and mental condition.	An SBP target range of 130–139 mmHg is recommended for people older than 80 years, if tolerated.
DBP targets	**DBP targets**
A DBP target of <90 mmHg is always recommended, except in patients with diabetes, in whom values <85 mmHg are recommended.	A DBP target of <80 mmHg should be considered for all hypertensive patients, independent of the level of risk and comorbidities.
Initiation of drug treatment	**Initiation of drug treatment**
Initiation of antihypertensive therapy with a two-drug combination may be considered in patients with markedly high baseline BP or at high CV risk.	It is recommended to initiate an antihypertensive treatment with a two-drug combination, preferably in a SPC. The exceptions are frail older patients and those at low risk and with grade 1 hypertension (particularly if SBP is <150 mmHg).
Resistant hypertension	**Resistant hypertension**
Mineralocorticoid receptor antagonists, amiloride, and the alpha-1 blocker doxazosin should be considered if no contraindication exists.	Recommended treatment of resistant hypertension is the addition of low-dose spironolactone to existing treatment, or the addition of further diuretic therapy if intolerant to spironolactone, with either eplerenone, amiloride, higher-dose thiazide/thiazide-like diuretic or a loop diuretic, or the addition of bisoprolol or doxazosin.
Device-based therapy for hypertension	**Device-based therapy for hypertension**
In case of ineffectiveness of drug treatment, invasive procedures such as renal denervation and baroreceptor stimulation may be considered.	Use of device-based therapies is not recommended for the routine treatment of hypertension, unless in the context of clinical studies and RCTs, until further evidence regarding their safety and efficacy becomes available.

Recommendation Grading							
	Grade I		Grade IIa		Grade IIb		Grade III

New concepts
BP measurement • **Wider use of out-of-office BP measurement with ABPM and/or HBPM, especially HBPM**, as an option to confirm the diagnosis of hypertension, detect white-coat and masked hypertension, and monitor BP control.
Less conservative treatment of BP in older and very old patients • **Lower BP thresholds and treatment targets for older patients**, with emphasis on considerations of biological rather than chronological age (i.e. the importance of frailty, independence, and the tolerability of treatment). • Recommendation that **treatment should never be denied or withdrawn on the basis of age**, provided that treatment is tolerated.
A SPC treatment strategy to improve BP control • **Preferred use of two-drug combination** therapy for the initial treatment of most people with hypertension. • **A single-pill treatment strategy for hypertension** with the preferred use of SPC therapy for most patients. • **Simplified drug treatment algorithms** with the preferred use of an ACE inhibitor or ARB, combined with a CCB and/or a thiazide/thiazide-like diuretic, as the core treatment strategy for most patients, with beta-blockers used for specific indications.
New target ranges for BP in treated patients • **Target *BP ranges* for treated patients** to better identify the recommended BP target and **lower safety boundaries for treated BP**, according to a patient's age and specific comorbidities.
Detecting poor adherence to drug therapy • A strong emphasis on the **importance of evaluating treatment adherence** as a major cause of poor BP control.
A key role for nurses and pharmacists in the longer-term management of hypertension • **The important role of nurses and pharmacists** in the education, support, and follow-up of treated hypertensive patients is emphasized as part of the overall strategy to improve BP control.

ABPM = ambulatory blood pressure monitoring; ACE = angiotensin-converting enzyme; AF = atrial fibrillation; ARB = angiotensin receptor blocker; BP = blood pressure; CCB = calcium channel blocker; CV = cardiovascular; CVD = cardiovascular disease; HBPM = home blood pressure monitoring; HMOD = hypertension-mediated organ damage; SCORE = Systematic COronary Risk Evaluation; SPC = single-pill combination.

ESC European Society of Cardiology European Heart Journal (2018) 39, 3021–3104 doi:10.1093/eurheartj/ehy339

ESC/ESH GUIDELINES

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Table 3 Classification of office blood pressure[a] and definitions of hypertension grade[b]

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension[b]	≥140	and	<90

BP = blood pressure; SBP = systolic blood pressure.
[a]BP category is defined according to seated clinic BP and by the highest level of BP, whether systolic or diastolic.
[b]Isolated systolic hypertension is graded 1, 2, or 3 according to SBP values in the ranges indicated.
The same classification is used for all ages from 16 years.

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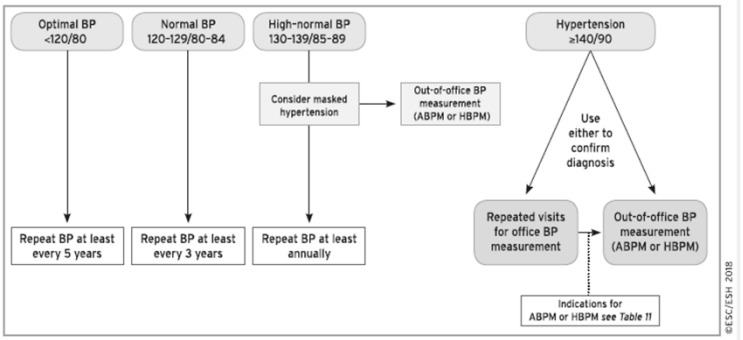

Figure 2 Screening and diagnosis of hypertension. ABPM = ambulatory blood pressure monitoring; BP = blood pressure; HBPM = home blood pressure monitoring.
[a]After detecting a specific BP category on screening, either confirm BP elevation with repeated office BP measurements on repeat visits or arrange use of out-of-office BP to confirm the diagnosis of hypertension.

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ESC/ESH GUIDELINES

2018 ESC/ESH Guidelines for the management of arterial hypertension

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Hypertension disease staging	Other risk factors, HMOD, or disease	BP (mmHg) grading			
		High normal SBP 130-139 DBP 85-89	Grade 1 SBP 140-159 DBP 90-99	Grade 2 SBP 160-179 DBP 100-109	Grade 3 SBP ≥180 or DBP ≥110
Stage 1 (uncomplicated)	No other risk factors	Low risk	Low risk	Moderate risk	High risk
	1 or 2 risk factors	Low risk	Moderate risk	Moderate to high risk	High risk
	≥3 risk factors	Low to Moderate risk	Moderate to high risk	High Risk	High risk
Stage 2 (asymptomatic disease)	HMOD, CKD grade 3, or diabetes mellitus without organ damage	Moderate to high risk	High risk	High risk	High to very high risk
Stage 3 (established disease)	Established CVD, CKD grade ≥4, or diabetes mellitus with organ damage	Very high risk	Very high risk	Very high risk	Very high risk

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Figure 1 Classification of hypertension stages according to blood pressure levels, presence of cardiovascular risk factors, hypertension-mediated organ damage, or comorbidities. CV risk is illustrated for a middle-aged male. The CV risk does not necessarily correspond to the actual risk at different ages. The use of the SCORE system is recommended for formal estimation of CV risk for treatment decisions. BP = blood pressure; CKD = chronic kidney disease; CV = cardiovascular; DBP = diastolic blood pressure; HMOD = hypertension-mediated organ damage; SBP = systolic blood pressure; SCORE = Systematic COronary Risk Evaluation.

FATTORI DI RISCHIO

Stili di Vita

Dieta Ipercalorica o ricca in Acidi grassi Saturi e Colesterolo

Fumo

Consumo eccessivo di Alcol

Sedentarietà

FRC Modificabili

Ipertensione Arteriosa

Elevato LDL-Colesterolo

Ridotto HDL-Colesterolo

Elevati Trigliceridi

Iperglicemia/Diabete

Obesità

Fattori Trombogenici

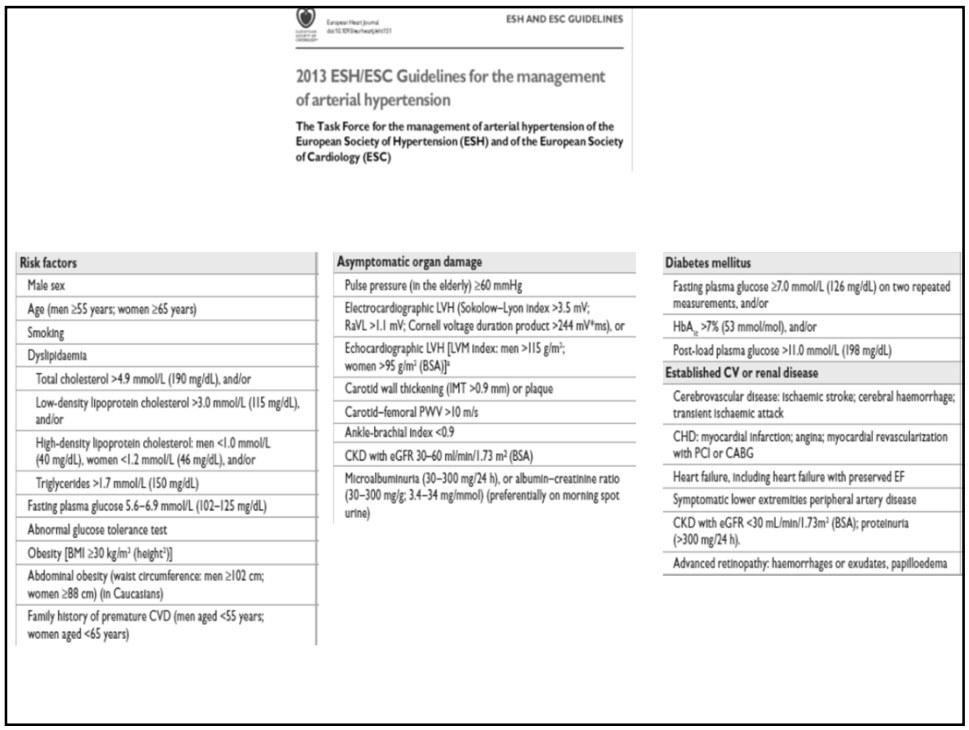
FRC Non Modificabili

Età

Sesso

Storia familiare di CHD o altra malattia aterosclerotica precoce (maschi<55 a, femmine<65 a

Anamnesi positiva per CHD o altra malattia aterosclerotica



ESH AND ESC GUIDELINES

2013 ESH/ESC Guidelines for the management of arterial hypertension

The Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC)

Risk factors
Male sex
Age (men ≥55 years; women ≥65 years)
Smoking
Dyslipidaemia
Total cholesterol >4.9 mmol/L (190 mg/dL), and/or
Low-density lipoprotein cholesterol >3.0 mmol/L (115 mg/dL), and/or
High-density lipoprotein cholesterol: men <1.0 mmol/L (40 mg/dL), women <1.2 mmol/L (46 mg/dL), and/or
Triglycerides >1.7 mmol/L (150 mg/dL)
Fasting plasma glucose 5.6–6.9 mmol/L (102–125 mg/dL)
Abnormal glucose tolerance test
Obesity [BMI ≥30 kg/m² (height²)]
Abdominal obesity (waist circumference: men ≥102 cm; women ≥88 cm) (in Caucasians)
Family history of premature CVD (men aged <55 years; women aged <65 years)

Asymptomatic organ damage
Pulse pressure (in the elderly) ≥60 mmHg
Electrocardiographic LVH (Sokolow–Lyon index >3.5 mV; RaVL >1.1 mV; Cornell voltage duration product >244 mV²ms), or
Echocardiographic LVH [LVM index: men >115 g/m²; women >95 g/m² (BSA)]²
Carotid wall thickening (IMT >0.9 mm) or plaque
Carotid–femoral PWV >10 m/s
Ankle-brachial index <0.9
CKD with eGFR 30–60 ml/min/1.73 m² (BSA)
Microalbuminuria (30–300 mg/24 h), or albumin–creatinine ratio (30–300 mg/g; 3.4–34 mg/mmol) (preferentially on morning spot urine)

Diabetes mellitus
Fasting plasma glucose ≥7.0 mmol/L (126 mg/dL) on two repeated measurements, and/or
HbA_{1c} >7% (53 mmol/mol), and/or
Post-load plasma glucose >11.0 mmol/L (198 mg/dL)
Established CV or renal disease
Cerebrovascular disease: ischaemic stroke; cerebral haemorrhage; transient ischaemic attack
CHD: myocardial infarction; angina; myocardial revascularization with PCI or CABG
Heart failure, including heart failure with preserved EF
Symptomatic lower extremities peripheral artery disease
CKD with eGFR <30 mL/min/1.73m² (BSA); proteinuria (>300 mg/24 h).
Advanced retinopathy: haemorrhages or exudates, papilloedema

FATTORI DI RISCHIO CHE INFLUENZANO LA PROGNOSI

FR CARDIOVASCOLARI UTILIZZATI PER LA STRATIFICAZIONE

PA sistolica e diastolica

Età: uomini > 55 aa.
donne > 65 aa.

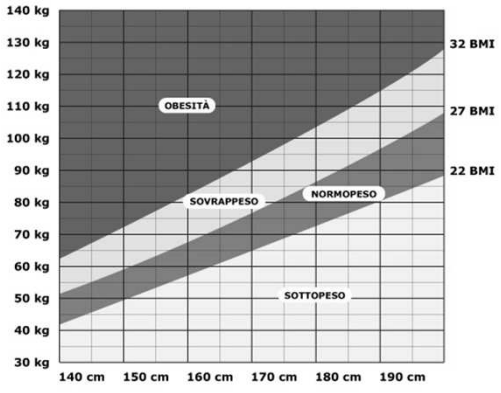
Fumo di sigaretta

Dislipidemia: col tot > 250 mg/dl o c-LDL > 155 mg/dl
o c-HDL < 40 mg/dl (uomini), < 48 mg/dl (donne)

Familiarità per mal. cardiovascolari precoci
(< 55 aa. uomini, < 65 aa. donne)

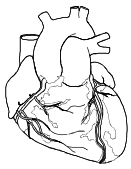
OBESITA' - SOVRAPPESO

Classificazione	BMI (kg/m²)
	Cut-off principali
Sottopeso	< 18,50
Magrezza severa	<16,00
Magrezza moderata	16,00 – 16,99
Magrezza leggera	17,00 – 18,49
Normopeso	18,50 – 24,99
Sovrappeso	≥ 25,00
Pre-obesità	25,00 – 29,99
Obesità	≥ 30,00
Obesità I classe	30,00 – 34,99
Obesità II classe	35,00 – 39,99
Obesità III classe	≥ 40,00

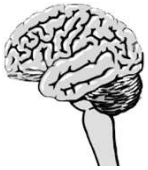


IPERTENSIONE: AZIONE SUGLI ORGANI BERSAGLIO

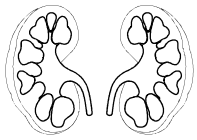
Ipertensione



Angina pectoris
Angina instabile
Infarto miocardico
Morte improvvisa
Scompenso cardiaco

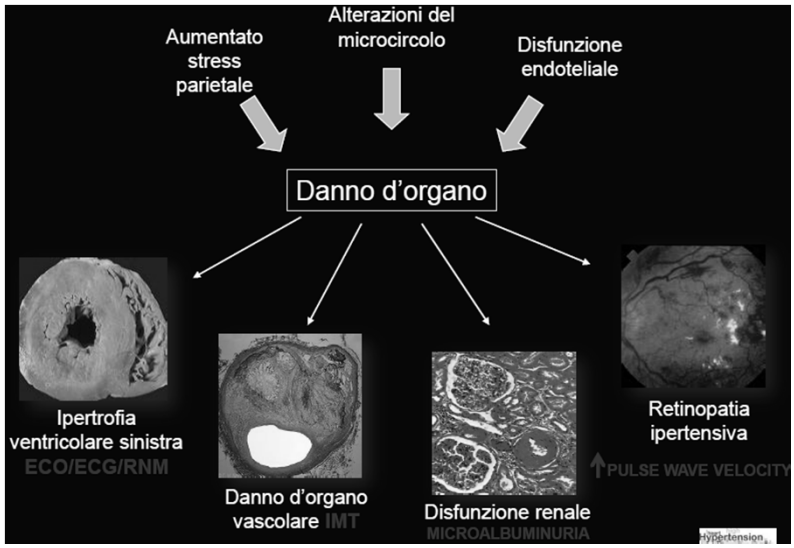


TIA
Ictus ischemico
Ictus emorragico

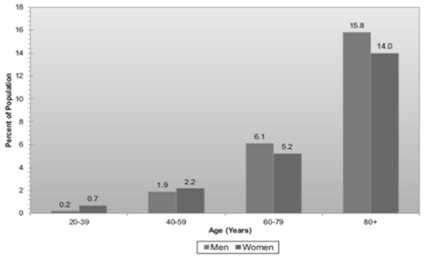


Patologia renovascolare
Insufficienza renale

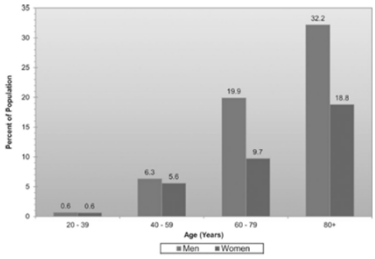
Claudicatio
Aneurisma
Ischemia degli arti



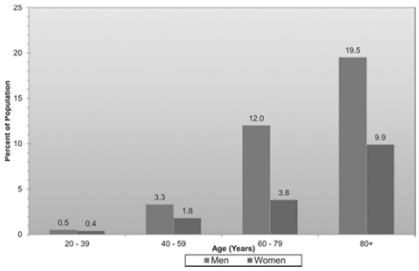
Prevalence of stroke by age and sex



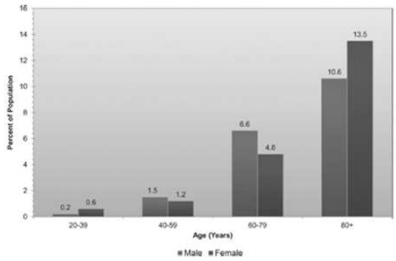
Prevalence of coronary heart disease by age and sex



Prevalence of myocardial infarction by age and sex



Prevalence of heart failure by sex and age



AHA Statistical 2016 Update, Circulation 2016

FATTORI DI RISCHIO CHE INFLUENZANO LA PROGNOSI

TARGET ORGAN DAMAGE (TOD)

IPERTROFIA VENTRICOLARE SX (IVS) **(ECG, ECOCARDIOGRAMMA)**

EVIDENZA DI ISPESSIMENTO DELLA PARETE ARTERIOSA O DI PLACCHE ATEROSCLEROTICHE **(ECODOPPLER)**

LIEVE INCREMENTO DELLA CREATININA SIERICA (M 1.3 – 1.5 mg/dl)

MICROALBUMINURIA (30 – 300 mg/24h

ECCESSO DI RISCHIO IN IPERTESI CON IVS ALL'ECG

5 VOLTE PIU' ALTO PER INFARTO MIOCARDICO

6 - 7 VOLTE PIU' ALTO PER SCOMPENSO CARDIACO

3 - 10 VOLTE PIU' ALTO PER ICTUS

FATTORI DI RISCHIO CHE INFLUENZANO LA PROGNOSI

DIABETE MELLITO

GLICEMIA A DIGIUNO > 126 mg/dl

GLICEMIA POSTPRANDIALE > 198 mg/dl

FATTORI DI RISCHIO CHE INFLUENZANO LA PROGNOSI

CONDIZIONI CLINICHE ASSOCIATE

MALATTIE CEREBROVASCOLARI:
STROKE ISCHEMICO, EMORRAGIA CEREBRALE, TIA

PATOLOGIE CARDIACHE:
IMA, ANGINA, RIVASCOLARIZZAZIONE CORONARICA,
SCOMPENSO CARDIACO CONGESTIZIO

PATOLOGIE RENALI:
NEFROPATIA DIABETICA,
DANNO RENALE (creatinina M > 1.5 mg/dl)
PROTEINURIA (> 300 mg/24 h)

VASCULOPATIA PERIFERICA

RETINOPATIA AVANZATA:
EMORRAGIE OD ESSUDATI, PAPILLEDEMA

VALUTAZIONE DIAGNOSTICA

SCOPI

- **Stabilire i livelli pressori**
- **Identificare le cause di ipertensione secondaria**
- **Stratificazione prognostica del rischio**

PROCEDURE

1) **Misurazione ripetuta della pressione arteriosa**
2) **Valutazione della storia clinica**
3) **Esame fisico**
4) **Valutazione strumentale e di laboratorio**

DEFINIZIONE DI IPERTENSIONE

Table 9 Definitions of hypertension according to office, ambulatory, and home blood pressure levels

Category	SBP (mmHg)		DBP (mmHg)
Office BP[a]	≥140	and/or	≥90
Ambulatory BP			
Daytime (or awake) mean	≥135	and/or	≥85
Night-time (or asleep) mean	≥120	and/or	≥70
24 h mean	≥130	and/or	≥80
Home BP mean	≥135	and/or	≥85

BP = blood pressure; DBP = diastolic blood pressure; SBP = systolic blood pressure.
[a]Refers to conventional office BP rather than unattended office BP.

MISURAZIONE DELLA PRESSIONE ARTERIOSA

- **Sfigmomanometria clinica ambulatoriale** **(≥140/90 mmHg)**
- **Misurazione domiciliare (automisurazione)** **(≥ 135/85 mmHg)**
- **Monitoraggio delle 24 ore** **(≥ 130/85 mmHg)**

MONITORAGGIO DELLE 24 ORE

INDICAZIONI

- **Considerevole variabilità tra i valori pressori ambulatoriali**
- **Elevati valori pressori in pz. con rischio cardiovascolare basso**
- **Marcata discrepanza tra valori pressori ambulatoriali e domiciliari**
- **Sospetta ipertensione secondaria**
- **Monitoraggio terapia (efficacia antipertensiva – copertura 24 ore ecc.)**
- **Dipping notturno**
- **Finalità di ricerca**

Table 11 Clinical indications for home blood pressure monitoring or ambulatory blood pressure monitoring

Conditions in which white-coat hypertension is more common, e.g.: • Grade I hypertension on office BP measurement • Marked office BP elevation without HMOD
Conditions in which masked hypertension is more common, e.g.: • High–normal office BP • Normal office BP in individuals with HMOD or at high total CV risk
Postural and post-prandial hypotension in untreated and treated patients
Evaluation of resistant hypertension Evaluation of BP control, especially in treated higher-risk patients Exaggerated BP response to exercise
When there is considerable variability in the office BP
Evaluating symptoms consistent with hypotension during treatment
Specific indications for ABPM rather than HBPM: • Assessment of nocturnal BP values and dipping status (e.g. suspicion of nocturnal hypertension, such as in sleep apnoea, CKD, diabetes, endocrine hypertension, or autonomic dysfunction)

ABPM = ambulatory blood pressure monitoring; BP = blood pressure; CKD = chronic kidney disease; CV = cardiovascular; HBPM = home blood pressure monitoring; HMOD = hypertension-mediated organ damage.

VALUTAZIONI STRUMENTALI E DI LABORATORIO

Table 14 Routine workup for evaluation of hypertensive patients

Routine laboratory tests
Haemoglobin and/or haematocrit
Fasting blood glucose and glycated HbA_{1c}
Blood lipids: total cholesterol, LDL cholesterol, HDL cholesterol
Blood triglycerides
Blood potassium and sodium
Blood uric acid
Blood creatinine and eGFR
Blood liver function tests
Urine analysis: microscopic examination; urinary protein by dipstick test or, ideally, albumin:creatinine ratio
12-lead ECG

eGFR = estimated glomerular filtration rate; ECG = electrocardiogram; HbA_{1c} = haemoglobin A1c.

VALUTAZIONI STRUMENTALI E DI LABORATORIO

Table 15 Assessment of hypertension-mediated organ damage

Basic screening tests for HMOD	Indication and interpretation
12-lead ECG	Screen for LVH and other possible cardiac abnormalities, and to document heart rate and cardiac rhythm
Urine albumin:creatinine ratio	To detect elevations in albumin excretion indicative of possible renal disease
Blood creatinine and eGFR	To detect possible renal disease
Fundoscopy	To detect hypertensive retinopathy, especially in patients with grade 2 or 3 hypertension
More detailed screening for HMOD	
Echocardiography	To evaluate cardiac structure and function, when this information will influence treatment decisions
Carotid ultrasound	To determine the presence of carotid plaque or stenosis, particularly in patients with cerebrovascular disease or vascular disease elsewhere
Abdominal ultrasound and Doppler studies	• To evaluate renal size and structure (e.g. scarring) and exclude renal tract obstruction as possible underlying causes of CKD and hypertension • Evaluate abdominal aorta for evidence of aneurysmal dilatation and vascular disease • Examine adrenal glands for evidence of adenoma or phaeochromocytoma (CT or MRI preferred for detailed examination); see section 8.2 regarding screening for secondary hypertension • Renal artery Doppler studies to screen for the presence of renovascular disease, especially in the presence of asymmetric renal size
PWV	An index of aortic stiffness and underlying arteriosclerosis
ABI	Screen for evidence of LEAD
Cognitive function testing	To evaluate cognition in patients with symptoms suggestive of cognitive impairment
Brain imaging	To evaluate the presence of ischaemic or haemorrhagic brain injury, especially in patients with a history of cerebrovascular disease or cognitive decline

ABI = ankle-brachial index; CKD = chronic kidney disease; CT = computed tomography; ECG = electrocardiogram; eGFR = estimated glomerular filtration rate; HMOD = hypertension-mediated organ damage; LEAD = lower extremity artery disease; LVH = left ventricular hypertrophy; MRI = magnetic resonance imaging; PWV = pulse wave velocity.

VALUTAZIONI STRUMENTALI E DI LABORATORIO

Valutazione più completa (specialistica)

- **Ipertensione complicata: valutazione della funzionalità cardiaca, cerebrale e renale**
- **Cercare la presenza di forme secondarie di ipertensione: misurazione di renina, aldosterone, ormoni corticosteroidi, catecolamine, arteriografia; ecografia renale e surrenalica, TC, RMN.**

Table 18 Sensitivity to detect treatment-induced changes, reproducibility and operator independence, time to changes, and prognostic value of changes provided by markers of hypertension-mediated organ damage

Marker of HMOD	Sensitivity to changes	Reproducibility and operator independence	Time to changes	Prognostic value of the change
LVH by ECG	Low	High	Moderate (>6 months)	Yes
LVH by echocardiogram	Moderate	Moderate	Moderate (>6 months)	Yes
LVH by CMR	High	High	Moderate (>6 months)	No data
eGFR	Moderate	High	Very slow (years)	Yes
Urinary protein excretion	High	Moderate	Fast (weeks to months)	Moderate
Carotid IMT	Very low	Low	Slow (>12 months)	No
PWV	High	Low	Fast (weeks to months)	Limited data
Ankle-brachial index	Low	Moderate	Slow (>12 months)	Moderate

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CMR = cardiac magnetic resonance; ECG = electrocardiogram; eGFR = estimated glomerular filtration rate; HMOD = hypertension-mediated organ damage; IMT = intima-media thickness; LVH = left ventricular hypertrophy; PWV = pulse wave velocity.

OBIETTIVI DEL TRATTAMENTO

Può essere raccomandato in tutti i pz. ipertesi ridurre la PA a valori inferiori ai 140/90 mmHg ed eventualmente a valori ancora più bassi, se ben tollerati

Il raggiugimento in corso di terapia di valori pressori sistolici < 140 mHg può essere difficile, specie negli anziani

Nei pazienti diabetici la PA deve essere ridotta a valori < 130/80 mmHg

OBIETTIVI DEL TRATTAMENTO

L'obiettivo primario del trattamento del pz. iperteso è quello di ottenere la massima riduzione del rischio cardiovascolare globale a lungo termine.

E' perciò richiesto il trattamento di tutti i fattori di rischio reversibili identificati, una gestione appropriata delle condizioni cliniche associate, oltre al trattamento dell'ipertensione stessa

Trattamento antiipertensivo iniziale

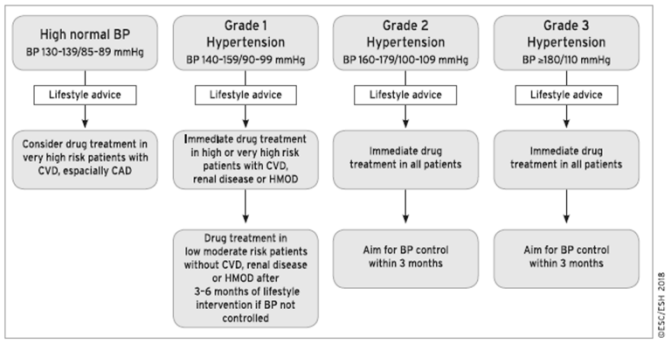

Figure 3 Initiation of blood pressure-lowering treatment (lifestyle changes and medication) at different initial office blood pressure levels. BP = blood pressure; CAD = coronary artery disease; CVD = cardiovascular disease; HMOD = hypertension-mediated organ damage.

Trattamento antiipertensivo iniziale

Table 19 Summary of office blood pressure thresholds for treatment

Age group	Office SBP treatment threshold (mmHg)					Office DBP treatment threshold (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke/TIA	
18 - 65 years	≥140	≥140	≥140	≥140[a]	≥140[a]	≥90
65 - 79 years	≥140	≥140	≥140	≥140[a]	≥140[a]	≥90
>80 years	≥160	≥160	≥160	≥160	≥160	≥90
Office DBP treatment threshold (mmHg)	≥90	≥90	≥90	≥90	≥90	

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BP = blood pressure; CAD = coronary artery disease; CKD = chronic kidney disease; DBP = diastolic blood pressure; SBP = systolic blood pressure; TIA = transient ischaemic attack.
[a]Treatment may be considered in these very high-risk patients with high–normal SBP (i.e. SBP 130–140 mmHg).

Trattamento antiipertensivo iniziale

Other risk factors, asymptomatic organ damage or disease	Blood Pressure (mmHg)			
	High normal SBP 130–139 or DBP 85–89	Grade 1 HT SBP 140–159 or DBP 90–99	Grade 2 HT SBP 160–179 or DBP 100–109	Grade 3 HT SBP ≥180 or DBP ≥110
No other RF	• No BP intervention	• Lifestyle changes for several months • Then add BP drugs targeting <140/90	• Lifestyle changes for several weeks • Then add BP drugs targeting <140/90	• Lifestyle changes • Immediate BP drugs targeting <140/90
1–2 RF	• Lifestyle changes • No BP intervention	• Lifestyle changes for several weeks • Then add BP drugs targeting <140/90	• Lifestyle changes for several weeks • Then add BP drugs targeting <140/90	• Lifestyle changes • Immediate BP drugs targeting <140/90
≥3 RF	• Lifestyle changes • No BP intervention	• Lifestyle changes for several weeks • Then add BP drugs targeting <140/90	• Lifestyle changes • BP drugs targeting <140/90	• Lifestyle changes • Immediate BP drugs targeting <140/90
OD, CKD stage 3 or diabetes	• Lifestyle changes • No BP intervention	• Lifestyle changes • BP drugs targeting <140/90	• Lifestyle changes • BP drugs targeting <140/90	• Lifestyle changes • Immediate BP drugs targeting <140/90
Symptomatic CVD, CKD stage ≥4 or diabetes with OD/RFs	• Lifestyle changes • No BP intervention	• Lifestyle changes • BP drugs targeting <140/90	• Lifestyle changes • BP drugs targeting <140/90	• Lifestyle changes • Immediate BP drugs targeting <140/90

BP = blood pressure; CKD = chronic kidney disease; CV = cardiovascular; CVD = cardiovascular disease; DBP = diastolic blood pressure; HT = hypertension; OD = organ damage; RF = risk factor; SBP = systolic blood pressure.

MODIFICHE DELLO STILE DI VITA

- **L'abolizione del fumo**
- **Il calo ponderale**
- **La riduzione del consumo di alcolici, se eccessivo**
- **L'esercizio fisico**
- **La dieta iposodica**
- **Incremento di apporto alimentare di frutta e verdura**
- **Riduzione dei grassi totali alimentari**

STRATEGIE TERAPEUTICHE

Nella maggior parte dei casi il trattamento dovrebbe essere iniziato gradualmente e valutato nell'arco di alcune settimane

A seconda dei valori pressori di base e della presenza o meno di altri fattori, è ragionevole iniziare la terapia con uno o due farmaci, entrambi a basso dosaggio

E' probabile che per raggiungere il target pressorio sia necessario l'impiego di una terapia di associazione tra due o più farmaci.

Entrambe le strategie terapeutiche presentano vantaggi e svantaggi.

SCELTE TERAPEUTICHE

I principali benefici dipendono principalmente dalla riduzione dei valori pressori e solo in minima parte dal tipo di farmaco impiegato

Le classi principali di farmaci antipertensivi - diuretici, β-bloccanti, calcioantagonisti diidropiridinici, ACE-inibitori, bloccanti recettoriali dell'AT II – (inibitori diretti della renina) sono tutte indicate come scelte terapeutiche con cui iniziare e proseguire il trattamento

Identificare una classe come prima scelta è di relativa importanza vista la frequente necessità di associare due o più farmacici

Tali farmaci si differenziano però per alcuni specifici effetti farmacologici con conseguenti indicazioni differenti

Tollerabilità diversa da paziente a paziente

SCELTE TERAPEUTICHE

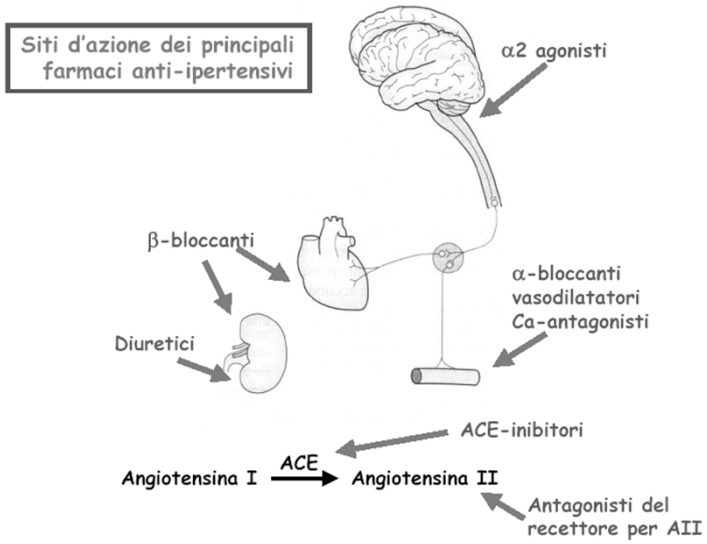
Altri farmaci

- **α-bloccanti**
- **farmaci che agiscono a livello del SNC (clonidina, metildopa)**
- **farmaci agenti sui recettori imidazolinici I_1**
- **agonisti dei recettori α_2 adrenergici**

Non sono consigliati come prima scelta

Consigliabile invece il loro utilizzo in terapia di associazione

Alcuni di questi hanno indicazioni ed usi specifici



SCELTE TERAPEUTICHE

La scelta terapeutica sarà influenzata da numerosi fattori:

- **I farmaci impiegati in passato;**
- **Il profilo di rischio del paziente;**
- **La presenza o meno di TOD, diabete e CCA;**
- **Il costo dei vari farmaci;**
- **L'eventuale preferenza espressa dal paziente.**

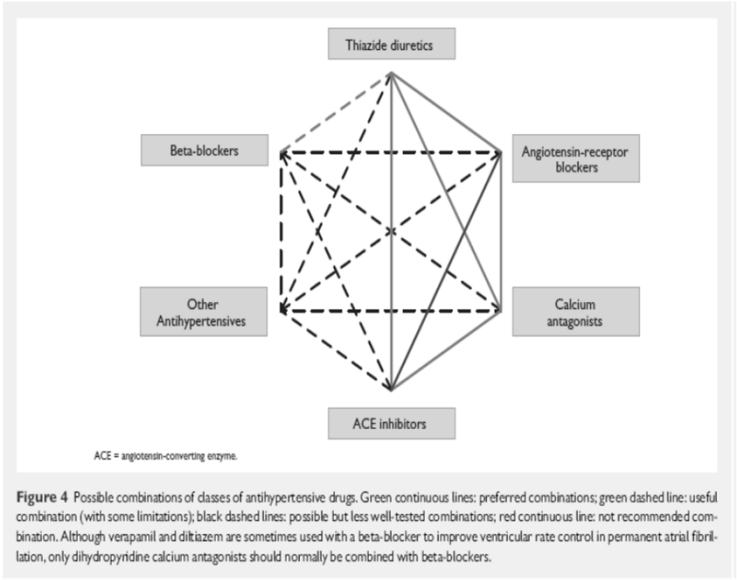


Figure 4 Possible combinations of classes of antihypertensive drugs. Green continuous lines: preferred combinations; green dashed line: useful combination (with some limitations); black dashed lines: possible but less well-tested combinations; red continuous line: not recommended combination. Although verapamil and diltiazem are sometimes used with a beta-blocker to improve ventricular rate control in permanent atrial fibrillation, only dihydropyridine calcium antagonists should normally be combined with beta-blockers.

Table 20 Compelling and possible contraindications to the use of specific antihypertensive drugs

Drug	Contraindications	
	Compelling	Possible
Diuretics (thiazides/thiazide-like, e.g. chlorthalidone and indapamide)	• Gout	• Metabolic syndrome • Glucose intolerance • Pregnancy • Hypercalcaemia • Hypokalaemia
Beta-blockers	• Asthma • Any high-grade sinoatrial or atrioventricular block • Bradycardia (heart rate <60 beats per min)	• Metabolic syndrome • Glucose intolerance • Athletes and physically active patients
Calcium antagonists (dihydropyridines)		• Tachyarrhythmia • Heart failure (HFrEF, class III or IV) • Pre-existing severe leg oedema
Calcium antagonists (verapamil, diltiazem)	• Any high-grade sinoatrial or atrioventricular block • Severe LV dysfunction (LV ejection fraction <40%) • Bradycardia (heart rate <60 beats per min)	• Constipation
ACE inhibitors	• Pregnancy • Previous angioneurotic oedema • Hyperkalaemia (potassium >5.5 mmol/L) • Bilateral renal artery stenosis	• Women of child-bearing potential without reliable contraception
ARBs	• Pregnancy • Hyperkalaemia (potassium >5.5 mmol/L) • Bilateral renal artery stenosis	• Women of child-bearing potential without reliable contraception

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ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; HFrEF = heart failure with reduced ejection fraction; LV = left ventricular.

IPERTENSIONE NON COMPLICATA

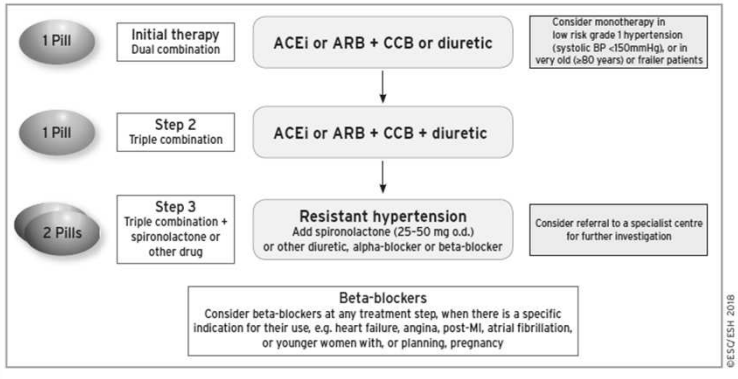


Figure 4 Core drug treatment strategy for uncomplicated hypertension. The core algorithm is also appropriate for most patients with HMOD, cerebrovascular disease, diabetes, or PAD. ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; CCB = calcium channel blocker; HMOD = hypertension-mediated organ damage; MI = myocardial infarction; o.d. = omni die (every day); PAD = peripheral artery disease.

IPERTENSIONE E MALATTIA CORONARICA

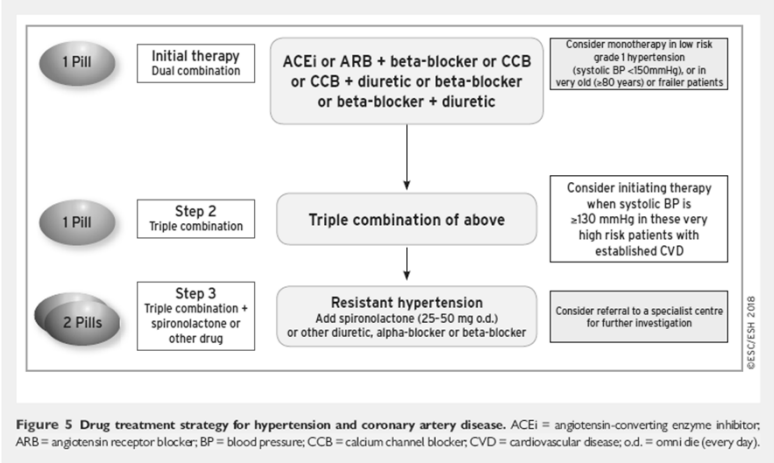


Figure 5 Drug treatment strategy for hypertension and coronary artery disease. ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BP = blood pressure; CCB = calcium channel blocker; CVD = cardiovascular disease; o.d. = omni die (every day).

IPERTENSIONE E MALATTIA RENALE

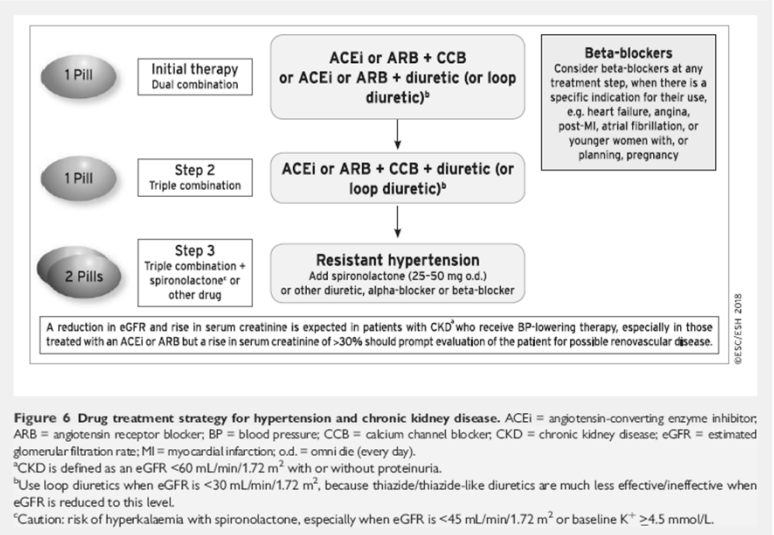
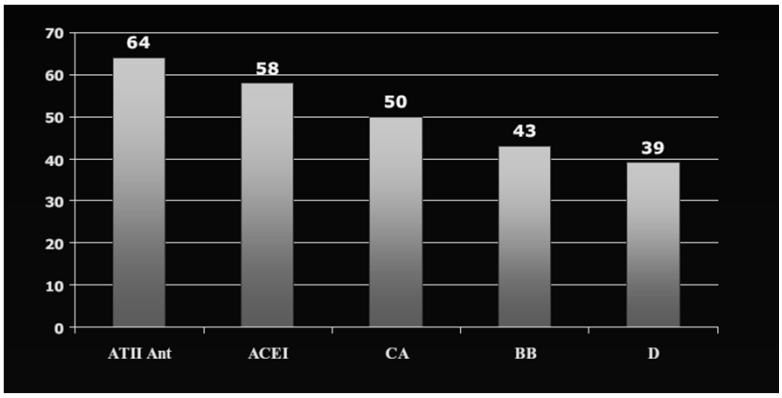


Figure 6 Drug treatment strategy for hypertension and chronic kidney disease. ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BP = blood pressure; CCB = calcium channel blocker; CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; MI = myocardial infarction; o.d. = omni die (every day).
[a]CKD is defined as an eGFR <60 mL/min/1.72 m^2 with or without proteinuria.
[b]Use loop diuretics when eGFR is <30 mL/min/1.72 m^2, because thiazide/thiazide-like diuretics are much less effective/ineffective when eGFR is reduced to this level.
[c]Caution: risk of hyperkalaemia with spironolactone, especially when eGFR is <45 mL/min/1.72 m^2 or baseline K^+ ≥4.5 mmol/L.

Persistenza di terapia a 1 anno



IPERTENSIONE RESISTENTE ALLA TERAPIA

- **Ipertensione da cause secondarie**
- **Scarsa compliance alla terapia**
- **Assunzione di farmaci che aumentano la pressione**
- **Incapacità di modificare lo stile di vita:**
  - **incremento ponderale**
  - **elevato consumo di bevande alcoliche**
- **Ipervolemia plasmatica:**
  - **terapia diuretica inadeguata**
  - **insufficienza renale progressiva**
  - **elevato introito alimentaredi sodio**
- **Ipertensione ambulatoriale isolata (da camice bianco)**
- **Mancato utilizzo di bracciali di dimensioni adeguate in pazienti con arti voluminosi**

IPERTENSIONE RESISTENTE ALLA TERAPIA: Caratteristiche del paziente

- Elevati valori basali di PA
- Età avanzata
- Obesità
- Eccessivo introito di sale
- Malattia renale cronica
- Diabete

Table 28 Medications and other substances that may increase blood presssure[397]

Medication/substance	
Oral contraceptive pill	Especially oestrogen containing; cause hypertension in ~5% of women, usually mild but can be severe
Diet pills	For example, phenylpropanolamine and sibutramine
Nasal decongestants	For example, phenylephrine hydrochloride and naphazoline hydrochloride
Stimulant drugs	Amphetamine, cocaine, and ecstasy; these substances usually cause acute rather than chronic hypertension
Liquorice	Chronic excessive liquorice use mimics hyperaldosteronism by stimulating the mineralocorticoid receptor and inhibiting cortisol metabolism
Immunosuppressive medications	For example, cyclosporin A (tacrolimus has less effect on BP and rapamycin has almost no effect on BP) and steroids (e.g. corticosteroids and hydrocortisone)
Antiangiogenic cancer therapies	Antiangiogenic drugs such as VEGF inhibitors (e.g. bevacizumab), tyrosine kinase inhibitors (e.g. sunitinib), and sorafenib have been reported to increase BP
Other drugs and substances that may raise BP	Anabolic steroids, erythropoietin, non-steroidal anti-inflammatory drugs, and herbal remedies (e.g. ephedra and ma huang)

BP = blood pressure; VEGF = vascular endothelial growth factor.

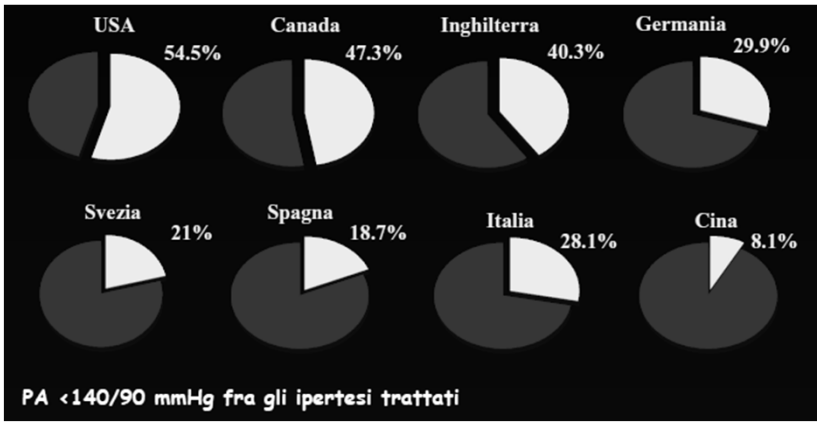
8.9 Women, pregnancy, oral contraception, and hormone-replacement therapy

Management of hypertension in pregnancy

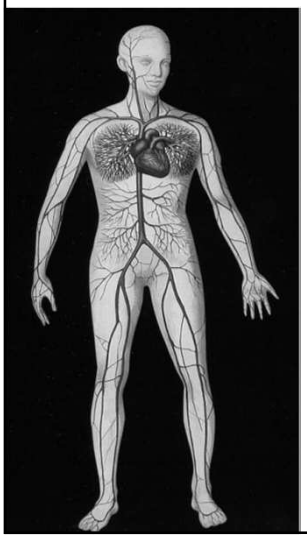
Recommendations	Class[a]	Level[b]
In women with gestational hypertension, pre-existing hypertension superimposed by gestational hypertension, or with hypertension and subclinical organ damage or symptoms, initiation of drug treatment is recommended when SBP is ≥140 mmHg or DBP ≥90 mmHg.	I	C
In all other cases, initiation of drug treatment is recommended when SBP is ≥150 mmHg or DBP is ≥95 mmHg.	I	C
Methyldopa, labetalol, and CCBs are recommended as the drugs of choice for the treatment of hypertension in pregnancy.[447,448]	I	B (methyldopa)
	I	C (labetalol or CCBs)
ACE inhibitors, ARBs, or direct renin inhibitors are not recommended during pregnancy.	III	C
SBP ≥170 mmHg or DBP ≥110 mmHg in a pregnant woman is an emergency, and admission to hospital is recommended.	I	C
In severe hypertension, drug treatment with i.v. labetalol, oral methyldopa, or nifedipine is recommended.	I	C
The recommended treatment for hypertensive crisis is i.v. labetalol or nicardipine and magnesium.	I	C
In pre-eclampsia associated with pulmonary oedema, nitroglycerin given as an i.v. infusion is recommended.	I	C
In women with gestational hypertension or mild pre-eclampsia, delivery is recommended at 37 weeks.[453]	I	B
It is recommended to expedite delivery in pre-eclampsia with adverse conditions, such as visual disturbances or haemostatic disorders.	I	C

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; CCB = calcium channel blocker; DBP = diastolic blood pressure; i.v. = intravenous; SBP = systolic blood pressure.
[a]Class of recommendation.
[b]Level of evidence.

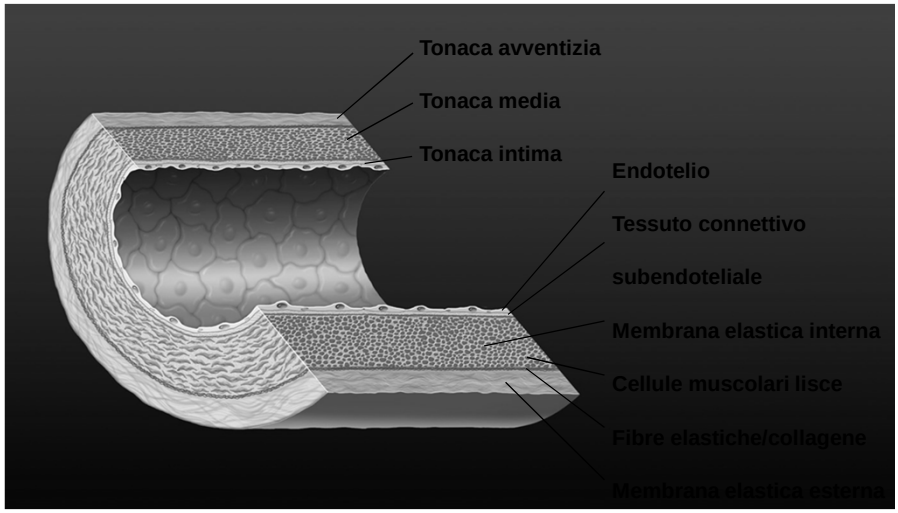
Percentuale di controllo della PA



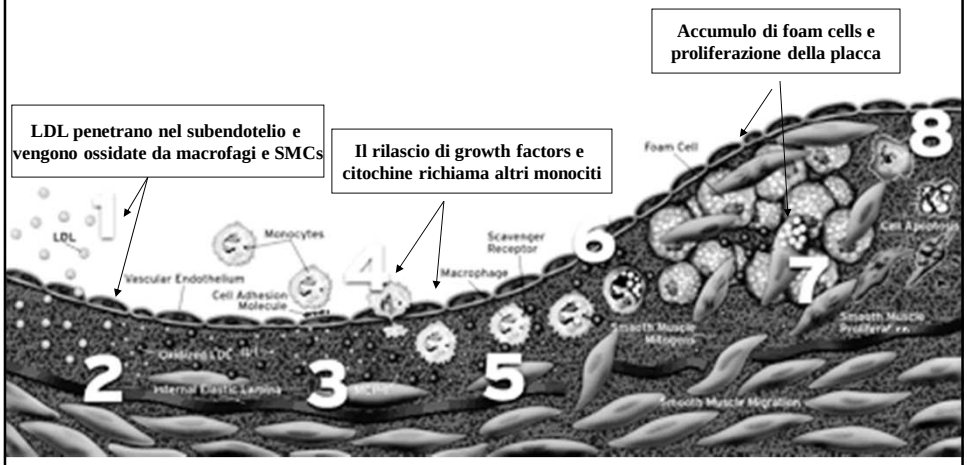
MALATTIA ATEROSCLEROTICA



Parete arteriosa normale

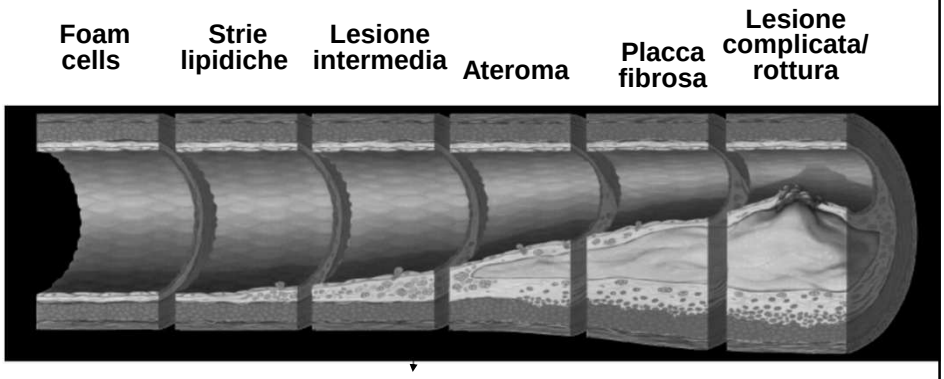


Sviluppo della placca aterosclerotica



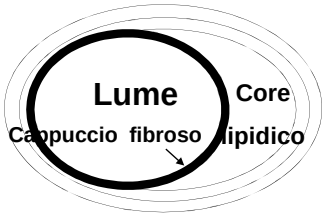
Faxon et al, Circulation 2004

Evoluzione dell'aterosclerosi



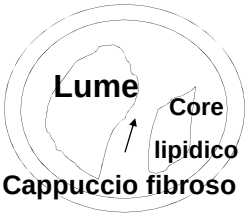
Pepine, Am J Cardiol 1998

Integrità della placca



Placca Vulnerabile

- Cappuccio fibroso sottile
- Infiltrato cellulare infiammatorio: attività proteolitica
- Placca ricca in lipidi



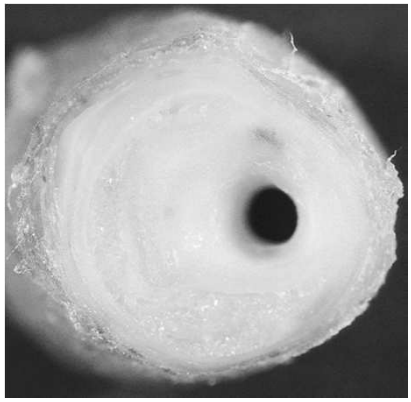
Placca Stabile

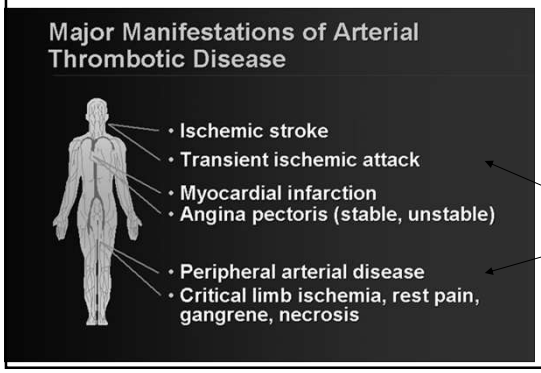
Cappuccio fibroso spesso

- Cellule muscolari lisce: matrice
- extracellulare più rappresentata
- Placca povera in lipidi

Libby, Circulation 1995

Manifestazioni cliniche





Placca vulnerabile
Placca stabile

Cardiopatia ischemica: definizione

Squilibrio richiesta metabolica - apporto di O_2 al miocardio

Manifestazioni cliniche

arresto cardiaco primario

angina pectoris

infarto miocardico

scompenso cardiaco

aritmie

FISIOPATOLOGIA

- In condizioni basali, l'estrazione di O_2 è circa del 70%.
- In caso di aumento della domanda metabolica il solo meccanismo di compenso è dato dal proporzionale aumento del flusso coronarico.
- Importanza della diastole.

Determinanti del consumo di O2

frequenza cardiaca (↑ consumo O_2, ↓ flusso coronarico)

contrattilità (↑ consumo O_2)

tensione della parete miocardica (post-carico) in rapporto a pressione sviluppata all'interno e precarico (↑ all'aumentare delle resistenze e del ritorno venoso)

Effetti dell'ischemia a livello miocardico I

Alterazioni metaboliche

- ↓pH,↓ATP
- radicali liberi

Alterazioni elettriche

- ischemia
- lesione
- necrosi

Effetti dell'ischemia a livello miocardico II

Alterazioni meccaniche

-effetti sulla contrazione ventricolare (↓ gettata sistolica e portata cardiaca, depressione globale del ventricolo sx, scompenso)

-effetti sulla funzione diastolica: ostacolo al riempimento del ventricolo

Angina pectoris: classificazione

Criterio fisiopatologico

Angina primaria (variazioni dinamiche: spasmo, a. di Prinzmetal)

Angina secondaria (↑fabbisogno metabolico, limitato da stenosi coronariche)

Classificazione

Criterio descrittivo

Angina spontanea (primaria pura): episodi a riposo o imprevedibili, senza causa scatenante

Angina da sforzo a soglia fissa (secondaria pura): sintomatologia evocata, in modo riproducibile e prevedibile, sempre dallo stesso livello di attività fisica

Angina mista (primaria + secondaria): episodi a riposo e da sforzo, questi ultimia soglia variabile

Classificazione

Criterio clinico-prognostico

Angina stabile (forma cronica): scarsa evolutività

Angina instabile: spiccata tendenza alla evoluzione (IMA, morte improvvisa)

European Heart Journal (2013) 34, 2949–3003
doi:10.1093/eurheartj/eht296

2013 ESC guidelines on the management of stable coronary artery disease

The Task Force on the management of stable coronary artery disease of the European Society of Cardiology

Table 4 Traditional clinical classification of chest pain

Typical angina (definite)	Meets all three of the following characteristics: • substernal chest discomfort of characteristic quality and duration; • provoked by exertion or emotional stress; • relieved by rest and/or nitrates within minutes.
Atypical angina (probable)	Meets two of these characteristics.
Non-anginal chest pain	Lacks or meets only one or none of the characteristics.

Sintomi

Dolore

- Qualità
- Localizzazione
- Irradiazioni
- Durata
- Fattori precipitanti
- Risposta alla trinitrina

Sintomi

Insufficienza ventricolare sx (dispnea)

Astenia (↓ funzione sistolica, ↓ portata, ↓ perfusione muscolare)

Aritmie (instabilità elettrica): sincopi, lipotimie

NO SINTOMI (angina silente)

Esame obiettivo

Dati scarsissimi:

difficoltà di osservare il paziente in corso di crisi anginosa

obiettività spesso negativa in corso del periodo critico

Diagnosi strumentale

Radiografia del torace
ECG a riposo
ECG da sforzo
ECG dinamico secondo Holter
Ecocardiografia
Scintigrafia miocardica (Tl201)
Cateterismo cardiaco e coronarografia

Terapia

Norme comportamentali (fumo, dislipidemia, eccesso ponderale, esercizio fisico regolare)
Nitroderivati
Calcio-antagonisti
β-bloccanti
Angioplastica
Rivascolarizzazione cardiochirurgica

European Heart Journal (2013) 34, 2949–3003
doi:10.1093/eurheartj/eht296

ESC GUIDELINES

2013 ESC guidelines on the management of stable coronary artery disease

The Task Force on the management of stable coronary artery disease of the European Society of Cardiology

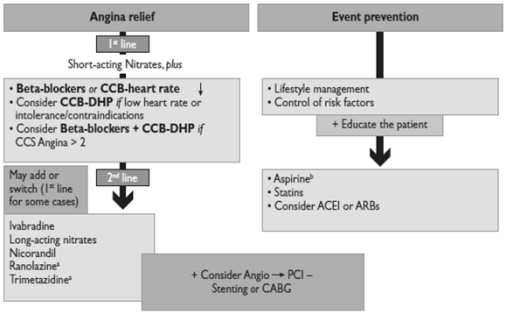


Figure 4 Medical management of patients with stable coronary artery disease. ACEI = angiotensin converting enzyme inhibitor; CABG = coronary artery bypass graft; CCB = calcium channel blockers; CCS = Canadian Cardiovascular Society; DHP = dihydropyridine; PCI = percutaneous coronary intervention.
[a]Data for diabetics.
[b]If intolerance, consider clopidogrel

Infarto miocardico acuto: definizione

__Necrosi ischemica__ di un settore del muscolo cardiaco, come conseguenza della __brusca__ e __prolungata__ diminuzione del flusso ematico coronarico

Epidemiologia

- Approximately every 43 seconds, an American will have an MI (AHA computation).
- On the basis of data from the ARIC study[4] of the NHLBI:
  - —This year, ≈635 000 Americans will have a new coronary event (defined as first hospitalized MI or CHD death), and ≈300 000 will have a recurrent event. It is estimated that an additional 155 000 silent MIs occur each year. That assumes that ≈21% of the 735 000 first and recurrent MIs are silent.
  - —The estimated annual incidence of MI is 525 000 new attacks and 210 000 recurrent attacks.
  - —Average age at first MI is 65.0 years for men and 71.8 years for women.

AHA Statistical 2015 Update, Circulation 2015

Etiopatogenesi

trombosi, occlusione subtotale o totale in assenza di circolo collaterale

ischemia → ↑ increzione catecolamine → ↑ FC, RP e contrattilità = ↑ consumo O2

Cause

Trombo sovrapposto su placca ateromasica
Spasmo coronarico
Ipotensione grave
Embolo dall'atrio o ventricolo sx
Trauma della arterie coronariche
Dissecazione aortica
Aortiti, vasculiti
Dissecazione coronarica spontanea
Fibrosi coronarica post-radioterapia
Insufficienza coronarica da disordini metabolici
Anomalie congenite delle coronarie
Uso di cocaina

Fasi del danno ischemico I

Fase del danno metabolico

A distanza di pochi secondi dall'interruzione del circolo coronarico, si registra un viraggio dalla glicolisi aerobia a quella anaerobia, con insufficiente produzione di ATP e accumulo di acido lattico

Fase del danno funzionale

Dopo circa 1 minuto di glicolisi anaerobia, le cellule miocardiche cessano di contrarsi.Da una parte, c'è un risparmio energetico. Dall'altra, un danno funzionale alla contrattilità ventricolare

Fasi del danno ischemico II

Fase del danno morfologico reversibile

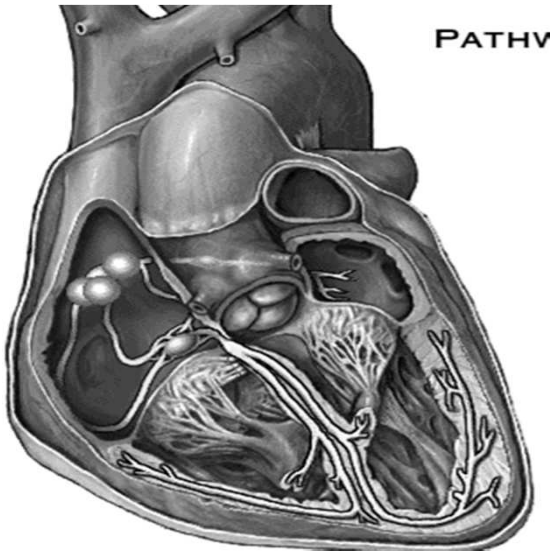
Dopo pochi minuti di ischemia, al ME si osserva rigonfiamento dei mitocondri con deplezione dei granuli di glicogeno. Nell'uomo, la durata di questo periodo (entro il quale la riperfusione può assicurare completa restitutio ad integrum) è variabile, e dipende dalle condizioni del circolo coronarico, e dalle entità e durata dell'occlusione.

Fase del danno morfologico irreversibile

Al di là del periodo reversibile, necrosi ischemica.

Localizzazione

quanto più è prossimale l'ostruzione, tanto più estesa è la necrosi



Coronarie

SX (parete anteriore e laterale, punta e gran parte del setto)

→ **tronco comune** ↗ **discendente anteriore**: IMA settale

↘ **circonflessa**: IMA laterale

DX (parete infero-basale ventricolo sx, parete posteriore del setto e ventricolo dx)

→ parte infero-basale ventricolo sx ➔ IMA inferiore

Sintomi

Dolore

Qualità

Localizzazione

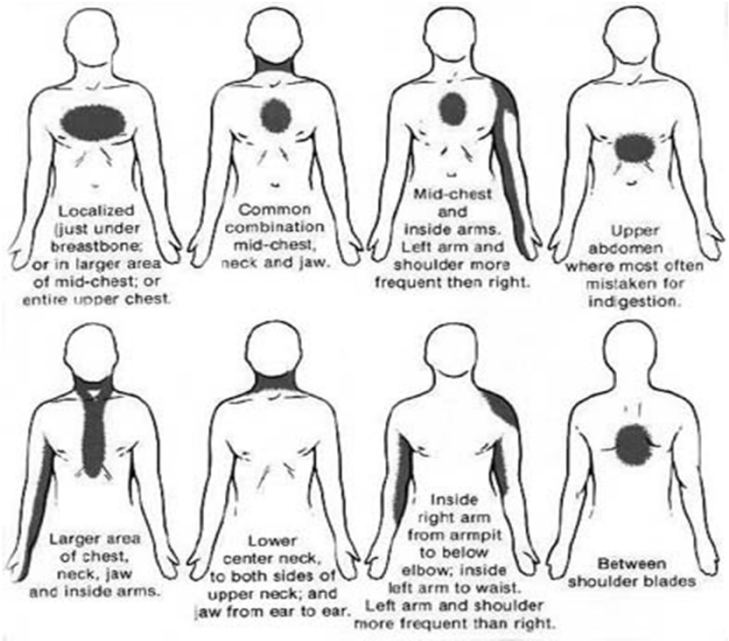
Irradiazioni

Durata

Fattori precipitanti

Risposta alla trinitrina

Localizzazioni del dolore



Sintomi

NB: *spesso* l'IMA ha un'anamnesi *negativa* e risulta essere la *prima* manifestazione di cardiopatia ischemica

Spesso non vi è fattore scatenante:

-la > parte avviene in corso di riposo o attività leggera

-2-13% in corso di esercizio fisico intenso

-8-23% durante il sonno

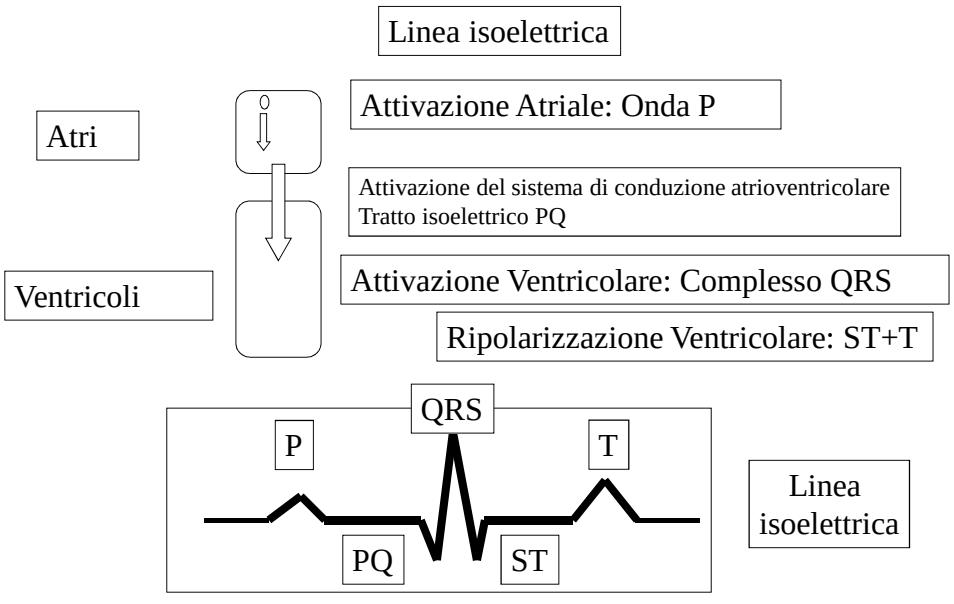
Attenzione ad anziani e diabetici

Accertamenti diagnostici

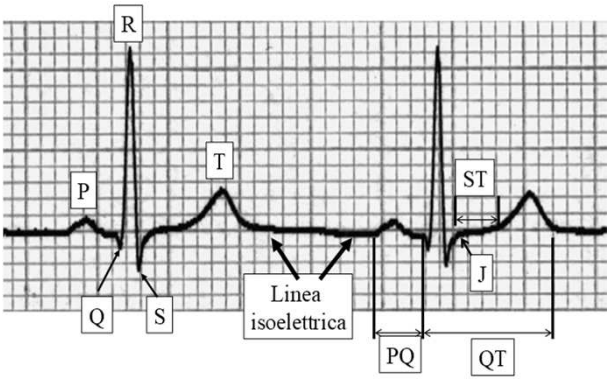
-Indici aspecifici (leucocitosi neutrofila, VES, iperglicemia)

-ECG

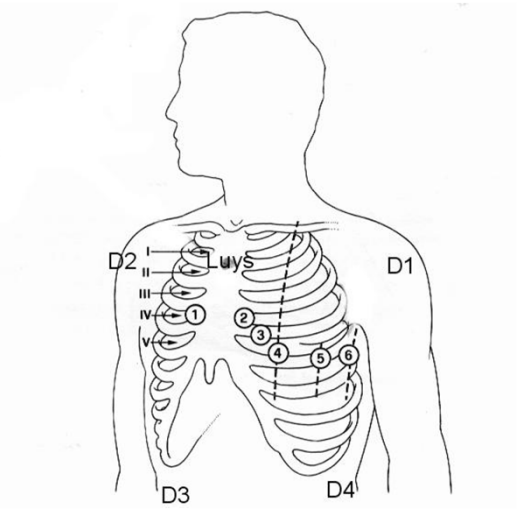
Cenni sull'elettrocardiogramma

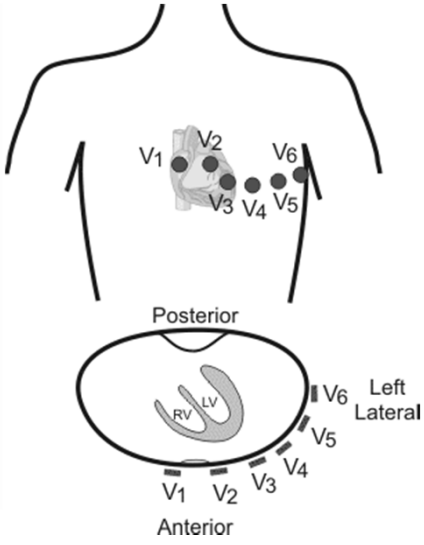


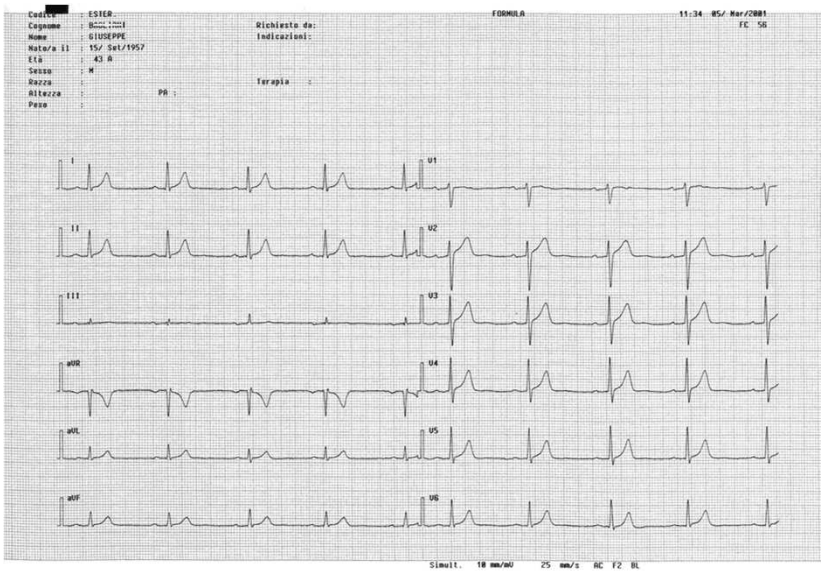
Cenni sull'elettrocardiogramma

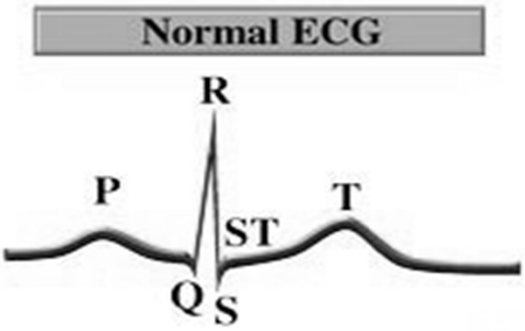


Derivazioni precordiali

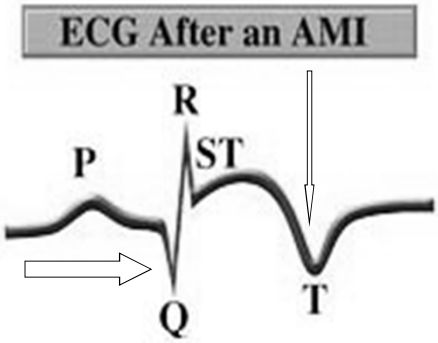








Caratteristiche tipiche sono la elevazione del segmento ST, inversione dell'onda T e un allargamento dell'onda Q.

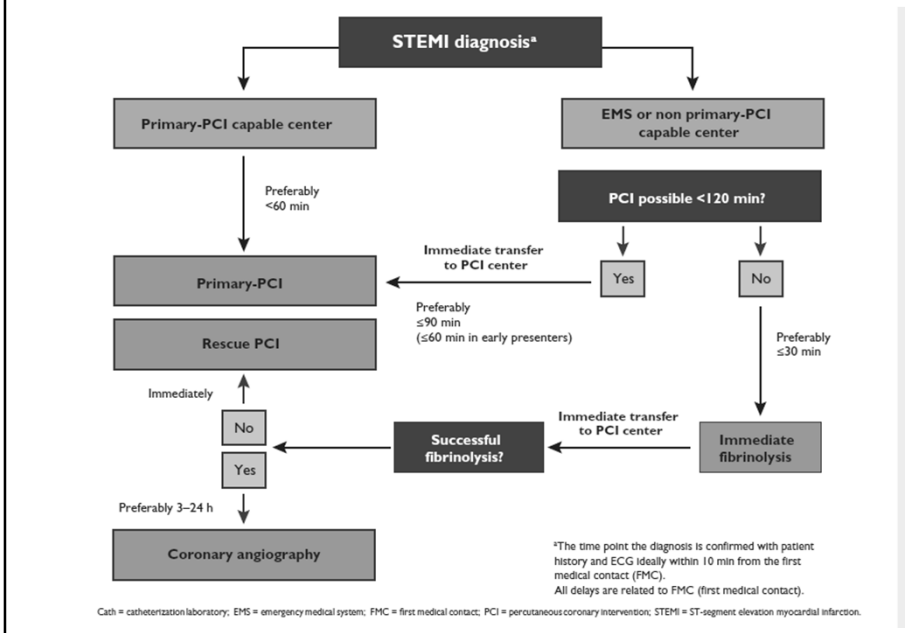


European Heart Journal (2012) **33**, 2569–2619
doi:10.1093/eurheartj/ehs215

ESC GUIDELINES

ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

The Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC)



Cath = catheterization laboratory; EMS = emergency medical system; FMC = first medical contact; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction.

Accertamenti diagnostici

-Enzimi cardiaci mioglobina, troponina I, CK-MB massa (CPK, CPK-MB, LDH, SGOT)

-Ecocardiografia (evidenza di zone di alterata cinesi)

-Esami radiosotopici: Tc99 pirofosfato: area “calda”
Tl201 area “fredda”

Complicanze

ARITMICHE

Extrasistoli ventricolari

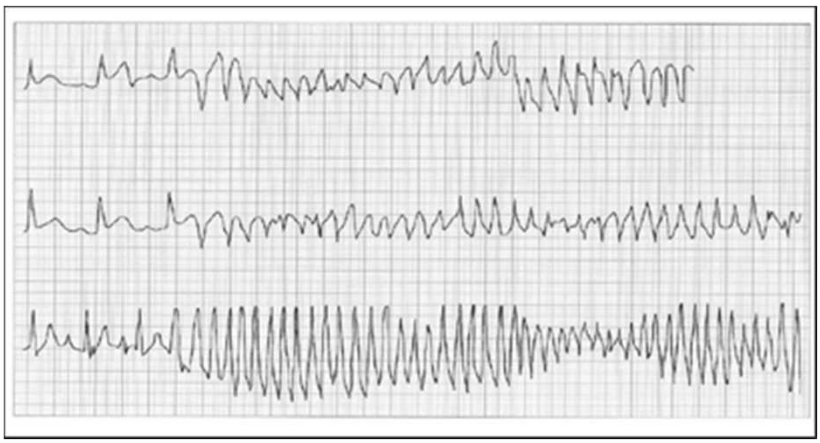
Tachicardia ventricolare

Aritmie sopraventricolari

Bradicardie

Blocchi

Fibrillazione ventricolare



Complicanze

MECCANICHE (EMODINAMICHE)

Shock cardiogeno primario (prime ore:3-4%)

Shock cardiogeno secondario (primi gg: 5%)

Rottura del setto (primi gg: 1%)

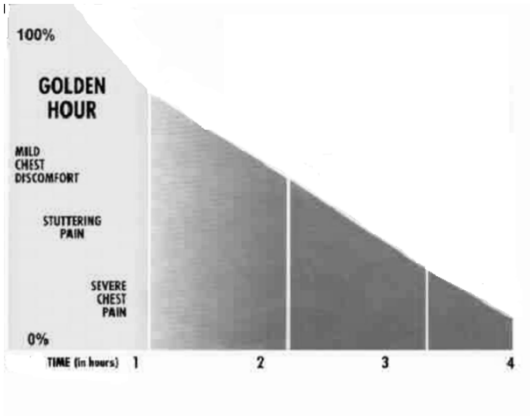
Insufficienza mitralica acuta (primi gg: 2%)

Rottura parete libera (morte improvvisa)

Estensione al ventricolo dx (precoce: 20% ima ant)

Aneurisma ventricolo sx (dopo alcune settimane: 12% ima ant

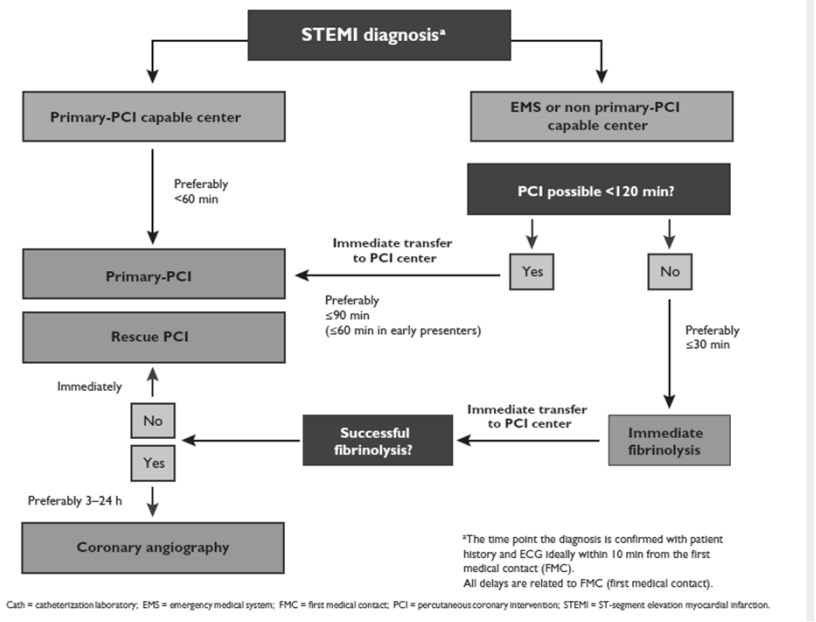
Terapia (fase iniziale)



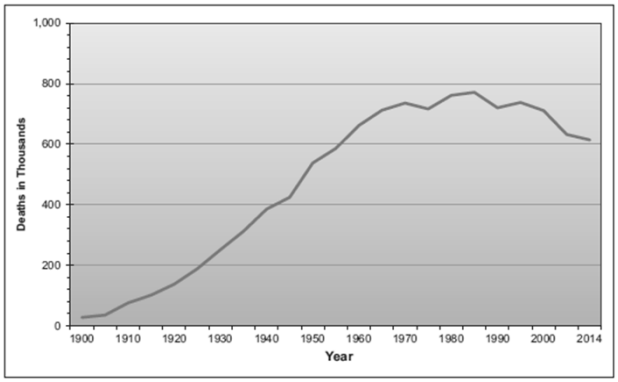
Ossigenoterapia
Analgesia
Trombolisi
Antiaggreganti piastrinici
Anticoagulanti
Beta-bloccanti
Calcio-antagonisti
Nitroderivati
ACE-inibitori

"L'ORA PREZIOSA" O GOLDEN HOUR.

Percentuale di muscolo cardiaco che può essere salvato da un intervento precoce in terapia intensiva in base alla rapidità dell'intervento

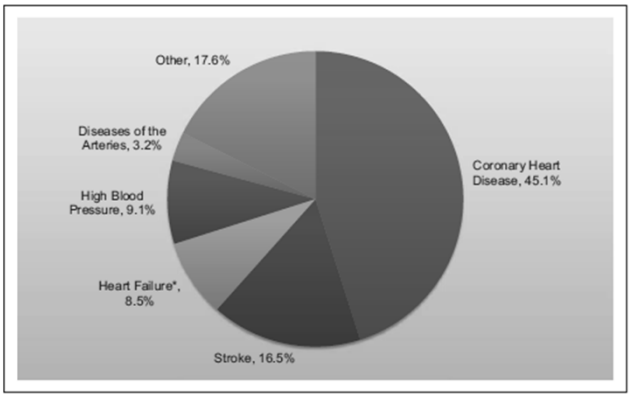


Decessi attribuibili a cause cardiache (USA)



AHA Statistical 2017 Update, Circulation 2017

Decessi attribuibili a cause cardiache (USA)



AHA Statistical 2017 Update, Circulation 2017

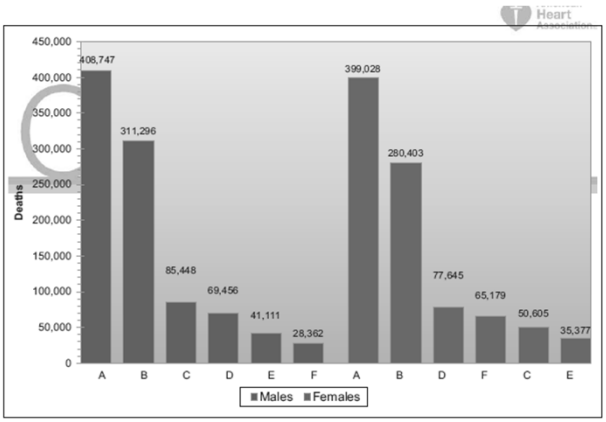


Chart 13-12. Cardiovascular disease and other major causes of death for all males and females (United States: 2014).
A indicates cardiovascular disease (*International Classification of Diseases, 10th Revision codes* I00–I99); B, cancer (C00–C97); C, accidents (V01–X59 and Y85–Y86); D, chronic lower respiratory disease (J40–J47); E, diabetes mellitus (E10–E14); and F, Alzheimer disease (G30).
Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

AHA Statistical 2017 Update, Circulation 2017

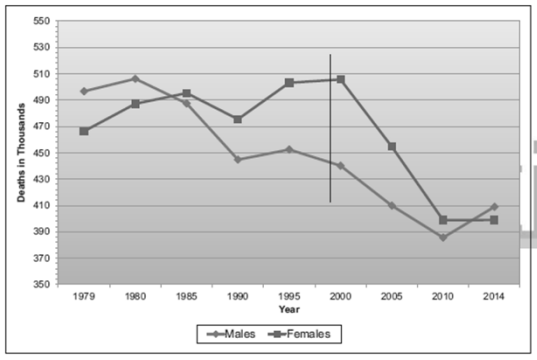


Chart 13-19. Cardiovascular disease (CVD) mortality trends for males and females (United States: 1979–2014).
CVD excludes congenital cardiovascular defects (*International Classification of Diseases, 10th Revision* [*ICD-10*] codes I00–I99). The overall comparability for cardiovascular disease between the *International Classification of Diseases, 9th Revision* (1979–1998) and *ICD-10* (1999–2013) is 0.9962. No comparability ratios were applied.
Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.

AHA Statistical 2017 Update, Circulation 2017

MEDICINA DI GENERE: COME PRENDERSI CURA DELLE DIFFERENZE

AHA Scientific Statement

Acute Myocardial Infarction in Women

A Scientific Statement From the American Heart Association

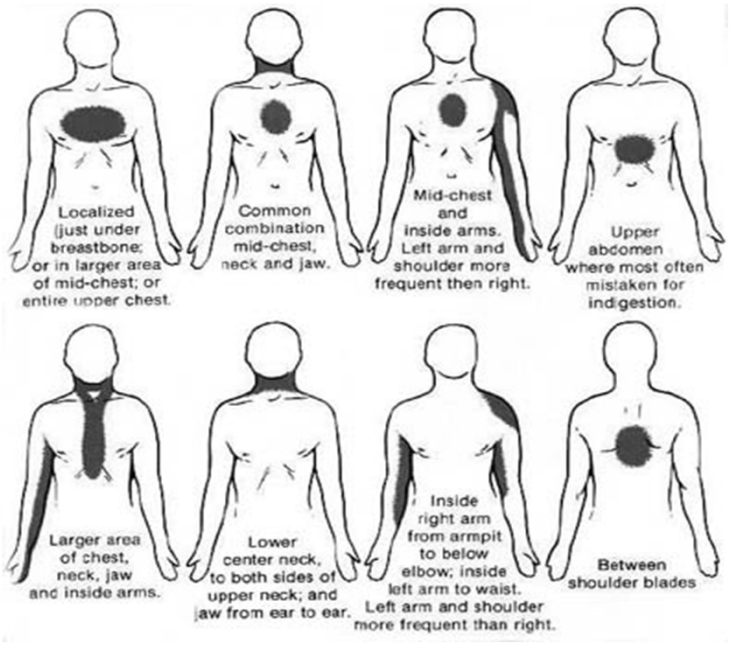
Laxmi S. Mehta, MD, FAHA, Chair; Theresa M. Beckie, PhD, FAHA, Co-Chair; Holli A. DeVon, PhD, RN, FAHA; Cindy L. Grines, MD; Harlan M. Krumholz, MD, SM, FAHA; Michelle N. Johnson, MD, MPH; Kathryn J. Lindley, MD; Viola Vaccarino, MD, PhD, FAHA; Tracy Y. Wang, MD, MHS, MSc, FAHA; Karol E. Watson, MD, PhD; Nanette K. Wenger, MD, FAHA; on behalf of the American Heart Association Cardiovascular Disease in Women and Special Populations Committee of the Council on Clinical Cardiology, Council on Epidemiology and Prevention, Council on Cardiovascular and Stroke Nursing, and Council on Quality of Care and Outcomes Research

Table 1. Typical Versus Atypical Symptoms in Women Presenting With AMI

Typical Symptoms	Atypical Symptoms
Chest pain/discomfort (pressure, tightness, squeezing)	Chest pain: sharp, pleuritic, burning, aching, soreness, reproducible
Additional symptoms with chest pain Radiation of pain to jaw, neck, shoulders, arm, back, epigastrium Associated symptoms: dyspnea, nausea, vomiting, lightheadedness, diaphoresis	Other symptoms excluding chest pain Unusual fatigue Unusual shortness of breath Upper back/chest pain Neck, jaw, arm, shoulder, back, epigastric pain Flu-like symptoms Dizziness Generalized scared/anxiety feeling Generalized weakness Indigestion Palpitations

AMI indicates acute myocardial infarction.

Mehta et al, Circulation 2016



AHA Scientific Statement

Preventing and Experiencing Ischemic Heart Disease as a Woman: State of the Science

A Scientific Statement From the American Heart Association

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McSweeney et al, Circulation 2016

Table 1. Sex-Related Differences in the Cardiovascular System

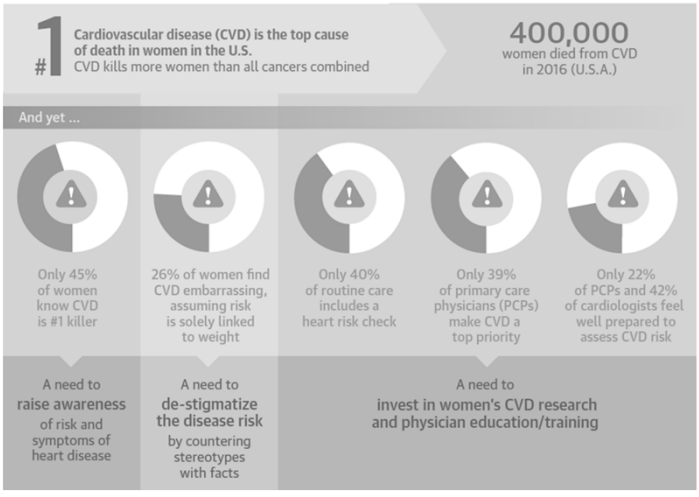
Parameter	Manifestations
Anatomy	Dimensions that are smaller in women (adjust for age and race): left ventricular mass, ventricular wall thickness, left atrial dimension, left ventricular end-diastolic dimension, and vessel size
Hormonal influences	Estrogen and progesterone are most influential in women; testosterone is predominant in men
	Menstruation can affect hematologic and electrocardiographic indexes
Cardiovascular function	Stroke volume is women is 10% less
	Pulse rate in women is 3–5 bpm faster
	Ejection fraction is higher in women
Physiology	Women have reduced sympathetic and enhanced parasympathetic activity
	Women have lower plasma concentrations of norepinephrine
Cardiovascular adaptations	In response to stress, women experience an increased pulse rate, resulting in increased cardiac output; men have increased vascular resistance, resulting in increased BP
	Women are more sensitive to altitude or body positioning changes and experience more orthostatic hypotension and syncope
Hematologic indexes	Women have a lower number of circulating red blood cells per unit volume of plasma (resulting in a lower hematocrit)
	Because of a lower hemoglobin, women have a lower oxygen-carrying capacity; this is balanced by women having a lower oxygen consumption
Electrocardiographic and electrophysiological indexes	Women on average have a longer corrected QT interval and a shorter sinus node recovery time
	Drug-induced torsades de pointes is more common in women
	Sudden cardiac death and atrial fibrillation are less common in women

McSweeney et al, Circulation 2016

Knowledge, Attitudes, and Beliefs Regarding Cardiovascular Disease in Women

The Women's Heart Alliance

C. Noel Bairey Merz, MD,[a] Holly Andersen, MD,[b] Emily Sprague, MA,[c] Adam Burns, MPP,[c] Mark Keida, PhD,[c] Mary Norine Walsh, MD,[d] Phyllis Greenberger, MSW,[e] Susan Campbell, MPH,[f] Irene Pollin, MSW, PhD(Hon),[g] Cassandra McCullough, MBA,[h] Nancy Brown, BA,[i] Marjorie Jenkins, MD,[j] Rita Redberg, MD,[k] Paula Johnson, MD,[l] British Robinson, MA, JD(Hon)[m]

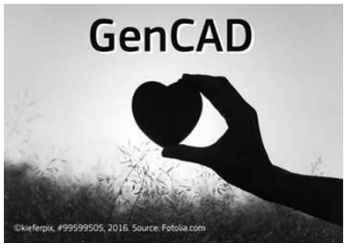


Bairey Merz et al, J Am Coll Cardiol 2017

GenCAD: Gender-specific mechanisms in coronary artery disease in Europe

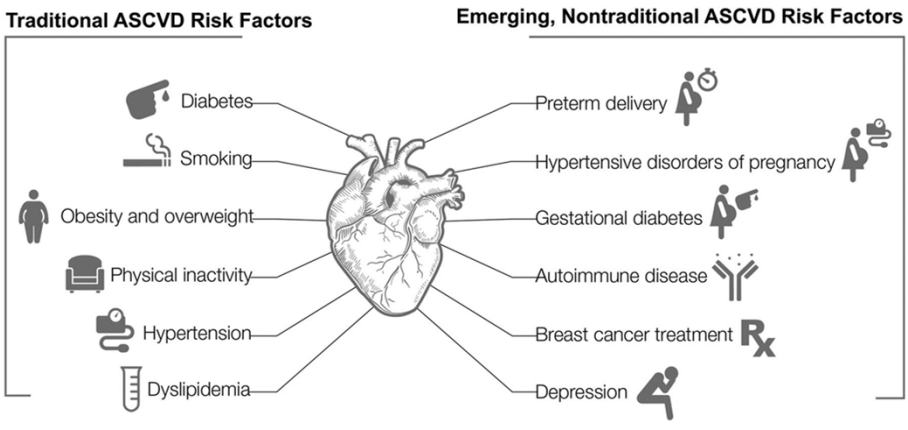
European Commission

Public Health



GENCAD Conference 2: Gender and health – awareness, facts, and European perspectives
Brussels, 11th October 2017

Traditional and nontraditional atherosclerotic cardiovascular disease (ASCVD) risk factors in women

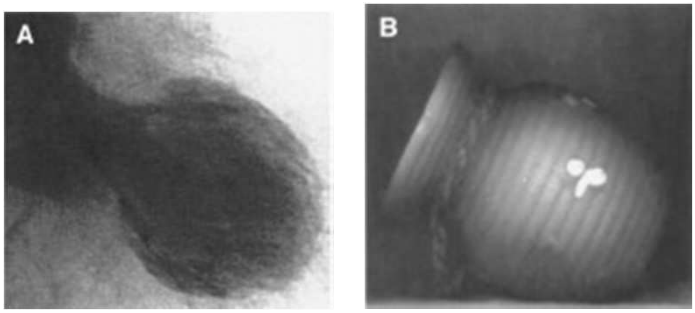


American Heart Association.

Mariana Garcia et al. Circ Res. 2016;118:1273-1293

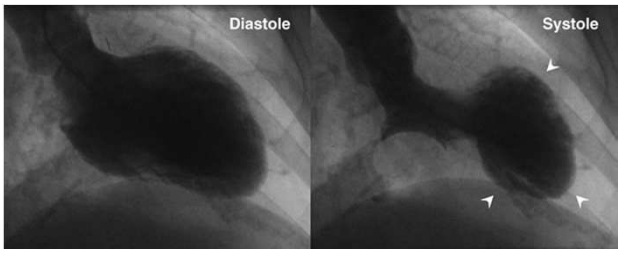
Cardiomiopatia Tako-Tsubo

Descritta per la prima volta in Giappone nel 1990. Il nome deriva dalle parole Tako (polpo) e Tsubo (giara)



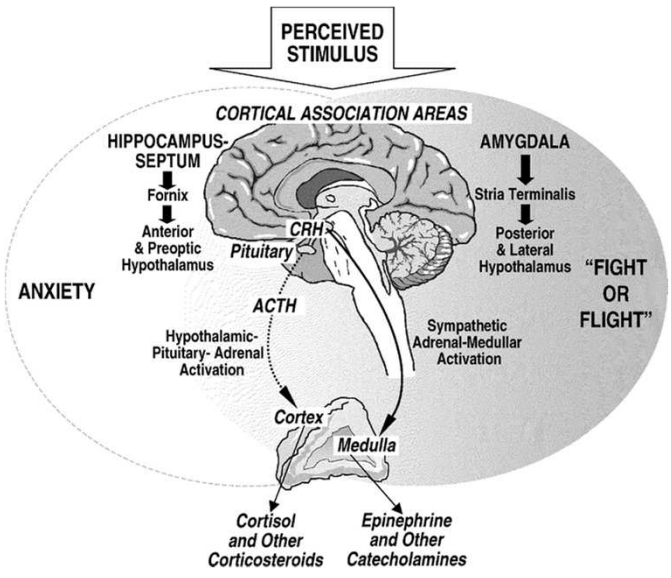
Cardiomiopatia Tako-Tsubo

Disfunzione transitoria delle sezioni apicale e medio-ventricolare del ventricolo sx, in assenza di malattia coronarica significativa, e spesso scatenata da uno stress (emotivo o fisico).



Cardiomiopatia Tako-Tsubo

- Fino al 2% delle sospette sindromi coronariche acute (SCA) (donne 6-9%, uomini 0.5%)
- Più frequente nelle donne (~90%), >80% dei casi in postmenopausa
- Mortalità intra-ospedaliera: 1-2%
- Recupero della funzione del Vsx: 1-4 sett.
- Recidive (10% ad un follow-up di 4 anni)



Triggers 'emotivi'

- Morte, malattia grave o grave lesione riguardante un membro della famiglia, un amico, il proprio animale
- Cattive notizie (diagnosi di grave malattia, divorzio di un famigliare)
- Grave litigio
- Aggressione
- Coinvolgimento in azione legale
- Incidente d'auto
- Trasloco
- Perdita economica (affari, gioco, licenziamento)
- Disastri naturali (terremoti..)
- Party a sorpresa
- Public speaking

European Heart Journal Advance Access published March 2, 2016

European Heart Journal
doi:10.1093/eurheartj/ehv757

CLINICAL RESEARCH
Heart failure/cardiomyopathy

Happy heart syndrome: role of positive emotional stress in takotsubo syndrome

Table 1 Happy heart events (n = 20)

Patient 1	Birthday party
Patient 2	Son's wedding
Patient 3	Meeting after 50 years with friends from high school
Patient 4	Preparing 50th wedding anniversary (pleasant anticipation)
Patient 5	Positive job interview
Patient 6	Wedding
Patient 7	Favourite driver won race car competition
Patient 8	Becoming grandmother
Patient 9	Surprise farewell celebration
Patient 10	Son's company opening
Patient 11	Favourite rugby team won game
Patient 12	Emotional speaking during a friend's birthday
Patient 13	Celebrating 80th birthday
Patient 14	Winning several jackpots at the casino
Patient 15	Celebration of normal PET-CT scan
Patient 16	Visiting opera with her family
Patient 17	Family party
Patient 18	Unexpected visit from favourite nephew
Patient 19	Grandchildren visiting from London (abroad)
Patient 20	Becoming great grandmother

Triggers 'fisici'

- Procedure chirurgiche e cardiochirurgiche
- Cause respiratorie
- Patologie gastroenteriche
- Patologie reumatologiche
- Patologie endocrine
- Patologie ematologiche
- Patologie infettive
- Dialisi
- Patologie neurologiche
- Sostanze illecite o farmaci: cocaina, abuso di antidepressivi, β2 stimolanti, adrenalina…
- Altri: traumi, ustioni, colpo di calore, abuso di energy drinks, puntura di medusa, ..

Attempted suicide as a trigger of Takotsubo syndrome: a minireview of available case reports

Roberto Manfredini[1,2,3] · Fabio Fabbian[1,2,3] · Rosaria Cappadona[1] · Beatrice Zucchi[1] · Pablo Jesus Lopez-Soto[2] · Maria Aurora Rodriguez-Borrego[2]

Takotsubo Cardiomyopathy and Acute Infectious Diseases: A Mini-Review of Case Reports

Alfredo De Giorgi, MD[1], Fabio Fabbian, MD[1], Marco Pala, MD[1], Claudia Parisi, MD[1], Elisa Misurati, MD[1], Christian Molino, MD[1], Arrigo Boccafogli, MD[1], Ruana Tiseo, MD[1], Susanna Gamberini, MD[2], Raffaella Salmi, MD[2], Francesco Portaluppi, MD[1], and Roberto Manfredini, MD[1]

Takotsubo syndrome and dialysis: an uncommon association?

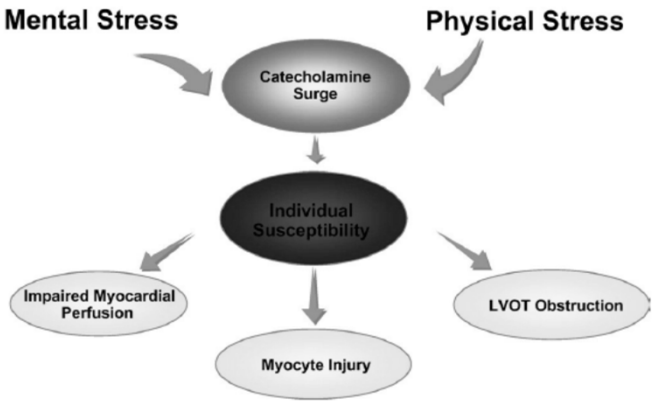
Roberto Manfredini[1,2,3], Fabio Fabbian[1,2,3], Alfredo De Giorgi[2], Rosaria Cappadona[1], Beatrice Zucchi[1], Alda Storari[2], Maria Aurora Rodriguez Borrego[3], Juan Manuel Carmona Torres[3] and Pablo Jesus Lopez Soto[3]

Breaking Heart
Chronobiologic Insights into Takotsubo Cardiomyopathy

Roberto Manfredini, MD[a,*], Raffaella Salmi, MD[b], Fabio Fabbian, MD[a], Fabio Manfredini, MD[c], Massimo Gallerani, MD[d], Eduardo Bossone, MD, PhD, FESC[e,f]

Heart and lung, a dangerous liaison-Tako-tsubo cardiomyopathy and respiratory diseases: A systematic review

Roberto Manfredini, Fabio Fabbian, Alfredo De Giorgi, Marco Pala, Alessandra Mallozzi Menegatti, Claudia Parisi, Elisa Misurati, Ruana Tiseo, Massimo Gallerani, Raffaella Salmi , Eduardo Bossone



Prasad et al, *Am Heart J* 2008

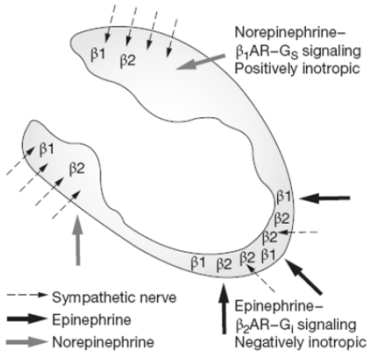


Figure 3 Schematic representation of the regional differences in response to high catecholamine levels, explaining stress cardiomyopathy.

High levels of circulating E might trigger a switch in intracellular signal trafficking, from G8 protein to Gi protein signaling through the β2AR. This change in signaling is negatively inotropic, and the effect is greatest at the apical myocardium, in which the density of β-adrenoceptors is highest.

Lyon et al, *Nat Clin Pract Cardiovasc Med* 2009

ESC European Society of Cardiology
European Heart Journal (2018) 39, 2032–2046
doi:10.1093/eurheartj/ehy076

CONSENSUS PAPER

International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology

Jelena-Rima Ghadri[1], Ilan Shor Wittstein[2], Abhiram Prasad[3], Scott Sharkey[4], Keigo Dote[5], Yoshihiro John Akashi[6], Victoria Lucia Cammann[1], Filippo Crea[7], Leonarda Galiuto[7], Walter Desmet[8,9], Tetsuro Yoshida[10], Roberto Manfredini[11], Ingo Eitel[12], Masami Kosuge[13], Holger M. Nef[14], Abhishek Deshmukh[3], Amir Lerman[3], Eduardo Bossone[15], Rodolfo Citro[15], Takashi Ueyama[16†] Domenico Corrado[17], Satoshi Kurisu[18], Frank Ruschitzka[1], David Winchester[19], Alexander R. Lyon[20,21], Elmir Omerovic[22,23], Jeroen J. Bax[24], Patrick Meimoun[25], Guiseppe Tarantini[17], Charanjit Rihal[3], Shams Y.-Hassan[26], Federico Migliore[17], John D. Horowitz[27], Hiroaki Shimokawa[28], Thomas Felix Lüscher[29,30], and Christian Templin[1]*

Ghadri et al, *Eur Heart J* 2018

ESC European Society of Cardiology
European Heart Journal (2018) 39, 2047–2062
doi:10.1093/eurheartj/ehy077

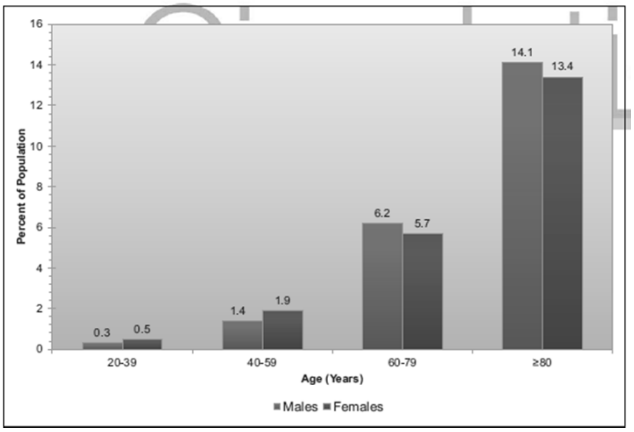
CONSENSUS PAPER

International Expert Consensus Document on Takotsubo Syndrome (Part II): Diagnostic Workup, Outcome, and Management

Jelena-Rima Ghadri[1], Ilan Shor Wittstein[2], Abhiram Prasad[3], Scott Sharkey[4], Keigo Dote[5], Yoshihiro John Akashi[6], Victoria Lucia Cammann[1], Filippo Crea[7], Leonarda Galiuto[7], Walter Desmet[8,9], Tetsuro Yoshida[10], Roberto Manfredini[11], Ingo Eitel[12], Masami Kosuge[13], Holger M. Nef[14], Abhishek Deshmukh[3], Amir Lerman[3], Eduardo Bossone[15], Rodolfo Citro[15], Takashi Ueyama[16†], Domenico Corrado[17], Satoshi Kurisu[18], Frank Ruschitzka[1], David Winchester[19], Alexander R. Lyon[20,21], Elmir Omerovic[22,23], Jeroen J. Bax[24], Patrick Meimoun[25], Guiseppe Tarantini[17], Charanjit Rihal[3], Shams Y.-Hassan[26], Federico Migliore[17], John D. Horowitz[27], Hiroaki Shimokawa[28], Thomas Felix Lüscher[29,30], and Christian Templin[1]*

Ghadri et al, *Eur Heart J* 2018

Prevalenza dello scompenso cardiaco (USA)



AHA Statistical 2017 Update, Circulation 2017

Definizione

Il cuore si definisce scompensato (o insufficiente) quando risulta incapace di pompare un flusso di sangue adeguato alle necessità dell'organismo.

Fisiopatologia

Il cuore adatta costantemente la propria gittata ai bisogni metabolici dell' organismo grazie alla combinazione di vari fattori (che determinano la gittata sistolica)

Pre-carico

Inotropismo

Post-carico

Frequenza cardiaca

Fisiopatologia

Come aumenta il cuore la propria gittata:

↑ frequenza cardiaca

↑ contrattilità

Gittata cardiaca:

gittata sistolica x frequenza cardiaca

Fisiopatologia

1. Pre-carico

Volume ematico totale
Distribuzione del volume ematico
Posizione del corpo
Tono venoso
Spremitura muscolare
Pressione intratoracica
Distensibilità cardiaca
Effetto aspirante
Contrazione atriale
Frequenza cardiaca

Fisiopatologia

2. Stato inotropo del miocardio

Catecolamine circolanti
Farmaci inotropi positivi
Effetto inotropo della frequenza
Ipossia, ipercapnia, acidosi
Farmaci inotropi negativi
Perdita di tessuto contrattile

Fisiopatologia

3. Post-carico

Se il cuore si adatta ad un aumento di pressione mantenendo costante la gittata sistolica, finisce per compiere un lavoro *maggiore*.

↑ consumo energetico (contrazione) → ↑ consumo di O2 = ↓ rendimento meccanico

Fisiopatologia

Rendimento (per una macchina) = $\frac{\text{lavoro svolto}}{\text{energia spesa}}$

Il cuore ha un rendimento *basso*: 20-25% dell'energia consumata

Quanto maggiore è il fattore pressione nel determinare il lavoro cardiaco, tanto minore è il rendimento.

Se un aumento del post-carico si mantiene a lungo nel tempo, il ventricolo mette in atto un altro meccanismo di compenso (l'*ipertrofia*).

Cause di scompenso (I)

Carico di lavoro eccessivo
-di pressione (ipertensione, stenosi aortica)
-di volume (insufficienza aortica, mitralica)

Perdita di parte del tessuto contrattile (cardiopatia ischemica)

Compromissione diffusa delle fibre miocardiche
(cardiomiopatie, miocarditi)

Cause di scompenso (2)

Condizioni che impongono una cronica gittata eccessiva
anemia grave, ipertiroidismo, fistola A/V, beri-beri, m. di Paget

Carico di lavoro acuto improvviso

Ostacolo al riempimento cardiaco

Meccanismi di adattamento cardiaco

Aumento del pre-carico (legge di Starling)

Stimolazione inotropa adrenergica

Ipertrofia del miocardio

a) sovraccarico di pressione (ispessimento pareti ventricolari: fibre più grosse)

b) sovraccarico di volume (dilatazione della cavità ventricolare: allungamento singole cellule muscolari)

Meccanismi di adattamento sistemico

Ipoperfusione periferica

↑ estrazione O_2 dal sangue arterioso (↑2-3 DPG), ↓ saturazione O_2 (dal 70 al 55%)

Redistribuzione della gittata cardiaca → attivazione SNA → stimolazione FC e contrattilità

vasocostrizione arteriolare in alcuni distretti (cute e visceri) mantenendo la perfusione a cuore e cervello. Il rene non viene protetto: ipoperfusione → attivazione SRAA.

Sintomi

Dispnea

conseguenza della congestione polmonare che provoca edema interstiziale e riduce la distensibilità dei polmoni.

- ↑lavoro dei muscoli respiratori. All'inizio, si manifesta solo per sforzi intensi, poi, con il progredire della malattia, per sforzi di minore entità, ed infine a riposo.

Classificazione NYHA

- **Classe I** pazienti cardiopatici senza sintomi
- **Classe II** pazienti cardiopatici che stanno bene a riposo ed hanno sintomi (dispnea) solo per sforzi di intensità ordinaria
- **Classe III** pazienti cardiopatici che stanno bene a riposo ed hanno sintomi (dispnea) anche per sforzi di intensità inferiore alla ordinaria
- **Classe IV** pazienti cardiopatici con sintomi anche a riposo

Heart failure functional status assessment

Class I	Class II	Class III	Class IV
Asymptomatic heart failure ejection fraction (EF) <40%	Mild symptomatic heart failure with ordinary exertion	Moderate symptomatic heart failure with less than ordinary exertion	Symptomatic heart failure at rest

I sintomi: FATICA

Assenza di correlazione tra emodinamica centrale, in particolare pressioni di riempimento ventricolare, e la severità dell'intolleranza allo sforzo

Determinante della fatica: *disfunzione muscolare*

- atrofia muscolare
- alterazione del metabolismo muscolare

Cause:

- ridotta perfusione del muscolo
- anormalità istologiche e biochimiche
- decondizionamento fisico
- malnutrizione
- stato catabolico
- atrofia, ridotta forza muscolare e resistenza

Alcune domande per investigare la dispnea e la fatica
- In che circostanze le manca il fiato?
- Quanto riesce a camminare senza problemi in pianura? E in salita? **- Quanti scalini riesce a salire?**
- Riesce a portare i pacchi della spesa? Fino a che peso?
- Ha difficoltà sessuali?
- Riesce a lavarsi senza sintomi ? E a vestirsi? E a mangiare? **- E' autonomo nei movimenti dentro casa (andare in bagno, scendere la letto,..)?**
- Ha tosse? Tossisce quando fa uno sforzo? Ha catarro?
- Quanti cuscini usa la notte per dormire? Ha degli episodi improvvisi di fame d'aria di notte? Comincia a tossire quando si sdraia? Ha bisogno di mettersi seduto nel letto? O di alzarsi? Ha bisogno di utilizzare l'ossigeno?

I sintomi: EDEMA

Sintomo di ritenzione idrica/congestione

- edemi declivi (arti inferiori, sacrale, generalizzato)
- nicturia, oliguria
- nausea, perdita di appetito
- senso di pienezza post-prandiale
- dolore e tensione addominale
- stipsi
- dolore al quadrante superiore dx (congestione epatica)
- contrazione della diuresi e aumento del peso

Alcune domande per investigare la ritenzione idrica
- Urina meno del solito? Urina più di giorno o di notte?
- Ha mai le caviglie gonfie? Le rimane evidente il segno dell'elastico della calza? Le scarpe sono diventate strette?
- E' aumentato di peso?
- Deve allargare la cintura dei pantaloni?
- Si sente subito pesante dopo mangiato? Ha dolori all'addome?
- Ha ridotto l'appetito? E' diventato stitico?

Altri sintomi: TOSSE

In genere di tipo non produttivo, a volte associata ad emottisi

Scatenata da: sforzo fisico, decubito supino, riposo notturno

La genesi risiede nella congestione della mucosa bronchiale con eccessiva produzione di muco; la rottura dei capillari bronchiali distesi e congesti causata dalla tosse provoca l'emottisi

I segni di congestione

- edema agli arti inferiori (eventuali cambiamenti cutanei e sottocutanei)
- ascite
- congestione polmonare (rantoli fini e crepitanti, versamento pleurico)
- stima della pressione venosa: con paziente in decubito a 45 gradi, sul lato destro valutazione del turgore giugulare e del reflusso epato-giugulare
- epatomegalia

Scompenso sinistro: quadro clinico

Insufficienza cardiaca sinistra

- **dispnea progressiva, da sforzo poi a riposo, tosse notturna, ortopnea, dispnea parossistica notturna, edema polmonare**

Scompenso dstro: quadro clinico

Insufficienza cardiaca destra

- **ipertensione venosa grande circolo**
- **stasi epatica: dilatazione vene giugulari, aumento volume fegato (epatomegalia molle dolente), edemi arti inferiori (molli, indolori e simmetrici; aumento di peso)**
- **versamenti liquidi in sierose: pleure, peritoneo, pericardio (fino al quadro dell'anasarca)**

Terapia

Riposo e norme igieniche
Farmaci inotropi
Riduttori del pre-carico
Diuretici
Vasodilatatori
Riduttori del post- carico

European Heart Journal (2016) **37**, 2129–2200
doi:10.1093/eurheartj/ehw128

ESC GUIDELINES

2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)

Table 1.1 Classes of recommendations

Classes of recommendations	Definition	Suggested wording to use
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective.	Is recommended/is indicated
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.	
Class IIa	*Weight of evidence/opinion is in favour of usefulness/efficacy.*	Should be considered
Class IIb	*Usefulness/efficacy is less well established by evidence/opinion.*	May be considered
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.	Is not recommended

Table 1.2 Level of evidence

Level of evidence A	Data derived from multiple randomized clinical trials or meta-analyses.
Level of evidence B	Data derived from a single randomized clinical trial or large non-randomized studies.
Level of evidence C	Consensus of opinion of the experts and/or small studies, retrospective studies, registries.

Pharmacological treatments indicated in patients with symptomatic (NYHA Class II-IV) heart failure with reduced ejection fraction

Recommendations	Class [a]	Level [b]	Ref [c]
An ACE-I[d] is recommended, in addition to a beta-blocker, for symptomatic patients with HFrEF to reduce the risk of HF hospitalization and death.	I	A	2, 163–165
A beta-blocker is recommended, in addition an ACE-I[d], for patients with stable, symptomatic HFrEF to reduce the risk of HF hospitalization and death.	I	A	167–173
An MRA is recommended for patients with HFrEF, who remain symptomatic despite treatment with an ACE-I[d] and a beta-blocker, to reduce the risk of HF hospitalization and death.	I	A	174, 175

ACEI = angiotensin-converting enzyme inhibitor; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; MRA = mineralocorticoid receptor antagonist; NYHA = New York Heart Association.
[a]Class of recommendation.
[b]Level of evidence.
[c]Reference(s) supporting recommendations.
[d]Or ARB if ACEI is not tolerated/contraindicated